Impact of Safflower Seed Oil on Serum Lipid Level and Immunocompetence of Rats

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Abstract: *Hypolipidemic, antioxidant and immune activities of safflower seed oil (SSO) were carried out to ascertain the claim of its utilization against diseases. High cholesterol fed diet rats exhibited a significant increase in total plasma lipid and atherogenicity and a significant decrease in high-density lipoproteins (HDL). Administration of linoleic acid is purified by safflower seed oil (LSO) or linoleic acid (LA) to hypercholesterolemic rats (LSO and LA groups, respectively) significantly ameliorated lipid parameters. Furthermore, malondialdehyde levels decreased and the efficiency of antioxidant defense system was improved compared to hypercholesterolemic group. Liver histological sections showed lipid storage in hepatocytes of hyperlipemia model group and an improvement was noted in both LSO and LA groups. Compared with three groups of LA, the level of tumor necrosis factor α (TNF-α) in high-fat diet with middle dose of LSO group was significantly increased. The current results suggested that LSO and LA have antiatherogenic immunity enhancement and hepatoprotective effects and the efficacy of LSO in lowering blood lipid and enhancing immunity is better than that of LA. LSO is worth further development and utilization.*

Keywords: *Safflower seed oil, Linoleic acid, Hyperlipidemia, Immune activity.*

Introduction

The link between diet and health has been recognized since ancient times. People have treated their patients with foods believed to have phamaceutical values. Linoleic acid (LA) is known to has important biological activity and its absence in a normal diet has been described as responsible for the development of a wide variety of diseases. LA is a polyunsaturated fatty acids (PUFA) belonging to the n-6 PUFA family. It may be one of the functional components related to the function of anticancer, anti-atherogenesis, antibacterial, anti-virus or regulation of immunity [12, 16]. Safflower seed oil (SSO) is an inexpensive source of several PUFA types, thus it is widely used for isolation and purification of PUFA. SSO is very interesting because of its extraordinary chemical properties. Its seed oil can be a nice resource of LA with a content of 73.5% of the total fatty acid [2, 7]. LA has received increasing attention over the last years due to its importance to human health. LA was shown to have several beneficial effects such as preventing coronary heart diseases, hypertriglyceridemia, blood platelet aggregation and lowering blood cholesterol, thus reducing the risk of arteriosclerosis, inflamation and several carcinomas. Several studies using animal models have reported positive effects of dietary LA, such as anti-obesity, anticarcinogenic activities and modulation

of immune functions. In particular, it has been proposed that LA has potential antiatherogenic properties [8, 15, 19].

Epidemiological studies have shown association between the consumption of diets rich in polyunsaturated fatty acids and a lower risk of chronic diseases like cancer, heart disease, and stroke [1, 17]. LA has been shown to exert various potent physiological functions such as antidiabetic, antiobese, anticarcinogenic and antihypertensive properties. This means LA can be effective to prevent lifestyle diseases or metabolic syndromes. Lipid disorders and inflammation are implicated in many pathological circumstances, such as cardiovascular and neurodegenerative diseases, rheumatoid, cancer and metabolic syndrome diseases [6, 10]. Lipid control is considered to be essential for the prevention and treatment of most of these disorders.

Naturally occurring vegetable oils and fats contain triacylglycerols as a major ingredient. Fats and oils are essential compositions of human food as they are important sources of energy, essential fatty acids and fat soluble vitamins [22]. The unutilized calories from dietary fat are a concern for health conscious individuals as it may lead to obesity. To address these concerns, PUFA are also being regarded for addressing concerns of health conscious individuals. Recent years, studies has shown that some foods or food ingredients (functional foods or ingredients) are potential to improve immunity activity, dyslipidemia and metabolic syndrome [13, 18].

It has been reported that feeding high levels of n-6 PUFA suppresses the cell-mediated immune response. Recent years, studies has shown that some foods or food ingredients such as LA are potential to improve metabolic syndrome, especially dyslipidemia, diabetes and obesity. Increasingly, more health experts are acknowledging that natural food is a good alternative medicine against many diseases [3, 5]. Anti-platelet agents, lipid-lowering agents such as statins decrease the risk of vascular events to a moderate but important degree. But these medicines are not without conspicuous side effects. Most lipid-lowering drugs have side effects, not long-term use, and hypolipidemic effects of using natural food control have been proven to be safe and eligible [20]. Most current hyperlipidemia drugs are expensive and have potential side effects, so research is increasingly focusing on natural alternative medicines that lessen blood lipid levels.

