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ORIGINAL ARTICLE

Dietary effect of *Pleurotus eryngii* on biochemical function and histology in hypercholesterolemic rats

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Abstract This work was conducted to investigate diet supplement of king oyster mushroom fruiting bodies on biochemical and histological changes in hypercholesterolemic rats. Six-week old female Sprague–Dawley albino rats were divided into three groups of 10 rats each. The feeding of 5% powder of the fruiting bodies of *Pleurotus eryngii* to hypercholesterolemic rats reduced their plasma total cholesterol, triglyceride, low-density lipoprotein, total lipid, phospholipids, and LDL/HDL ratio by 24.05%, 46.33%, 62.50%, 24.63%, 19.22%, and 57.14%, respectively. Mushroom also significantly reduced body weight in hypercholesterolemic rats. However, it had no adverse effects on plasma albumin, total bilirubin, direct bilirubin, creatinine, blood urea nitrogen, uric acid, glucose, total protein, calcium, sodium, potassium, chloride, inorganic phosphate, magnesium, and enzyme profiles. Feeding mushroom increased total lipid and cholesterol excretion in feces. The plasma lipoprotein fraction, separated by agarose gel electrophoresis, indicated that *P. eryngii* significantly reduced plasma β and pre- β -lipoprotein, while increased α -lipoprotein. A histological study of hepatic cells by conventional hematoxylin–eosin and oil red O staining showed normal findings for mushroom-fed hypercholesterolemic rat. The present study suggests that 5% *P. eryngii*

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diet supplement provided health benefits by acting on the atherogenic lipid profile in hypercholesterolaemic rats. Therefore, king oyster mushroom could be recommended as a natural cholesterol lowering substance within the human diet.

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1. Introduction

Pleurotus eryngii is known as king oyster mushroom. It is a popular and commercially cultivated edible mushroom in Korea (Ro et al., 2007). Mushrooms have long been widely appreciated for their good flavor and texture. Recently, they are recognized as a nutritious food as well as an important source of biologically active compounds of medicinal purposes (Alam et al., 2009a).

Increased plasma levels of total cholesterol (TC), low-density lipoprotein (LDL) and very low-density lipoprotein (VLDL) as well as lowered levels of high-density lipoprotein (HDL) cholesterol have been identified as major risk factors in the development of coronary artery disease (Alberts et al., 1989). Edible mushrooms are rich in their high-fiber content, sterols, proteins, microelements and a low calorific value that are almost ideal for diets designed to prevent cardiovascular diseases (Alam et al., 2008; Breene, 1990). Early attempts to identify inhibitors of cholesterol synthesis resulted in the development of inhibitors that could affect stages in the biosynthetic pathway for cholesterol formation. A major rate-limiting step in the pathway is at the level of the microsomal enzyme 3-hydroxy-3-methylglutaryl-coenzyme-A (HMG-CoA) reductase. HMG-CoA reductase occurs early in the biosynthetic pathway and is among the first committed steps to cholesterol formation that catalyzes the reductions of HMG-CoA into mevalonate (Rodwell et al., 1976).

The genus *Pleurotus* has several species that produce mevinolin (Gunde-Cimerman et al., 1993). Oyster mushroom has been shown to produce the highest amount of lovastatin in the fruiting bodies, especially in the lamellae or gills.

The addition of 5% dried fruiting bodies of oyster mushroom to a high-cholesterol diet effectively reduced cholesterol accumulation in the plasma and liver of experimental rats redistributing cholesterol in favor of HDL, reduced production of TC, VLDL, and LDL, reduced cholesterol absorption and HMG-CoA reductase activity in the liver (Hossain et al., 2003). It has been suggested that mushrooms could be recommended as a natural cholesterol reducing substance within the human diet. In spite of the medicinal importance or the therapeutic potential of *P. eryngii*, there have not been studies on antihyperlipidemic activities. Moreover, comprehensive studies on the antihyperlipidemic properties of this mushroom are not available. The aim of the present study is to evaluate the antihyperlipidemic potentials from the fruiting bodies of *P. eryngii* in atherogenic lipid and liver function, and histology of hypercholesterolemic rats.

2. Materials and methods

This study was carried out from February 2010 to January 2011 at the Animal House and Laboratory of Applied Microbiology, Division of Life Sciences and the experimental protocols were approved by ethical committee of the University of Incheon, Republic of Korea. All experimental procedures were

performed in accordance with the guide for the care and use of experimental animals.

