Study on Biological Activity of *Pinus Armandi Franch* Seed Oil

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Abstract: *Pinus armandi franch is a unique specialty plant in China and its seed oil can be a nice resource of linoleic acid (LA) with a content of 63% of the total fatty acid. LA has many pharmaceutical applications and the absence of LA in the diet is chargeable for the generation and the development of different anomalous diseases. The hypolipidemic effect of Pinus armandi franch seed oil (PAFO) was studied in the work. Fifty rats were involved in the study with ten rats in each group. One group is normal, whereas the others were hypercholesterolemic. Hypolipidemic effects were investigated in both plasma and tissues. After 42 days of feeding, the arteriosclerosis index (AI) decreased by administration of PAFO in comparison with the control group fed with high-fat diet. In both normal and hypercholesterolemic condition plasma cholesterol, low-density lipoprotein, and triacylglycerols were reduced by feeding the rats with PAFO. However, the high-density lipoprotein levels increased with the administration of PAFO. It demonstrated PAFO could clear up cholesterol that could form deposits in the artery walls, promote fat metabolism, prevent metabolic disorders of lipoproteins.*

Keywords: *Pinus armandi franch, Seed oil, Linoleic acid, Hypolipidemic effect.*

Introduction

Linoleic acid (LA) is a functional polyunsaturated fatty acid and it is attracting augmenting consideration because of their close connection to human health. LA has some advantageous results regarding coronary heart diseases [6]. Hyperlipidemia is the major risk factors of heart disease such as atherosclerosis, stroke, and death. Hypertension diabetes mellitus, dyslipidemia, and obesity are important risk factors for coronary heart disease (CHD) and many medicines for the diseases are with noticeable side effects [18]. In the last years some authors have emphasized the role of Mediterranean diet in precaution of some diseases including atherosclerosis. These authors attributed the positive effect of such diets to their low saturated and high polyunsaturated fatty acids (PUFA) content. Diets low in saturated fatty acids and high in polyunsaturated fatty acids are efficient in controlling blood lipid levels [2, 3, 10]. Correlations in lipid synthesis in mammalian tissue have received much attention and enzymatic steps in approaches for the net activities of general triglycerides and phosphatides have been certainly known [14, 15]. When a typical diet high in saturated fat is replaced with a southern Mediterranean-type diet, plasma cholesterol levels were decreased [1]. Experiments *in vitro* and on laboratory animals have demonstrated that low density

lipoprotein (LDL) cholesterol oxidation is restrained by olive oil constituents [13, 17]. Investigations of hyperlipidemic subjects have demonstrated that dietary oils impact lipid peroxidation and antioxidant levels, and lead to favorable changes in the lipid status [8, 12]. Zevenbergen et al. [22] investigated that the amount of linoleic acid required to prevent undesirable effects of C18 trans fatty acids. The amount of essential fatty acids in the diet also affected the mitochondrial composition and function [5, 19, 20].

Animal experiments and clinical studies indicated that there was the near relationship between hyperlipidemia and lipid peroxidation, but the accurate mechanism was not fully understood [9]. It was proved by clinical medicines and dietary regulation to reduce blood-fat levels, can reduce the incidence of coronary heart disease and atherosclerosis risk [11]. Most lipid-lowering drugs like cholestyramine, colestipol, statins decrease serum triglycerides and LDL-cholesterol to various extents and increase high-density lipoprotein (HDL) cholesterol levels. However, these drugs have also numerous and significant side effects, not long-term use [4].

Functional foods are being given more consideration in recent years due to their continuing, safe and effective therapeutic effects. Despite the fact that several works affirming that diets rich in polyunsaturated fatty acids n-6 reduce total cholesterol (TC), HDL and LDL concentrations, the impact of dietary PUFA n-6 on lipoproteins has also caused scientific debate [16]. Therefore, more studies should be done to clarify all these controversial data about the influence of monounsaturated fatty acid (MUFA) and PUFA on lipid profile in humans.

Pinus armandi franch seed oil (PAFO) is an inexpensive source of several polyunsaturated fatty acid types, thus it could be widely used for isolation and purification of PUFA [7]. LA has been associated with improved achievement in experimental some animals colitis, and is capable of creating an anti-inflammatory consequence without compromising resistance to infection. The diet can be improved by modifying the amount and the type of fat ingested, which also conditions the lipid profile, without side effects and a much lower cost than drugs [21]. As the hypolipidemic effects of using natural food control have been proven to be safe and eligible, we believe that the development of health food products such as natural products with the function of lowering the blood fat will become a significant research direction.