PUFA are of particular interest, as they are incorporated into the membranes of all cells including those of the immune system. Altered PUFA composition of the immune cells can influence their function through several mechanisms. Studies on mice suggest that the PUFA composition of the maternal diet has perinatal programming effects on the offspring's immune response. The essentiality of linoleic acid was discovered by some authors in pioneer research performed with rats fed fat-free diets and replenished with chemically methylated linoleic acid or linoleic acid in naturally occurring oils. Those authors distinguished the syndromes of long-term linoleic acid deficiency, specifically poor reproduction, dermatitis, poor growth, and low immunity [21]. LA is one of the essential fatty acid with a chain length of 18 carbon atoms, showing anti-inflammatory, hypocholesterol and immune activity [8].

This study was carried out to investigate the use of linoleic acid is purified by safflower seed oil (LSO) in serum lipid and immune function as a natural dietary supplement substance. To achieve this purpose, different levels of LSO were added into the basal diet, and studied to determine their effects on triglyceride (TG), high-density lipoproteincholesterol (HDL-C), superoxide dismutase (SOD), interleukin 6 (IL-6) and tumor necrosis factor α (TNF-α) were

compared with the control group. In the present study, the goal of the treatment with LSO is to reduce the increased LDL and TG, and to increase the HDL. Thus the present study was formulated to assess the hypolipidemic and immune effect of LSO, LA, in high-fat diet induced hyperlipidemic model.

Materials and methods

Materials

Safflower seeds were obtained from Shanxi Agricultural Seed Station (Taiyuan, People's Republic of China). All biochemical reagents and assay kits for biochemical variables were obtained from Shanghai Mingdian Biological Engineering Co. Ltd, China. All other reagents are analytical grade from Sinopharm Chemical Reagent Co. Ltd. safflower seed oil was prepared by means of solvent extraction method and LA was purified from safflower seed oil by means of urea adduction. High purity linoleic acid (99.5%) was prepared in our laboratory.

Animals and experimental procedure

Eighty male adult rats of Sprague-Dawley strain with an initial body weight of about 95 g were purchased from Shanxi Experimental Animal Center, China. Animals were housed in normal conditions (temperature 22 ± 2 ℃, a minimum relative humidity of 40%, 12 h light/dark cycle) and had free access to water and diet, in an approved animal house facility at Shanxi Experimental Animal Center, China. The control animals were given standard chow diets, whereas experimental groups received diets containing LSO and high-fat diets containing 7 wt% lard, 5.8 wt% egg yolk powder, 5 wt% sucrose, 0.5 wt% cholesterol, 0.2 wt% bile salt and 81.5 wt% basic diets. The animals were fed for a total period of 60 days. After accommodating to the laboratory circumstances for one week, rats were divided into eight groups of ten each and fed diets optionally during 60 days of experimental period. The ordinary guidelines on the use of living animals in scientific research and the guidelines for care and use of laboratory animals controlled by the Shanxi Experimental Animal Center Research Ministry were followed. At the end of the experimental period and after 12 h of fasting, Blood was collected in heparined tubes by aortic puncture of each rat and plasma samples, taken after centrifugal separation at 4000 rpm for 15 min, were kept at -20 ℃ until analysis. Fresh liver portions were cut, washed with cold saline and quickly weighed. 400 mg were homogenized (15%, w/v) in phosphate buffer (0.1 M, pH 7.4), centrifuged at 4000 rpm for 15 min and clear supernatants were stored at -20 ℃ until analyzed of various biochemical parameters.

