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Protective effect of dietary apricot kernel oil supplementation on cholesterol levels and antioxidant status of liver in hypercholesteremic rats

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Abstract

Apricot kernel oil is a rich source of MUFA and PUFA, including mainly oleic (about 70%) and linoleic acids, respectively. In addition, apricot kernel oil could be considered as a good source of bioactive compounds such as tocopherols and phytosterols consisting mainly of the γ-isomer and β-sitosterol, respectively. Given to its high content of oleic acid, apricot kernel oil is considered as a healthy supplement in diet. In the present study, we investigated the effects of apricot kernel oil supplementation on cholesterol and malondialdehyde (MDA) levels and glutathione peroxidase (GPx) and catalase (CAT) activity in hypercholesteremic rats. Hypercholesteremia was produced by feeding rats with a semisynthetic diet that contained high cholesterol and cholic acid. A high cholesterol diet caused a decrease in CAT and GPx activity, while apricot kernel oil caused a significant activity increase of these enzymes ($P < 0.05$). The group fed with apricot kernel oil supplementation showed higher enzyme activities than sunflower oil groups irrespective of cholesterol ($P < 0.05$). Results of the present study indicate that apricot kernel oil causes improvement in liver antioxidant status of rats in comparison to sunflower oil which is a commonly consumed vegetable oil.

Key words: Apricot kernel oil, cholesterol, sunflower oil, malondialdehyde, catalase, glutathione peroxidase.

Introduction

Apricot, *Prunus armeniaca* L., is a member of the Rosaceae, subfamily Prunoideae. Very few apricot cultivars are grown commercially throughout the world. Further, cultivars grown in one region of a country would be virtually unknown outside that region. Turkey is the largest producer of apricot (538,000 metric tons/yr) in the world. The hard outer woody part of the pits represents about 35,000 metric tons/yr, kernels within the pits constitute 7,000 metric tons/yr [1]. The kernels are mainly used in production of cosmetics, medicines and scents, while pits are used as fuel. Percentage of kernel in the pit varies from 18.8 to 38.0. The reported oil content of kernels varies from 27.7 to 66.7%, majority of FAs being oleic (58.3–73.4%) and linoleic acids (18.8–31.7%) [2,3]. The contents of unsaturated FA (91.5–91.8%), saturated FA (7.2–8.3%), neutral lipids (95.7–95.2%), glycolipids (1.3–1.8%) and phospholipids (2.0%) of apricot kernel oil have been reported[2,3]. The kernel oil contains 11.8 mg/100 g campesterol, 9.8 mg/100 g stigmasterol and 177.0 mg/100 g sitosterol. Kernels contain thiamine, riboflavin, niacin, vitamin C, α-tocopherol,γ-tocopherol and δ-tocopherol [2, 3]. In organisms, endogenous and exogenous free radicals can damage lipids, proteins, carbohydrates and nucleic acids which themselves end up as new free radicals [4-6]. Among all biomolecules, lipids are the most sensitive molecules to free radical attacks. Double bonds in fatty acids form peroxide products by reacting with free radicals, and lipid radicals can be formed subsequently upon removal of electrons [7, 8]. As a result of lipid peroxidation, harmful degradative products [namely malondialdehyde (MDA)] can be formed in cell membranes. Malondialdehyde shows both mutagenic and carcinogenic effects by changing membrane properties [9, 10]. Organisms protect themselves from harmful effects of free radicals by antioxidant defense mechanisms. The antioxidant system involves both enzymatic and non-enzymatic agents. The first step in the enzymatic system is superoxide dismutase (SOD), which catalyzes the dismutation of superoxide anion ($O^{\cdot 2-}$) to H_2O_2. The conversion of H_2O_2 to H_2O by either glutathione peroxidase (GPx) or catalase forms the second step of enzymatic system. Superoxide dismutase and GPx enzyme activities and the balance between them is very crucial for protection against oxidative stress [11-13]. Lipid-soluble vitamin E is a non-enzymatic antioxidant which plays a significant role in the protection of cell membrane and against LDL cholesterol as well. This vitamin can reduce free radicals and most importantly breaks the chain reaction in lipid peroxidation [14, 15]. The measure of total antioxidant activity (AOA), which is the cumulative action of all the antioxidants present in plasma, provides an insight into the delicate balance *in vivo* between oxidants and antioxidants [16].

In the present study, our objective was to investigate the effects of apricot kernel oil and sunflower oil on liver and blood lipid peroxidation. Glutathione peroxidase (GPx) and catalase (CAT) activities and MDA levels were measured to shed some light on the effects of dietary oils on lipid peroxidation in rats.

Materials and Methods

Materials: All reagents and chemicals were purchased from Sigma (St. Louis, MO) and were the highest grade available.