PAFO was tested in this work and assessed them for potential use as a new oil crop to provide a dietary amplification by investigating the effects of the seed oil in the diet on triglycerides, plasma cholesterol, and atherosderosis index of rats. This work reported the hypolipidemic activities of the essential oil produced by the *Pinus armandi franch* seed. It is significant that the hypolipidemic of biological activity of PAFO is reported here for the first time.

Materials and methods

Preparation of seeds and purification of LA

Pinus armandi franch seeds were supplied by Painuo Biotechnology Co. Ltd, China. All biochemical reagents and assay kits for biochemical variables were obtained from Nanjing Jiancheng Bioengineering Institute, China. All other reagents are analytical grade from Sinopharm Chemical Reagent Co., Ltd. *Pinus armandi franch* seed oil was prepared by means of supercritical CO_2 extraction and LA was purified from *Pinus armandi franch* seed oil by means of molecular distillation.

Experimental animals

Male Sprague-Dawley rats, weighing 180 ± 10 g, were grouped into five groups (ten rats in each group) by random distribution and housed in individual cages, under a 12 h light/dark

cycle, 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 PAFO and high-fat diets containing 10 wt% lard, 10 wt% egg yolk powder, 5 wt% sucrose, 1.5 wt% cholesterol, 0.2 wt% bile salt and 73.3 wt% basic diets. The animals were fed for a total period of 42 days. Animals were given a fresh diet daily, and leftover diets were weighed and discarded. The gain in body weight of animals was monitored at regular intervals. The animals had free access to food and water throughout the study. After 42 days of feeding, rats were fasted overnight and sacrificed under diethyl ether anaesthesia. Blood was drawn by cardiac puncture, and serum was separated by centrifuging at 2000 rpm for 20 min at 4 ℃. The liver was removed and rinsed thoroughly with ice-cold saline, blotted, weighed and stored at –20 ℃ until analysed. Heart and adipose tissues were also removed, washed in ice-cold saline, blotted and stored at –20 ℃ until analysed.

Analysis of plasma lipids

Six items of blood lipids were measured by rat serum that included triglycerides (TG), TC, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), apolipoprotein A (ApoA), and apolipoprotein B (ApoB). Meanwhile, arteriosclerosis index AI = LDL-C/HDL-C was calculated. TG and TC were determined by enzyme colorimetry and LDL-C was determined by the method of polyethylene sulfuric acid precipitation. HDL-C was determined by phosphotungstic acid-magnesium precipitation. 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.

Statistical analysis

Results are represented as means ± standard deviation for each group. One way ANOVA was used for analyzing the data followed by a post hoc Tukey test to compare the control and treatment groups; p-values < 0.05 were considered as statistically significant. SPSS statistical software package version 22.0 was utilized for statistically analyzing the data.

Results and discussion

Effect of PAFO on body weight

The fat level was kept constant at 10% in all the dietary groups. Rats were in good health throughout the study and the amount of diet consumed in the different groups was comparable. The effect of feeding dietary lipids on body weight gain of normal and hypercholesterolemic rats is shown in Table 1.

Table 1. Effect of PAFO on weight of rats

Group	The dose given to the sample, mg/kg·d	*n*	Weight, g	
			Day 0	Day 42
NG	—	10	173.36 ± 9.0^{a}	346.23 ± 26.62^{a}
MG	—	10	174.19 ± 9.64^{a}	302.44 ± 17.01^{b}
LPAFO	100	10	172.21 ± 6.94^{a}	304.13 ± 16.52^{b}
MPAFO	160	10	173.21 ± 10.04^{a}	306.17 ± 23.61^{b}
HPAFO	200	10	170.18 ± 5.42^{a}	309.08 ± 9.71^{b}

Values are means ± SD (n = 10 rats). Values not sharing a common superscript within a row are statistically significant (p < 0.05). NG, normal diet; MG, high-fat diet; LPAFO, high-fat

diet with low dose of PAFO; MPAFO, high-fat diet with middle dose of PAFO; HPAFO, high-fat diet with high dose of PAFO.