Biochemical assays

For lipid profile analysis, serum total cholesterol, triacylglycerol, high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) cholesterol concentration were measured using commercial kits provided from Shanghai Mingdian Biological Engineering Co., Ltd. and a spectrophotometer (Shimadzu). The levels of total cholesterol (TC), triacylglycerol (TG), HDL-C, LDL-C was determined by using the relevant assay kits according to the instructions. The contents of total lipids in plasma separation and concentration were quantified gravimetrically by evaporating off the solvents using a rotary evaporator. Plasma lipid parameters for instance TC, TG and High-density lipoprotein-cholesterol (HDL-C) levels were measured by enzymatic methods, using commercial kits from Shanghai Mingdian Biological Engineering Co. Ltd. LDL-C was determined by the method of polyethylene sulfuric acid precipitation. Meanwhile, arteriosclerosis index AI = LDL-C/HDL-C was calculated. ApoA and ApoB were determined by immune

transmission turbidimetry and MK3 microplate assay was used. Superoxide dismutase (SOD) of serum and liver were measured by using commercial assay kits.

Antioxidant enzymes analysis

The determination of MDA (malonaldehyde), briefly, by thiobarbituric acid (TBA) method, MDA in the lipid peroxide degradation product can be condensed with TBA to form the red product, which has the maximum absorption peak at 532 nm. The content of MDA can be inferred by colorimetry. Units of SOD activity were expressed as the amount of enzyme required to inhibit the reduction of NBT by 50% and the activity was expressed as units per mg of protein. When the SOD inhibition rate in serum and tissue protein per mg reaches 50%, the corresponding SOD amount is one nitrite unit (NU), that is, one SOD activity unit (U). The activity of glutathione peroxidase (GPx) in the liver homogenate was measured by using commercial assay kits.

Histological studies

Medium portions of liver drawn from tested and control rats were fixed in 10% formalin, processed by standard procedure for paraffin embedding. Then 5 mg serial sections were cut and stained with hematoxylin-eosin.

Analysis of plasma TNF-α and IL-6

Serum levels of TNF-*α* and IL-6 were measured in rats. Blood samples from each group were labeled and placed in an anticoagulant tube. The blood samples were centrifuged at 4000 rpm for 15 min to separate the serum after resting for 15 min. Then placed in a low temperature box at -80 ℃ for preservation for inspection. The serum TNF-*α* and IL-6 levels were determined by ELISA.

The dose levels and test groups are summed up in Table 1.

Table 1. Dose levels and grouping

Group	***n***	**Weight, g**	**Feeding dose, mL/(kg·d)**
CD	10	95.32 ± 1.57	0
HFD	10	96.89 ± 1.72	0
LSOL	10	94.68 ± 1.81	50
LSOM	10	95.27 ± 1.67	80
LSOH	10	94.68 ± 1.66	100
LAL	10	95.38 ± 1.71	50
LAM	10	96.71 ± 1.27	80
LAH	10	94.99 ± 1.86	100

Note: CD – normal diet; HFD – high-fat diet; LSOL – high-fat diet with low dose of LSO; LSOM – high-fat diet with middle dose of LSO; LSOH – high-fat diet with high dose of LSO; LAL – high-fat diet with low dose of LA; LAM – high-fat diet with middle dose of LA; LAH – high-fat diet with high dose of LA.

Statistical analysis

The test results of each group were represented as the mean value ± standard deviation. Then, one-way analysis of variance (ANOVA) and the Turkey test were conducted in turns to

compare the results of the blank control group and the test groups. The *p*-values < 0.05 were considered as statistically significant. The statistical analysis was performed on SPSS Statistics 22.0.

Results and discussion

Effects of LSO and LA on weight gain, food intake and feed efficiency

After 60 days feeding, the weight gain and food intake of rats fed with high-fat diet were compared with those of normal control group, as shown in Table 2.