Animals and diets: Twenty four male Wistar rats (45-d-old) weighing 378±28 g were purchased from Experimental Animals Unit, Faculty of Medicine, Inonu University, Malatya, Turkey. These animals were reared at 22±2°C, 55±5% humidity, under a 12/12 h light/dark cycle. Diet and water were provided *ad libitum*. Body weight was measured every week throughout the study. Rats were randomly assigned to 3 groups, 8 animals in each. All groups received an experimental diet (Table 1). Rats were fed either a basal diet containing 20% crude protein (CP) or 3974 kcal/kg metabolizable energy (ME) or the basal diet supplemented with 0.5% cholesterol, 0.25% cholic acid and 7% sunflower oil or 0.5% cholesterol, 0.25% cholic acid and 7% kernel oil. All procedures were performed according to the guidelines for the Care and Use of Experimental Animals Unit at the University. The experiments were performed according to the guidelines of the Committee for the Purpose of Control and Supervision of Experiments on Animals, Malatya, Turkey, and with the approval of the Institutional Animal Ethics Committee.

Blood sampling and tissue preparations: At completion of the diet study, the animals were sacrificed by cervical dislocation. Blood samples were removed from the heart and collected into tubes (vacuum-packed). Plasma was separated by low-speed centrifugation at 1,500 *g* at 4°C for 30 min and was immediately analysed. Cholesterol, phospholipids and triacylglycerols were determined using an autoanalyser (Giesse H902). The liver tissue for enzyme activity studies was homogenized (PCV Kinematica Status Homogenizator) in ice-cold phosphate buffered saline (pH 7.4). The homogenate was sonified with an ultrasonifier (Bronson Sonifier 450) by 3 cycles (20-s sonications and 40-s pause on ice). The homogenate was centrifuged (10,000 x *g*, 10 min, 4°C) and cell-free supernatant was subjected to enzyme assay immediately.

CAT assay: CAT activity was measured at 37°C by following the rate of disappearance of hydrogen peroxide (H_2O_2) at 240 nm (e_{240} = 40 M^{-1} cm^{-1}) [17]. One unit of catalase activity is defined as the amount of enzyme catalyzing the degradation of 1 mmol of H_2O_2 per min at 37°C and specific activity corresponding to transformation of substrate (in mmol) (H_2O_2) per min per mg protein.

GSH-Px assay: GSH-Px activity was determined in a coupled assay with glutathione reductase by measuring the rate of NADPH oxidation at 340 nm using H_2O_2 as the substrate [18]. Specific activity is given as the amount of NADPH (mmol) disappeared per min per mg protein.

Lipid peroxidation assay (MDA): The analysis of lipid peroxidation was carried out as described by Buege and Aust [19] with a minor modification. The reaction mixture was prepared by adding 250 ml homogenate into 2 ml reaction solution (15% trichloroacetic acid: 0.375% thiobarbituric acid: 0.25 N HCl, 1:1:1, w/v) and heated at 100°C for 15 min. The mixture was cooled to room temperature, centrifuged (10,000 x *g* for 10 min) and the absorbance of the supernatant was recorded at 532 nm. MDA standard used was 1,1,3,3-tetra ethoxy propane. MDA results were expressed as nmol g^{-1} wet tissue.

Determination of protein: Protein levels of the tissue samples were measured by the Bradford method [20]. The absorbance measurement was taken at 595 nm using a UV-VIS spectrophotometer. Bovine serum albumin (BSA) was used as the protein standard.

Statistical analysis: Results are expressed as mean±SD. Multiple comparisons of the significant analysis of variance were performed by Duncan's multiple comparison test. A p-value < 0.05 was considered as statistically significant. All data were analyzed with the aid of a statistical package program (SPSS 9.0 for Windows).

Table 1. Composition of experimental diets (g/100 g diet).

Ingredient	Control	Group I	Group II
Casein	20	20	20
Sucrose	15	15	15
Cellulose	5	5	5
Vitamin &mineral mixture**	5	5	5
Sunflower oil	7	7	-
Apricot kernel oil	-	-	7
Cholic acid	0.25	0.25	0.25
Cholesterol	-	0.5	0.5
DL-Methionine	0.25	0.25	0.25
Cornstarch	47	47	47

**The vitamin-mineral premix provides the following (per kg): all-*trans*-retinyl acetate, 1.8 mg; cholecalciferol, 0.025 mg; all-*rac*-α-tocopherol acetate, 12,5 mg; menadione (menadione sodium bisulfate), 1.1 mg; riboflavin, 4.4 mg; thiamine (thiamine mononitrate), 1.1 mg; vitamin B-6, 2.2 mg; niacin, 35 mg; Ca-pantothenate, 10 mg; vitamin B-12, 0.02 mg; folic acid, 0.55 mg; *d*-biotin, 0.1 mg. manganese (from manganese oxide), 40 mg; iron (from iron sulfate), 12.5 mg; zinc (from zinc oxide), 25 mg; copper (from copper sulfate), 3.5 mg; iodine (from potassium iodide), 0.3 mg; selenium (from sodium selenite), 0.15 mg; choline chloride, 175 mg.