After 6 weeks, rats in the high-fat diet group showed the lowest weight gain. There was no significant difference in the food intake of the different groups in both normal and hypercholesterolemic rats. However, there was significant difference between the body weight gain of normal and hypercholesterolemic group ($p < 0.05$). There was no significant difference between the body weight gain of the group fed with high-fat diet and the group fed with PAFO in the normal case. On the other hand, the body weight gain decreased on feeding the rats with PAFO in hypercholesterolemic condition.

Effect of PAFO on organ index

The effect of dietary lipids on organ weights in normal and hypercholesterolemic cases is illustrated in Table 2. In the normal case there was a significant decrease ($p < 0.05$) in the weight of the liver, but no significant change in the weight of the heart and kidney on treating the rats with experimental diets in comparison with the control diet. Similar results were also found in hypercholesterolemic condition.

Organ index can reflect the physiological status of rats. Long-term consumption of high-fat diet can lead to the accumulation of lipid and pathological changes in the viscera of the tested animals which can be reflected by the ratio of organ. It could be seen from Table 2, there were significant differences in the liver weight/body weight between the low-dose and high-dose PAFO groups ($p < 0.05$). Table 2 indicated that the liver of hyperlipidemia rats was significantly increased due to excessive fat intake and the low dose group had no significant effect on reducing the liver weight/body weight ratio. The liver weight/body weight ratio of the medium and high dose PAFO group was significantly reduced ($p < 0.05$). The liver indexes of rats in the three dose groups were lower than that in the high-fat group by gavage PAFO and it was without causing liver enlargement. At present, many lipid-lowering drugs can cause the liver to swell obviously and PAFO appears to be both superior and safer by contrast.

Table 2. Effect of PAFO on organ index of rats

Group	*n*	Heart index, g/100g bw	Liver index, g/100g bw	Spleen index, g/100g bw	Kidney index, g/100g bw
NG	10	0.392 ± 0.039^{a}	3.644 ± 0.278^{a}	0.202 ± 0.016^{b}	0.789 ± 0.035^{a}
MG	10	0.383 ± 0.058^{a}	4.382 ± 0.285^{c}	0.163 ± 0.013^{a}	0.822 ± 0.027^{a}
LPAFO	10	0.406 ± 0.049^{a}	4.346 ± 0.258^{c}	0.197 ± 0.023^{b}	0.834 ± 0.057^{a}
MPAFO	10	0.394 ± 0.033^{a}	3.992 ± 0.191^{b}	0.208 ± 0.038^{b}	0.816 ± 0.048^{a}
HPAFO	10	0.386 ± 0.031^{a}	3.744 ± 0.211^{a}	0.213 ± 0.056^{b}	0.828 ± 0.077^{a}

In terms of the spleen weight/rat weight, there was a significant difference between the high-fat diet group and the normal control group. There was no significant difference among the low, medium and high dose group of PAFO and the normal group ($p > 0.05$). The ratio of the heart weight/rat weight and the kidney weight/body weight showed no significant difference between the groups ($p > 0.05$). The experiment showed that PAFO could reduce the ratio of the liver weight/rat weight on hyperlipidemia rats and improve the immune function of hyperlipidemia animals. PAFO could be used as a new type of lipid-lowering drug. It has many advantages and it is safer.

Effect of PAFO on lipid parameters in plasma

The blood lipid value was determined and the results were shown in Table 3. The TG value of the model group was significantly higher ($p < 0.05$) than that of the low, medium and high dose PAFO group and the normal group. There was no significant difference between the normal group and the high-dose PAFO group ($p > 0.05$). The results showed that PAFO could significantly reduce the value of serum TG in hyperlipidemic rats and it showed a dose-effect relationship. The total cholesterol values of the experimental dose groups were shown in Table 3, and the difference between the model group and the normal group was significant ($p < 0.05$). The difference in cholesterol level between the medium-dose and high-dose PAFO group was not significant ($p > 0.05$). The results showed that a certain dose of PAFO could reduce the TC content in serum of hyperlipidemic rats and there was a dose-effect relationship.

The value of HDL-C in the model group was significantly lower ($p < 0.05$) than that of the normal group after the rats were fed with high-fat forage. The value of HDL-C of the low, medium and high dose groups was significantly higher than that of the model group ($p < 0.05$). These results suggest that PAFO could increase the serum HDL cholesterol level in hyperlipidemic rats and it showed a dose-effect relationship. The LDL-C value of the model group was significantly higher than that of the normal group ($p < 0.05$) after the rats were fed with high-fat forage. The LDL-C value of the normal group was not significantly higher than that of the high dose PAFO group ($p > 0.05$). Therefore, a certain dose of PAFO has a dose-dependent effect on lowering LDL cholesterol in serum of high-fat rats.