2.1. Mushroom

Fresh fruiting bodies of *P. eryngii* were obtained from Hanultari mushroom farm, Korea. A pure culture was deposited in the Culture Collection and DNA Bank of Mushroom (CCDBM), Division of Life Sciences, University of Incheon, Korea and acquired accession number, IUM-4030. Fresh fruiting bodies were dried with hot air at 40 °C for 48 h and pulverized.

2.2. Animals

Thirty female Sprague–Dawley albino rats (101 ± 4.2 g, 6-weeks old, purchased from Central Lab. Animal Inc., Seoul, Korea) were used. All rats were acclimated to the animal room for 1 week. The rats were housed in an animal room at 23 ± 2 °C under a 12 h dark–light cycle (17:00–5:00 h) and relative humidity of 50–60%. Rats were divided into three feed groups: a basal diet (normocholesterolemic control rats; NC), basal diet with 1% cholesterol (hypercholesterolemic rats; HC), and a basal diet with 1% cholesterol and 5% *P. eryngii* powder (mushroom-fed hypercholesterolemic rats; HC + PE). The composition of the basal diet was as follows (in g/100 g): wheat flour 50; rice power 11.25; wheat bran 19; casein 8; egg white 10; soybean oil 1; table salt 0.5; vitamin mixture 0.125; mineral mixture 0.125. The composition of the vitamin mixture in the diet was as follows (g/100 g vitamin mixture): retinyl acetate 9.5×10^{-4} , cholecalciferol 1.2×10^{-3} , α -tocopherol acetate 0.05, thiamine hydrochloride 2.4, nicotinic acid 12, riboflavin 2.4, D-calcium pantothenate 9.6, pyridoxine hydrochloride 1.2, folic acid 9.5×10^{-2} , vitamin K 0.25, cyanocobalamine 9.5×10^{-3} , inositol 47.95 and ascorbic acid 24.0. The composition of the mineral mixture added to diet was as follows (g/100 g of mineral): calcium gluconate 28.5, K₂HPO₄ 17.3, CaCO₃ 26, MgSO₄ 12.6, KCl 12.6, CuSO₄ 0.06, FeSO₄ 0.3, MnSO₄ 0.55, NaF 2.5×10^{-4} , KI 9×10^{-4} , sodium molybdate 3×10^{-4} , SeO₂ 3×10^{-4} , and CrSO₂ 1.5×10^{-3} . Rats were fed for 42 days.

2.3. Plasma biochemical analysis

At the end of the experimental period, overnight-fasted animals were sacrificed under injectable anesthetic (Zoletil 50; VIRBAC Laboratories, Carros, France). Blood samples were collected with a disposable plastic syringe into heparinized tubes. Plasma was prepared by centrifugation at 2493×g for 10 min. Plasma triglyceride (TG) concentration was measured enzymatically using the glycerophosphate oxidase assay. Plasma total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C), total lipid (TL), and phospholipid (PL) levels were measured enzymatically by the

cholesterol oxidase assay (Burtis and Ashwood, 2006) using commercially available assay kits (Sekisui Medical Co., Ltd., Tokyo, Japan). Plasma albumin, total bilirubin, direct bilirubin, creatinine, blood urea nitrogen, uric acid, glucose, total protein, and electrolyte parameters, including calcium, sodium, potassium, chloride, inorganic phosphate, and magnesium were measured by standard methods using an auto analyzer (Hitachi 7600-210; Hitachi, Tokyo, Japan).

Very low density lipoprotein cholesterol was calculated as follows:

$$\text{VLDL-C} = [\text{TC} - (\text{HDL-C} + \text{LDL-C})].$$

2.4. Plasma enzyme analysis

The activity of the plasma alanine aminotransferase (ALT) and aspartate aminotransferase (AST) was determined using the kinetic method (Burtis and Ashwood, 2006). Plasma alkaline phosphatase (ALP) activity was determined using 4-nitrophenyl phosphate. ALP catalyzes the hydrolysis of 4-nitrophenyl phosphate, forming phosphate and free 4-nitrophenol, which is colorless in dilute acid solutions. But, under alkaline conditions 4-nitrophenol is converted into the 4-nitrophenoxide ion, which is an intense yellow color. The absorbance of this color compound was measured spectrophotometrically at 420 nm to determine plasma ALP activity.