Results

Food intake on body weight: There were no significant differences in body weights among the rats fed with different diets. No mortality was recorded in any group during the experimental period.

Effect of apricot kernel oil on serum lipid profile: The effect of apricot kernel oil supplementation on serum lipid profile in hypercholesteremic rats is summarized in Table 2. Keeping the rats on a high-cholesterol diet significantly increased the TC, TG and LDL levels in serum of Group I and II as compared to control group (p < 0.05). The serum levels of TC and LDL increased in apricot kernel oil supplementation Group II by 59% and 166% respectively, while HDL level decreased (30%) significantly (p < 0.05). The hepatic TG level remained unaffected compared to control group. Similarly to the results of TC, TG and LDL also increased by 19, 52.5 and 97.8%, respectively, and HDL level was reduced by 11.5% in sunflower oil supplemented Group I (p < 0.05).

Effect of apricot kernel oil on liver oxidative status: Table 3 shows that in Group II the MDA level of liver was significantly (p < 0.05) decreased compared to control and Group I, whereas a high cholesterol diet - sunflower oil (Group I) - showed enhanced

Table 2. Changes in the serum lipid levels of rats fed with different diets.

Parameter	Control	Group I	GroupII
Total cholesterol (mg/dl)	84.97±6.88	101.88±17.55	135.40±27.31
Triglycerides (mg/dl)	59.50±14.48	90.75± 9.74	56.50±7.33
HDL cholesterol (mg/dl)	31.97±3.05	28.29± 2.21	22.25±5.19
LDL cholesterol (mg/dl)	35.28±12.61	69.79±24.34	93.70±18.43

Control,normal diet;Group I, high cholesterol diet-sunflower oil;Group II, high cholesterol diet-apricot kernel oil. Results are mean ± SD for eight animals; one-way ANOVA; Duncan's multiple comparison test. Values that have a different significantly($P < 0.05$) with each other.

Table 3. Effect of apricot kernel oil feeding on concentrations of malondialdehyde (MDA), catalase (CAT) and glutathione peroxidase (GPx) in liver (n= 8).

Parameter	Control	Group I	Group II
CAT (U mg^{-1}protein)	778.61±11.35	642.22±86.18	819.97±69.94
GPx (U mg^{-1}protein)	4.24±0.32	4.33±1.09	6.25±1.06
MDA(nmol g^{-1} wet tissue)	63.04±2.21	73.37±5.36	55.14±8.24

Control, normal diet;Group I, high cholesterol diet-sunflower oil;Group II, high cholesterol diet-apricot kernel oil. Results are mean ± SD for eight animals; one-way ANOVA; Duncan's multiple comparison test. Values that have a different significantly($P < 0.05$) with each other.

levels of lipid peroxidation. Table 3 shows the CAT activity of liver tissue. CAT activity of liver tissue was significantly ($p < 0.05$) decreased by 17% after 45 days of feeding high cholesterol diet-sunflower oil (Group I)- relative to control group. Feding high cholesterol diet - apricot kernel oil (Group II) - however, resulted in a significant ($p < 0.05$) increase in CAT activity. GPx activity level of liver tissue was higher (45%) in apricot kernel oil supplemented hypercholesteremic rats (Group II) than sunflower oil supplemented hypercholesteremic rats (Group I) and control group ($p < 0.05$).