Table 3. Blood lipid levels in rats fed with different doses of PAFO

Group	*n*	TG, mmol/L	TC, mmol/L	HDL-C, mmol/L	LDL-C, mmol/L
NG	10	0.472 ± 0.073^{a}	1.192 ± 0.061^{a}	0.883 ± 0.062^{d}	0.613 ± 0.048^{a}
MG	10	1.017 ± 0.078^{c}	2.915 ± 0.138^{d}	0.613 ± 0.074^{a}	0.841 ± 0.064^{c}
LPAFO	10	0.723 ± 0.119^{b}	2.013 ± 0.133^{c}	0.629 ± 0.053^{a}	0.714 ± 0.083^{b}
MPAFO	10	0.712 ± 0.074^{b}	1.432 ± 0.119^{b}	0.704 ± 0.088^{b}	0.696 ± 0.038^{b}
HPAFO	10	0.532 ± 0.088^{a}	1.334 ± 0.078^{ab}	0.879 ± 0.042^{d}	0.601 ± 0.019^{a}

Effect of PAFO on apolipoprotein and arteriosclerosis index in rats

The effect of PAFO on ApoA and ApoB of normal and hypercholesterolemic rats is shown in Table 4. It could be seen that the ApoA value of the medium-dose and high-dose PAFO group was significantly higher than that of the model group ($p < 0.05$) and that of the high-fat model group was significantly lower than the normal group ($p < 0.05$). The results showed that PAFO could increase ApoA level in hyperlipidemia rats and the dose-effect relationship was observed. The ApoB value of each dose of PAFO group was significantly lower than that of the model group ($p < 0.05$) after the rats were fed with high-fat forage. The ApoB value of the normal group was significantly lower than that of the model group ($p < 0.05$). This indicated that PAFO had the effect of increasing ApoA and decreasing ApoB in rat serum and it had a dose-dependent effect.

After the rats were fed with high-fat forage, the arteriosclerosis index AI (LDL-C/HDL-C) of the model group was significantly higher than that of the normal group ($p < 0.05$). The AI value on high dose group of PAFO was significantly lower than that of the model group ($p < 0.05$). The results showed that PAFO could significantly improve lipid metabolism and inhibit the formation and development of atherosclerosis in rats.

Table 4. Apolipoprotein and arteriosclerosis index of rats fed with different doses of PAFO

Group	*n*	ApoA, g/L	ApoB, g/L	Serum AI value (LDL-C/HDL-C)
NG	10	0.242 ± 0.025^{b}	0.512 ± 0.023^{a}	0.701 ± 0.036^{a}
MG	10	0.181 ± 0.013^{a}	0.742 ± 0.041^{d}	1.487 ± 0.303^{cd}
LPAFO	10	0.193 ± 0.017^{a}	0.681 ± 0.024^{c}	1.282 ± 0.109^{bd}
MPAFO	10	0.241 ± 0.024^{b}	0.566 ± 0.023^{b}	1.112 ± 0.038^{b}
HPAFO	10	0.253 ± 0.036^{b}	0.513 ± 0.082^{a}	0.613 ± 0.123^{a}

Effect of PAFO on serum oxidation

The effect of PAFO on serum oxidation in rats is shown in Table 5. Compared with normal group, serum GSH-Px activity in model group rats decreased significantly ($p < 0.05$). It is explained that the feeding of PAFO could significantly improve the serum GSH-Px activity in rats fed with high-fat forage. It showed a dose-effect relationship. After the rats were fed with high-fat forage, serum TAC activity in the model group decreased significantly ($p < 0.05$) compared with that in the normal group. Serum TAC activity in the three dose groups was significantly higher than that in the model group ($p < 0.05$). Table 5 showed that after the rats were fed with high-fat forage, SOD activity in serum of rats in the high-fat model group decreased significantly ($p < 0.05$) compared with that in the normal control group and serum SOD activity in the medium and high dose group was significantly increased compared with that in the model group ($p < 0.05$). After the rats were fed with high-fat forage, MDA levels in the serum of rats in the model group increased significantly ($p < 0.05$), and MDA levels in the serum of the three dose groups decreased significantly ($p < 0.05$). It indicated that PAFO could significantly reduce MDA content in serum of high-fat rats and had a certain inhibitory effect on lipid peroxidation in serum of rats.