Table 2. Effects of LSO and LA on weight gain, food intake and feed efficiency

Group	Weight gain, g/60d	Food intake, g/60d	Feed efficiency, g/60d
CD	19.95 ± 2.12	641 ± 18.22	0.027 ± 0.008
HFD	37.27 ± 2.27	798 ± 17.87*	0.033 ± 0.009
LSOL	25.45 ± 1.42	737 ± 17.03	0.030 ± 0.007
LSOM	24.97 ± 1.68	696 ± 21.12	0.032 ± 0.006
LSOH	22.72 ± 2.14	673 ± 19.82	0.020 ± 0.003
LAL	34.85 ± 2.13	751 ± 18.82	0.036 ± 0.002
LAM	29.09 ± 3.78	718 ± 16.96	0.029 ± 0.002
LAH	25.55 ± 1.97	684 ± 17.87	0.035 ± 0.003

Note: * indicates the result is significantly different from that of the blank control group ($p < 0.05$); values represent means ± S.D. for $n = 10$ rats; values not sharing a common superscript within a row are statistically significant ($p < 0.05$).

As shown in Table 2, the rats given high-fat feed had increased significantly on weight gain and foot intake than those in the blank control group ($p < 0.05$). This means the long-term consumption of high-fat feeds disrupts the lipid metabolism in rats, and induces abnormal weight growth, which in turn leads to obesity. After being fed for 60 days, the food intake of rats fed with LSO or LA was lower than that of rats in the HFD by different degrees, indicating that the LSO and LA can suppress the appetite of rats to a certain extent.

There is no statistically significant difference between the rats fed with LSO or high-purity LA in terms of feed efficiency ($p > 0.05$). During the test, the rats in the HFD were docile, inactive and slow in eating; the rats in the blank control group were lively, active and fast in eating; the rats fed with LSO or LA fell between the above two groups in temperament and eating situation. During the feeding period, the rats in all groups saw a gradual increase in weight. The high-fat feed affected the digestion, absorption, metabolism and normal growth of rats.

Effects of LSO and LA on serum lipid of rats

Rats were fed with high-fat diet for 20 days, and triglyceride content was shown in Table 3. The same notes are used as those mentioned in Table 2. As shown in Table 3, the rats in the MG, which were given high-fat feed for 20 days, had much higher TG content in the serum than those in the CD. Hence, the modelling of hyperlipidemic rats is successful.

After feeding the rats for 60 days, the lipid lowering of LSO and LA at different doses was shown in Table 4 (the same notes are used as those mentioned in Table 2).

Table 3. TG contents in the serum after model building

Group	TG, mmol/L
CD	0.841 ± 0.088
HFD	2.903 ± 0.201
LSOL	2.987 ± 0.147
LSOM	2.985 ± 0.171
LSOH	2.958 ± 0.133
LAL	2.976 ± 0.195
LAM	2.939 ± 0.138
LAH	2.954 ± 0.109

Table 4. Effects of LSO and LA on lowering serum lipid in rats

Group	TG, mmol/L	TC, mmol/L	LDL, mmol/L	HDL-C, mmol/L
CD	0.976 ± 0.106	2.157 ± 0.277	0.427 ± 0.037	0.321 ± 0.038
HFD	1.647 ± 0.236	3.236 ± 0.437	0.767 ± 0.088	0.212 ± 0.032
LSOL	1.006 ± 0.158	2.086 ± 0.216	0.487 ± 0.068	0.231 ± 0.032
LSOM	1.107 ± 0.186	2.507 ± 1.211	0.613 ± 0.087	0.297 ± 0.038
LSOH	1.277 ± 0.178	2.377 ± 0.317	0.607 ± 0.078	0.258 ± 0.028
LAL	0.966 ± 0.122	2.337 ± 0.124	0.638 ± 0.048	0.209 ± 0.019
LAM	1.277 ± 0.167	2.212 ± 0.213	0.603 ± 0.092	0.212 ± 0.023
LAH	1.226 ± 0.123	2.121 ± 0.213	0.581 ± 0.159	0.279 ± 0.029

Table 4 compares the hypolipidemic effects of LSO and high-purity LA on rats. Compared with the rats in the CD, those in LSOL, LSOM and LSOH had very low TG contents in the serum, those in LSOL, LSOH, LAL, LAM and LAH had very low TC contents in the serum, those in LSOL had very low LDL contents in the serum, and those in LSOM had very high HDL-C contents in the serum. The rats in LSOM achieved a much higher HDL-C content than those in LAL and LAM. The above results show that both the LSO and LA can effectively alleviate hypolipidemia. Considering the various indices, it is concluded that the LSO has even better hypolipidemic effect than the LA.