Discussion

The present study investigated the effect of apricot kernel oil on the cholesterol metabolism and antioxidative status in the rats fed a hypercholesteremic diet. The results suggested that liver antioxidative effect of apricot kernel oil supplements was very potent in cholesterol-rich diet fed rats. The apricot kernel oil, which contained γ-tocopherol was the most abundant, with an average concentration of 475.11 mg kg^{-1} of oil, contributing 93.6% to the total, followed by α-tocopherol (19.51 mg kg^{-1} of oil), δ-tocopherol (12.64 mg kg^{-1} of oil) and a small amount of β-tocopherol (0.38 mg kg^{-1} of oil) [2, 3]. γ-Tocopherol is somewhat less potent in donating electrons than α-tocopherol and thus the former is slightly less powerful in antioxidant activity [21]. The antioxidative effects of apricot kernel oil could be related to its tocopherol content. Indeed, previous reports have shown that those tocopherols are the main antioxidant components in apricot oil [2, 3, 22]. In a concomitant cross-sectional study of Swedish and Lithuanian middle-aged men, Kristenson *et al*. [23] found that plasma γ-tocopherol concentrations were twice as high in the Swedish men, but the Swedish had a 25% lower incidence of CVD-related mortality than the Lithuanian. On 45-day feeding of the 0.5% of cholesterol diet, plasma levels of TC and LDL increased significantly ($P < 0.05$) (Table 1), while HDL was reduced ($p < 0.05$) and TG was not affected in apricot kernel oil supplemented hypercholesteremic rats (Group II) compared to control group (Table 2). Similarly, in sunflower oil supplemented hypercholesteremic rats (Group I) the levels of TC, TG and LDL were also increased, while HDL was reduced ($p < 0.05$) (Table 2). Previous studies are in agreement with our results, showing that the palm oil (PO), *Canarium schweinfurthii* oil (CSO) and *Dacryodes edulis* oil (DEO) decrease the HDL-cholesterol levels in serum [24]. In other studies fish oil diet decreased the plasma total cholesterol and HDL- cholesterol levels [25, 26]. We examined the effects of apricot kernel oil on some of the hepatic oxidative parameters of the experimental animals. The results demonstrated that oral administration of apricot kernel oil prevented the high-cholesterol diet-induced elevation of MDA and resulted in a significantly ($p < 0.05$) decrease in MDA content of liver homogenates (Table 3). However, recent studies indicate that PUFA are more susceptible to lipid peroxidation than SFA. Lipid peroxidation usually results in decreasing membrane fluidity, cell injury and may cause the formation of atherosclerotic plaques [27, 28]. The estimation of free radical activity was done through the determination of malondialdehyde (MDA) which is a by-product of lipid peroxidation [29]. As a result of the degradation of lipid peroxides, MDA forms and is used as an indicator of lipid peroxidation [30]. Halliwel and Chirico [31] demonstrated the higher stability of saturated and monounsaturated oils in lipid peroxidation than that of polyunsaturated fatty acids. There are numerous harmful effects of MDA reported [32, 33]. Cross linking with the membrane components, MDA causes inactivation of enzymes and receptors in membranes and thus changes membrane properties. Malondialdehyde also causes mutations by reacting with guanine nucleotide in DNA [34, 35]. A cholesterol-rich diet brings about remarkable modifications in antioxidant defense mechanisms. Studies have shown that hypercholesterolemia diminishes the antioxidant defense system and decreases the activities of SOD and CAT, elevating the lipid peroxide content [36] SOD, CAT, GPx and GR constitute a mutually supportive team of defense against reactive oxygen species (ROS) and preventing lipid peroxidation[37]. This radical modifies proteins and DNA, damages cellular membranes of mitochondria, nuclear envelop and endoplasmic reticulum [38-40]. CAT is a hemoprotein, localized in the peroxisomes and catalyses the decomposition of H_2O_2 to water and oxygen. GPx is a selenoenzyme, present predominantly in liver and catalyses the reaction of hydroperoxides with reduced glutathione to form glutathione disulphide (GSSG) and the reduction product of the hydroperoxide [41]. Increased activity of these antioxidant enzymes results in decreased formation of hydroxyl radical (OH^-) [42, 43]. In the present study, the activity of CAT in the liver of high cholesterol diet– sunflower oil (Group I) - was significantly ($p < 0.05$) decreased and no effect on GPx was observed compared with those of control rats. The activities of GPx and CAT in the liver of high cholesterol diet – apricot kernel oil (Group II) were significantly ($p<0.05$) increased compared with those of control rats (Table 3). Supplementation of apricot kernel oils feed of the rats fed with cholesterol rich-diet elevated the activities of CAT and GPx liver significantly ($p < 0.05$). These results suggested that tocopherol and phytosterol compounds present in the apricot kernel oils could improve the efficiency of superoxide anion to hydrogen peroxide due to increased GPx and CAT activity, which in turn detoxifies hydrogen peroxide and converts it to lipid hydroperoxides to nontoxic substances.

In conclusion, apricot kernel oil could be utilized successfully as a source of edible oils, as it is a rich source of MUFA and PUFA, including mainly oleic (about 70%) and linoleic acids, respectively. Al these have been associated with beneficial health effects besides the lower content of SFAs in this oil. Hence, apricot kernel oil may be used for the production of salad or frying oils. Apricot kernel oil contains higher amounts of the di-and tri-unsaturated forms of TAG, containing mainly oleic acid. In addition, apricot kernel oil could be considered as a good source of bioactive compounds such as tocopherols and phytosterols, consisting mainly of the γ-isomer and β-sitosterol, respectively. We found that in this model of experimental atherogenesis, the supplementation with apricot kernel oil exhibited an anti-hyperlipidemic action, reduced the lipid peroxidation process and enhanced the antioxidant defense system. Hence, this study may be important in terms of the potential nutritional, functional and economic utility of apricot kernel oils as a new source.

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