2.5. Fecal total lipid and cholesterol analysis

Feces were collected for 7 days before and at the end of 42 days, lyophilized, and then milled into powder. Total lipids were extracted with chloroform/methanol (2:1 v/v) according to the method of Folch et al. (1957). One gram of fecal powder was mixed with 10 ml of chloroform and 5 ml of methanol solution and stirred at 150 rpm for 3 days at room temperature. The suspension was filtered through Whatman No. 2 filter paper (Whatman, Maidstone, UK), the methanol was aspirated, and the chloroform was evaporated. The extracted lipids were then weighed. Two milliliters of H₂O was added, and a suspension was created using a bath sonicator. This suspension was used to estimate fecal cholesterol content, which was estimated by the enzymatic method using the cholesterol oxidase assay.

2.6. Plasma lipoprotein separation by agarose gel electrophoresis

Plasma lipoprotein fractions were determined by agarose gel electrophoresis (Kido et al., 2001). Three lipoprotein fractions were detected by electrophoresis, which will henceforth be referred to as β -lipoprotein (LDL), pre- β -lipoprotein (VLDL), and α -lipoprotein (HDL). Sample application (2 μ l), electrophoresis (80 V, 30 min.), staining (Fat Red 7B), drying, and densitometric scanning (525 nm) were performed automatically by the Helena TITAN GEL Lipoprotein Electrophoresis System (Helena Laboratories, Beaumont, TX, USA). After electrophoresis, lipoprotein fractions were visualized with enzymatic staining reagents. The visualized gel plate was scanned on a densitometer, and the lipoprotein scanning patterns were identified using analytical software (electrophoresis data bank, K.K. Helena Laboratories, Saitama, Japan). The scanned patterns were divided into lipoprotein fractions using the nadirs of the lipoprotein sequential curve. Lipoprotein levels were estimated from the area percentages and total concentrations.

2.7. Histological analysis of liver

Liver tissues were rapidly dissected, fixed in liquid nitrogen and 10% formalin solution, and stored until use at -80°C . A representative part of the frozen tissues was processed with a cryo microtome (Cryotome FSE Cryostat; Thermo Electron Corp., Cambridge, MA, USA) using sections 5- μ m thick and stained with oil red-O (Bayliss-High, 1990). A representative part of the formalin fixative liver tissues was processed for 4- μ m thick paraffin embedded sections using a microtome (Microtome HM 450; Thermo Electron Corp.) and then stained with hematoxylin and eosin. Both stained tissue samples were then examined and photographed under a light microscope to assess the presence of lipid. Digital images were obtained using an Olympus BX51 microscope equipped with a Camedia C3040ZOOM digital camera (Olympus America Inc., Melville, NY, USA). All images were taken under 40 \times magnification.

2.8. Statistical analysis

Results are expressed as means \pm SD. Intergroup differences were analyzed by a one-way analysis of variance followed by post-hoc tests. The SPSS ver. 11.5 (SPSS Inc., Chicago, IL, USA) were used for analysis. A $p \leq 0.05$ was considered statistically significant.

3. Results and discussion

3.1. Effect of *Pleurotus eryngii* on bodyweight

The results on the effect of *P. eryngii* in the body weight after 6 weeks among the NC, HC, and HC + PE rats were 243 ± 12.5 , 249 ± 11.9 , and 224 ± 12.4 , respectively. Feeding of *P. eryngii* reduced the body weight in hyper and normocholesterolemic rats by 16.89% and 13.38%, respectively. This finding is of special significance because obesity is associated with numerous diseases including diabetes, atherosclerosis, coronary heart disease and others (Alam et al., 2009b).

3.2. Effect of *Pleurotus eryngii* on plasma lipid profile

Plasma lipid profiles concentrations in NC, HC, and HC + PE group rats after *P. eryngii* feeding for 6 weeks have been presented in Table 1. Plasma TC, TG, HDL-C, LDL-C, VLDL-C, TL, and PL in HC rats increased by 17.09%, 36.68%, 12.23%, 22.35%, 19.01%, 19.82%, and 16.14%, respectively, compared with levels in NC rats, whereas these parameters decreased significantly by 24.05%, 46.33%, 10.90%, 62.50%, 19.79%, 24.63%, and 19.22%, respectively, in HC + PE rats compared with HC rats. The ratio of plasma LDL and HDL is shown in Fig. 1. In HC rats, this ratio increased by 8.89%, compared with NC rats, whereas this ratio was reduced significantly by 57.14% in HC + PE rats compared with HC rats.