Unreasonable dietary patterns, especially those with immoderate fat intake, can lead to obesity. Fortunately, high-fat fed rats treated with LSO had significant weight loss. This situation is closely related to the augment of bioenergy metabolism after ingestion of LSO. The PUFAs from LSO contributed to the research progress of anti-obesity. An eccentrically elevated blood lipid level can lead to deadly diseases. The bad habit of continuously eating high calorie diet is closely related to the prevalence of excessive metabolic syndrome. As reported, consumption of HFD significantly increased levels of serum TG, TC, and LDL-C, synchronously, reduced HDL-C levels, which was similar to the conclusions of previous studies [4, 11].

The present study was designed to assess, in hyperlipidemia rats, the effects of diet supplemented with LSO and LA on lipid profile and antioxidant activities in plasma and liver. Rats fed with diet rich in cholesterol resulted in an increase of TG, TC in plasma and liver and LDL-C levels, with decreased circulating HDL-C, thus affording a model for dietary hyperlipidemia. The increase of lipid parameters has been manifested to be a strong risk factor

for cardiovascular diseases in many populations. Fat is an important constituent of diet which controls serum and liver lipid levels. So amount and type of dietary fat affect serum TC. The results showed that LCA could improve HFD-fed induced hyperlipidemic rats, which provided a theoretical basis for the industrialization of LSO as a food supplement.

Effects of LSO and LA on antioxidant function

A high-fat diet can induce oxidative damage and lipid metabolism disorder. The antioxidant indices measured in the serum of each group of rats are listed in Table 5. The same notes are used as those mentioned in Table 2. Compared with the rats in the HFD, those in LAH had very high SOD contents in the serum, those in LSOL, LSOM, LSOH, LAL and LAM had general SOD contents in the serum, and those in all the six test groups had general GSH-Px, T-AOC and MDA values. Thus, the LA can promote the antioxidant function.

Table 5. Effects of LSO and LA on antioxidation of rat serum

Group	SOD, U/mL	GSH-Px, U/mL	T-AOC, U/mL	MDA, nmol/mL
CD	301.27 ± 31.12	177.81 ± 30.54	143.39 ± 4.34	7.59 ± 0.36
HFD	279.89 ± 23.72	155.17 ± 19.01	117.61 ± 6.02	8.06 ± 0.39
LSOL	250.21 ± 29.12	157.43 ± 16.33	122.39 ± 2.24	9.16 ± 0.29
LSOM	258.02 ± 13.87	162.03 ± 28.01	108.79 ± 3.19	8.94 ± 0.59
LSOH	274.31 ± 17.31	157.02 ± 25.82	122.01 ± 2.88	8.78 ± 0.43
LAL	316.91 ± 31.92	128.37 ± 16.58	114.01 ± 2.48	8.49 ± 0.23
LAM	255.61 ± 25.41	151.21 ± 26.07	124.97 ± 2.52	8.38 ± 0.56
LAH	390.81 ± 15.59	183.80 ± 10.07	122.89 ± 3.23	8.72 ± 0.23

Oxidative damage, due to free radicals, is connected with several diseases including diabetes, hypertension and cardiovascular diseases. The administration of antioxidants results in improved status in both patients and animal model. It has been also reported that consumption of PUFA lowers the blood pressure. The antihypertensive effect of PUFA may be raised by changes in prostaglandin synthesis, by alteration of membrane fatty acid composition, and succedent changes in membrane functions.

In agreement with previous reports, we observed a decrease in the activities of the antioxidant enzymes GPx, CAT and SOD in plasma and liver of hyperlipidemia rats, as compared to those of controls. Considering the endogenous stress-related markers (SOD, CAT and GPx), our results manifested that LCA and LA could improve efficiency the superoxide radical's transition to hydrogen peroxide and SOD activity in hyperlipidemia rats following deactivation of hydrogen peroxide by glutathione peroxidase. The increase of SOD activity might constitute a protection against superoxide anion elevation. Because SOD catalyzed the disintegration of superoxide anions to hydrogen peroxide (H_2O_2), this enzyme held back the further generation of free radicals [9].