The results show that feeding 5% *P. eryngii* to hypercholesterolemic rats significantly ameliorated the plasma atherogenic lipid profiles in experimentally induced HC rats. Rats are particularly resistant to the development of hypercholesterolemia and atherosclerosis (Andrus et al., 1956) and have a strong ability to maintain their plasma cholesterol levels (Fujioka et al., 1995). Therefore, to induce hypercholesterolemia or atherosclerosis in rats, cholesterol feeding is used with other additives,

Table 1 Effects of *Pleurotus eryngii* on plasma lipid profiles in hypercholesterolemic rats.

Parameter	NC (n = 10)	HC (n = 10)	HC + PE (n = 10)
Plasma parameters			
TC (mg/dl)	103.0 ± 5.3a	120.6 ± 10.3b	91.6 ± 3.8a
TG (mg/dl)	63.8 ± 11.3a	87.2 ± 12.8b	46.8 ± 5.9c
HDL-C (mg/dl)	37.6 ± 2.9	42.2 ± 2.2	37.6 ± 4.1
LDL-C (mg/dl)	17.0 ± 5.8a	20.8 ± 2.3a	7.8 ± 1.8b
VLDL-C (mg/dl)	48.4 ± 6.3	57.6 ± 7.8	46.2 ± 4.1
TL (mg/dl)	328.0 ± 9.8a	393.0 ± 4.8b	296.2 ± 14.4c
PL (mg/dl)	158.6 ± 9.8a	184.2 ± 11.0b	148.8 ± 9.8a
AST	63.4 ± 9.1	70.8 ± 8.4	58.8 ± 8.1
ALT	57.4 ± 10.9a,b	65.6 ± 3.0a	48.2 ± 7.2b
ALP	164.8 ± 7.7a,b	177.2 ± 9.4a	148.4 ± 12.8b
Fecal parameters			
Total lipid (U/l)	24.6 ± 3.2a	55.5 ± 4.5b	61.3 ± 4.2b,c
Cholesterol (U/l)	3.8 ± 0.6a	13.4 ± 0.8c	14.8 ± 1.3c

Values are given means ± S.D. Values in the same row that do not share a common superscript are significantly different at $p \leq 0.05$.

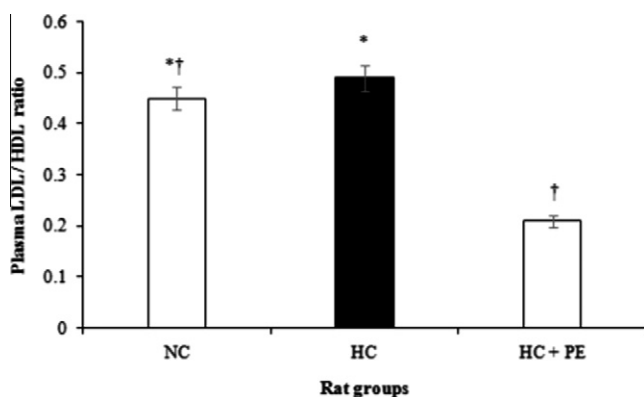


Figure 1 Effects of *Pleurotus eryngii* mushroom on plasma LDL/HDL ratio in hypercholesterolemic rats. Values represented are the means ± SD ($n = 10$). Different symbols indicate significant differences at $p \leq 0.05$.

including bile acids and propylthiouracil (an anti-thyroid drug), which increase intestinal absorption of cholesterol (Dolphin and Forsyth, 1983). However, in the present study, the addition of 1% cholesterol to the basal diet without bile acids and/or anti-thyroid drugs produced hypercholesterolemia in the rats, because cholesterol feeding itself increases bile acid secretion by approximately three to four-folds in rats (Uchida et al., 1996). The 17.09% increase in plasma cholesterol in the hypercholesterolemic rats in the present study was comparable with that reported by Bobek et al. (1995), who feed rats cholesterol (0.3%) diet with added bile acids (0.5%) and showed a 1.7-fold higher cholesterolemia in their cholesterol-fed rats than normal rats. In this experiment, feeding 5% mushrooms to hypercholesterolemic rats significantly repressed the increment of plasma cholesterol. The mechanism by which mushrooms reduce plasma lipoprotein levels in hypercholesterolemic rats is not clearly understood. Mushrooms contain the hypocholesterolemic agent mevinolin (Gunde-Cimerman et al., 1993), which may be involved in decreasing the activity of HMG-CoA reductase. Thus, feeding mushrooms may involve suppression of endogenous cholesterol biosynthesis by inhibiting HMG-CoA reductase activity.