Effects of LSO and LA on immune factors in rats

The immunity indices measured in the serum of each group of rats are listed in Table 6. The same notes are used as those mentioned in Table 2. Compared with the rats in the MG, those in the LAM had very low IL-6 in the serum, those in LSOL, LSOM and LSOH had similarly high IL-6 in the serum, and those in LSOL, LSOM, LSOH, LAL and LAM had very high TNF-α. The rats in LSOL, LSOM and LSOH showed much higher IL-6 and TNF-α values

than those in LAL, LAM and LAH. Therefore, the LSO boasts better immunity effects than the LA.

Table 6. Effects of LSO and LA on serum immune factors in rats

Group	IL-6, pg/mL	TNF-*α*, pg/mL
CD	301.19 ± 12.09	252.11 ± 9.17
HFD	286.01 ± 9.51	129.31 ± 5.90
LSOL	287.08 ± 11.54	207.08 ± 11.31
LSOM	312.81 ± 8.60	248.06 ± 17.12
LSOH	288.53 ± 13.31	188.46 ± 20.98
LAL	279.84 ± 5.14	193.44 ± 13.89
LAM	255.21 ± 16.18	187.31 ± 15.18
LAH	257.71 ± 8.64	151.03 ± 16.64

Cholesterol is the mother of all fat molecules in our bodies. It preserves neurotransmitter and brain feature, builds brain and nerve tissue, and nourishes the immune system. Our experiments testify in vivo the immunodepressive effect of LSO and LA. As a matter of fact, IL-6 release was obviously reduced in our medium-dose linoleic acid group. The LA groups IL-6 release was observably decreased compared to the control group. In addition, the effects of LSO on cytokine release seem to depend on the ester group to which these fatty acids are bound. Hwang reported that oral administration of n-6 fatty acids for 8 weeks decreases the content of linoleic acid and linolenic acid of monocyte membrane phospholipids and suppresses cytokine excretion [14]. In comparison to the oral route of administration the reduction of cytokine release was achieved in a markedly speeded up manner in our model by intravenous application.

In the present research, the rats in LSOM had much higher IL-6 contents than those in the MG, but the difference (9.46%) was not statistically significant ($p > 0.05$), and also much greater TNF-*α* content in the serum than the latter. Comparing the three groups fed with LSO and those fed with LA, it is learned that the LSO outperforms the LA in immunity enhancement. The excellent performance may come from the LA and *α*-Linolenic acid (ALA) contained in the LSO. Among them, the two components work together and they could dissolve dangerous clots in blood vessels, prevent cancer, diminish inflammation and slow down aging. The LA and ALA components work synergistically as a broad-spectrum immune promoter.

Histopathologic examination

According to the HE-strained liver tissues in Fig. 1, the liver cells of the rats in the NG were arranged normally in a cord-like pattern, with normal tissue structure, no inflammatory infiltration, and no vacuolar degeneration. For the rats in the LSOL, the liver cells were arranged chaotically, with a few lipid droplets, low inflammatory infiltration, and low vacuolar degeneration. For the rats in the LSOM, the liver cells were arranged chaotically, with very low vacuolar degeneration and no inflammatory infiltration. For the rats in the LSOH, the liver cells contained very few lipid droplets, with extremely low inflammatory infiltration and no inflammatory infiltration.

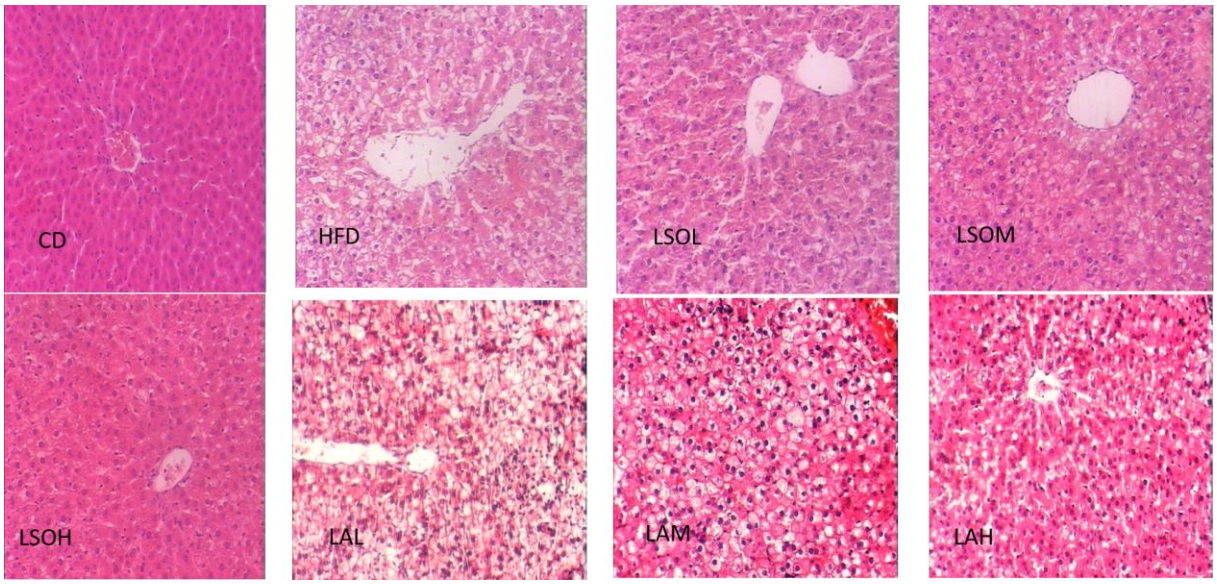

Fig. 1 HE-staining in liver organs of rats (×200)

For the rats in the LAL, LAM and LAH, the liver cells were arranged chaotically. With the growth in dose level, the number of lipid droplets and the vacuolar degeneration gradually declined. Slight inflammatory infiltration was observed on the liver cells of the rats in the LAM, while inflammatory infiltration was not seen on those of the rats in LAL or LAH.

The role of Mediterranean diet to take precautions against atherosclerosis and other diseases is proven. Epidemiological, clinical investigations and experiments researches indicated that diets, which include vegetable oils, improve lipid status and antioxidant activity in plasma and liver. These and other investigators attribute the positive influence of Mediterranean diet to low content of saturated and high content of mono- and polyunsaturated fatty acids in vegetable oils. Beneficial effects of diet, rich with nutritional products using flax and pumpkin seeds mixture, on hypercholesterolemic status in adult rats were also confirmed in some researchers' previous studies.

The pathological section of liver showed that there was a significant decrease in the liver total cholesterol level, non-HDL cholesterol level, and triglyceride level by treating the rats with LSO and LA in both normal and hypercholesterolemic condition. According to the histopathologic examination of the liver tissues from hyperlipidemic rats, with the growth in dose level of the LSO and LA, the number of lipid droplets and the vacuolar degeneration gradually declined. The liver protection mechanism of LSO remains to be researched.

Conclusion

Collectively, this research demonstrates that Linoleic acid is purified by LSO, can be prepared through urea adduction from safflower seed oil. This oil contains nutrient molecules like linolenic acid, tocotrienols and phytosterols. The current research has shown that the LSO and LA could reduce the TC and TG contents in the serum and liver of rats. The immune-enhancing effect of LSO was stronger than that of LA. Linoleic acid of LSO worked in concert with linolenic acid as a highly effective immune promoter. Therefore, LSO could be adopted as a low-calorie fat to combat obesity, and a hypolipidemic food to promote cardiovascular health. It could also be treated as a safe source of essential fatty acids in feed and food formulations.

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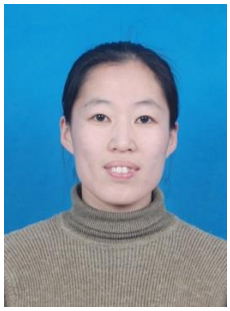

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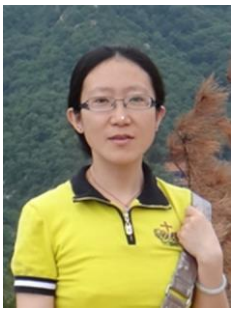

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