3.3. Effect of *Pleurotus eryngii* on plasma enzyme profiles

Lower plasma AST, ALT, and ALP concentrations were observed in king oyster mushroom-fed hypercholesterolemic rats than normocholesterolemic rats (Table 1). No significant difference was observed in the activities of plasma AST in the NC, HC, or HC + PE rats groups. Plasma ALT and ALP activities were significantly higher in HC rats than in NC rats, whereas 5% mushroom-fed hypercholesterolemic rat revealed decreased plasma AST, ALT, and ALP activities by 16.95%, 26.52%, and 16.25%, respectively.

Due to the increasing frequency of antihyperlipidemic drug use and their common side effects, there is a need to identify natural products with few or no side effects. Thus, development continues for highly effective natural ingredients from food, such as mushrooms, which decrease hyperlipidemia (Alarcón et al., 2003). Previous studies have shown that AST and ALT are typically elevated following cellular damage as a result of enzyme leakage from the cells into blood (Alam et al., 2011). Therefore, the increased enzyme activities resulting from the mushroom treatment may prevent oxidative damage by detoxifying reactive oxygen species; thus, reducing hyperlipidemia.

3.4. Effect of *Pleurotus eryngii* on fecal total lipid and cholesterol

The fecal total lipid and cholesterol of the 5% *P. eryngii*-fed hypercholesterolemic rats significantly increased by 2.5 and 3.9-folds, respectively, compared with NC rats (Table 1). Thus, the decreased plasma cholesterol may have attributed to such a mechanism.

The higher level of plasma HDL-C indicates that more cholesterol from peripheral tissues was returning to the liver for catabolism and subsequent excretion. Plasma VLDL-C and TG contents in mushroom-fed hypercholesterolemic rats were lower compared with hypercholesterolemic rats. VLDL-C is the major transport vehicle for TG from the liver to extrahepatic tissues, whereas LDL-C is not secreted as such in the liver but seems to be formed from VLDL-C after partial removal of TG by lipoprotein lipase (Mayes, 1997). LDL-C became the prime carrier for cholesterol after feeding cholesterol to the

Table 2 Effects of *Pleurotus eryngii* on biochemical and electrolytes function in hypercholesterolemic rats.

Parameters	NC	HC	HC + PE
Albumin (g/dl)	3.3 ± 0.2	3.4 ± 0.3	3.0 ± 0.1
Total bilirubin (mg/dl)	0.1 ± 0.0	0.1 ± 0.0	0.1 ± 0.0
Direct bilirubin (mg/dl)	0.0 ± 0.0	0.0 ± 0.1	0.0 ± 0.0
Creatinine (mg/dl)	0.6 ± 0.0	0.7 ± 0.1	0.6 ± 0.1
Blood urea nitrogen (mg/dl)	16.2 ± 2.3 ^{a,b}	17.4 ± 3.2 ^a	14.2 ± 0.8 ^b
Uric acid (mg/dl)	2.2 ± 0.5 ^a	4.8 ± 1.4 ^b	2.5 ± 1.6 ^a
Glucose (mg/dl)	106.0 ± 4.7 ^{a,b}	118.2 ± 10.7 ^a	92.4 ± 6.9 ^b
Total protein (g/dl)	7.2 ± 0.2	7.3 ± 0.4	6 ± 0.3
Calcium (mg/dl)	10.5 ± 0.2	10.9 ± 0.8	10.3 ± 0.2
Sodium (mEq/l)	142.8 ± 0.8	144.8 ± 2.3	143.4 ± 1.5
Potassium (mEq/l)	4.8 ± 0.3 ^a	7.5 ± 1.7 ^b	5.0 ± 0.5 ^{a,b}
Chloride (mEq/l)	102.4 ± 1.5	103.0 ± 1.9	102.4 ± 0.9
Inorganic Phosphate (mg/dl)	6.9 ± 0.7 ^a	11.6 ± 1.6 ^b	7.3 ± 0.9 ^a
Magnesium (mg/dl)	2.7 ± 0.2	3.6 ± 0.8	2.8 ± 0.3

Values are given means ± S.D. Values in the same row that do not share a common superscript are significantly different at $p \leq 0.05$.

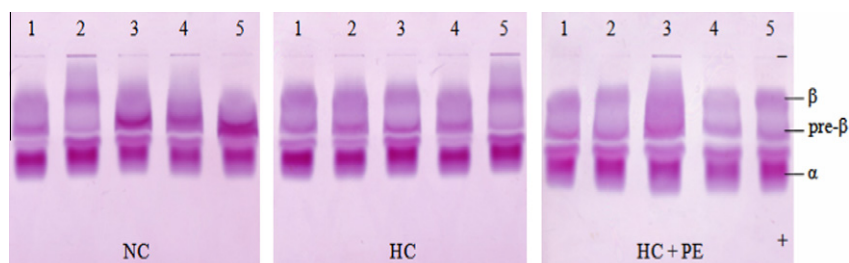


Figure 2 Separation of plasma lipoproteins by agarose gel electrophoresis. Lanes 1–5 represent the plasma lipoprotein fraction of five different rats from each group.

rats, leading to decreased VLDL-C and HDL-C contents in HC + PE rats.

3.5. Effect of *Pleurotus eryngii* on plasma biochemical and electrolyte function

The results of the plasma biochemical and electrolytes concentrations indicated that, blood urea nitrogen, uric acid, glucose, potassium, and inorganic phosphate in hypercholesterolemic rats significantly decrease by 18.39%, 47.92%, 21.83%, 33.33%, and 37.07%, respectively, compared with levels in mushroom-fed hypercholesterolemic rats. In contrast no significant difference was found for plasma albumin, total bilirubin, direct bilirubin, creatinine, total protein, calcium, sodium, and chloride levels among the normocholesterolemic, hypercholesterolemic, and mushroom-fed hypercholesterolemic rats (Table 2).

The glucose-lowering effect of propionate is associated with gluconeogenesis and the regulation of serum lipid levels (Yang et al., 2007). Reduction in plasma potassium, sodium, and chloride concentrations is one of the mechanisms of action of antihypertensive drugs, particularly diuretics (Jude et al., 2010). Diuretics act by diminishing sodium chloride reabsorption at different sites in the nephrons, thereby increasing urinary sodium chloride and water losses and consequently leading to decreased plasma levels of these electrolytes. Antonov et al. (1997) reported that plasma electrolyte contents increased significantly in hypertensive rats. Impaired function of Na, K-ATPase and the Na-H antiport, which is typical of

arterial hypertension, may promote an increase in plasma electrolytes.

3.6. Effect of *Pleurotus eryngii* on plasma lipoprotein fraction

The α -lipoprotein band was the fast-moving fraction and was located nearest the anode. The β -lipoprotein band was usually

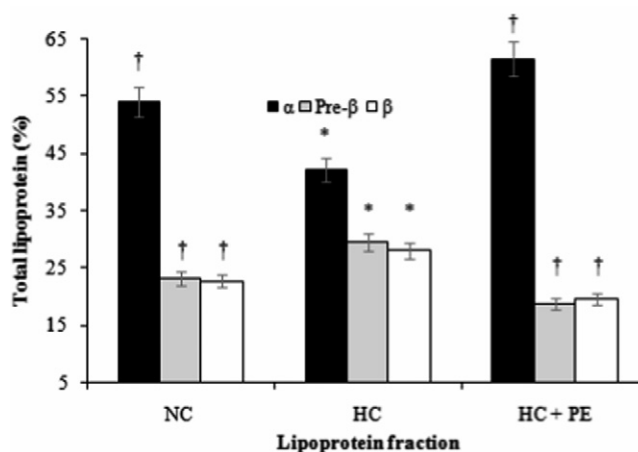


Figure 3 Effects of *Pleurotus eryngii* mushroom on the plasma lipoprotein fraction following agarose gel electrophoresis. Values represented are the means ± SD ($n = 10$). Different symbols indicate significant differences at $p \leq 0.05$.

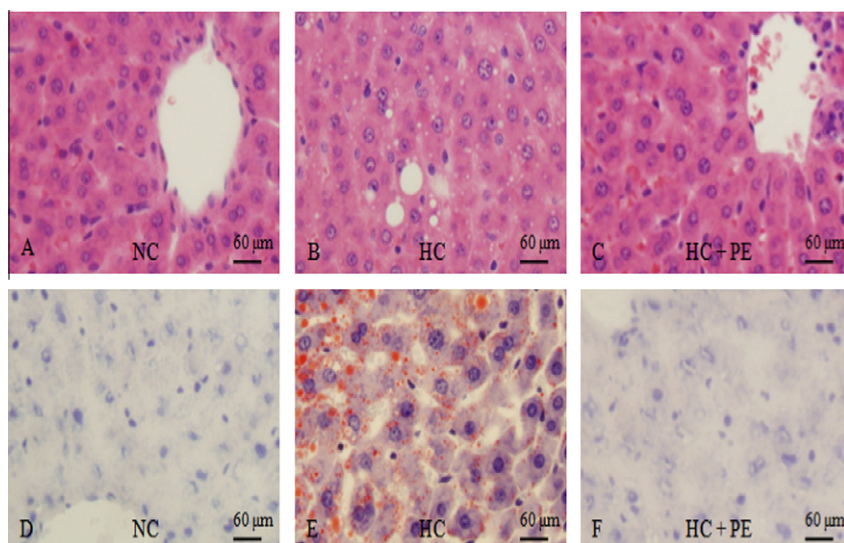


Figure 4 Effects of feeding *Pleurotus eryngii* mushroom on hepatocyte cells in hypercholesterolemic rats. A–C, hematoxylin–eosin stained photomicrographs at 40 \times ; D–F, photomicrographs of oil red O stain at 40 \times .

the most prominent fraction and was near the origin, migrating only slightly anodic to the point of application. The pre- β lipoprotein band migrated between α and β -lipoprotein (Fig. 2). The effects of feeding *P. eryngii* on the plasma lipoprotein fraction are presented in Fig. 3. The results indicated no significant difference in the lipoprotein fractions between NC and HC + PE rats. The results revealed that feeding 5% mushrooms significantly reduced plasma β -lipoprotein and pre- β lipoprotein but increased α -lipoprotein.

The hypocholesterolemic effect of mushrooms is mediated by the interplay of a complex mixture of substances (Bobek et al., 1996). A water-soluble gel-forming component of the fiber substance interacts with bile acids and affects micelle formation. Such substances might be interfering with the absorption of cholesterol in this manner.

3.7. Effect of *Pleurotus eryngii* on rat liver histology

The effect of *P. eryngii* on hepatocyte cells of hypercholesterolemic rats has been presented in Fig. 4. Liver tissues were stained with hematoxylin–eosin and oil red O. The hepatic cords were typically arranged and located in liver tissue near the central vein in the NC, HC, and HC + PE groups. Lipid droplets were observed only in the liver tissue of HC rats. This could be attributed to lipid accumulation in the hepatocyte cell cytoplasm.

Oxidized LDL induces the expression of scavenger receptors on the macrophage surface. These scavenger receptors promote the accumulation of modified lipoproteins, forming an early atheroma. The histological results indicated that the liver tissues of HC + PE rats were almost similar to NC rats and that the hepatic biosynthesis of cholesterol was suppressed, which might be due to a reduction in the activity of HMG-CoA (Keim et al. 1982). Hyperlipidemia is the leading risk factor for atherosclerosis, but the atherosclerotic pathological process could be slowed or reversed by reducing serum LDL, TGs, and PLs and increasing serum HDL. Several studies have demonstrated a protective effect of HDL in atherosclerosis and cardiovascular disease, whereas high levels of LDL constitute a risk factor. Excess LDL in the blood is deposited on the blood vessel walls

and becomes a major component of atherosclerotic plaque lesions, whereas HDL facilitates translocation of cholesterol from peripheral tissues, such as arterial walls, to the liver for catabolism (Li et al., 2010). Alam et al. (2011) observed that oyster mushrooms prevented the formation of atheromatous plaques and reduced the incidence and extent of atherosclerotic lesions in the aorta and coronary arteries as well as focal fibrosis in the myocardium of rabbits.

4. Conclusions

The present study demonstrated that feeding of 5% *P. eryngii* fruiting bodies significantly reduced the body weight and atherogenic lipid profiles, and it had no detrimental effects on the liver and kidney in hypercholesterolemic rats. On the basis of the results, it is suggested that mushroom intake has significant health benefits through the modulation of physiological functions that consist of various atherogenic lipid profiles in hypercholesterolemia. Therefore, *P. eryngii* can be a good source of nutrition that may also act as a prophylactic against hypercholesterolemia, hyperlipidemia and related complications, which are the risk factors of atherosclerosis.

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