Table 5. Effects of different doses of PAFO on serum oxidation in rats

Group	*n*	GSH-Px, U/mL	TAC, U/mL	SOD, U/mL	MDA, nmol/mL
NG	10	2191.43 ± 88.0^{c}	7.25 ± 0.47^{c}	362.61 ± 7.69^{c}	10.67 ± 0.87^{b}
MG	10	1687.79 ± 63.02^{a}	4.97 ± 0.21^{a}	305.87 ± 14.77^{a}	13.46 ± 0.70^{c}
LPAFO	10	1893.02 ± 84.98^{b}	5.72 ± 0.22^{b}	325.0 ± 11.03^{ab}	11.38 ± 0.81^{b}
MPAFO	10	2129.22 ± 92.21^{c}	7.14 ± 0.43^{c}	345.3 ± 12.13^{bc}	11.03 ± 0.72^{b}
HPAFO	10	2159.49 ± 21.23^{c}	7.32 ± 0.18^{c}	366.03 ± 10.01^{c}	9.01 ± 0.43^{a}

Effect of PAFO on liver oxidation

The effect of PAFO on liver oxidation of normal and hypercholesterolemic rats is shown in Table 6. The GSH-Px activity of liver tissue in model group rats significantly lower than the normal control group ($p < 0.05$) after the SD male rats were fed with high fat forage for six weeks and normal control group was significantly higher than that of middle and low PAFO group ($p < 0.05$). These results indicated that PAFO could significantly increase the activity of GSH-Px in liver tissue on rats fed with high fat forage and it showed a dose-dependent effect. TAC activity in liver tissues of model group was significantly lower than that of normal control group ($p < 0.05$). It had statistical significance. The comparison of SOD activity in the livers showed that the SOD activity in the liver of rats in the high-fat model group was significantly increased ($p < 0.05$). MDA level in rat liver of the model group increased significantly ($p < 0.05$), while MDA levels in the medium-dose PAFO group were no longer significantly different from those in the normal group ($p < 0.05$) after the rats were fed with

high-fat forage. These results suggested that PAFO could reduce MDA content in liver tissues of the rats fed with high-fat forage and inhibit lipid peroxidation in liver tissues of rats to some extent.

Table 6. Effects of different doses of PAFO on liver oxidation in rats

Group	*n*	GSH-Px, U/mg pro	TAC, U/mg pro	SOD, U/mg pro	MDA, nmol/mg pro
NG	10	693.41 ± 17.21^{c}	22.51 ± 1.94^{b}	28.47 ± 2.9^{b}	1.18 ± 0.07^{b}
MG	10	614.59 ± 16.61^{a}	15.94 ± 2.04^{a}	22.14 ± 2.01^{a}	1.57 ± 0.06^{d}
LPAFO	10	653.38 ± 17.11^{b}	17.23 ± 0.97^{a}	24.32 ± 1.41^{a}	1.36 ± 0.09^{c}
MPAFO	10	658.11 ± 15.39^{b}	20.76 ± 1.13^{b}	27.59 ± 0.79^{b}	1.21 ± 0.14^{b}
HPAFO	10	698.49 ± 24.44^{c}	22.01 ± 1.41^{b}	29.79 ± 1.18^{b}	1.03 ± 0.08^{a}

Conclusion

In conclusion, the present study has shown that the nutritionally enriched LA oil could be prepared by means of molecular distillation from *Pinus armandi franch* seed oil. This oil contained nutraceutical molecules such as γ-linolenic acid, tocotrienols and phytosterols. PAFO could reduce LDL cholesterol in addition to lowering triglyceride levels in serum and liver of experimental rats. Therefore, PAFO can be exploited as reduced calorie fat with antiobesity potential and also hypolipidemic potential for promoting cardiovascular health. It could be a safe source of essential fatty acids in feed and food formulations.

Acknowledgements

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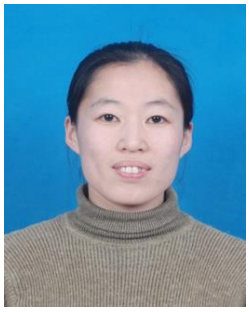

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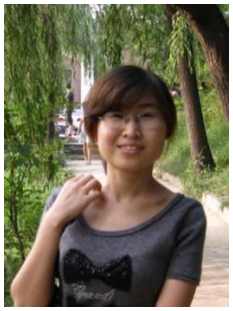

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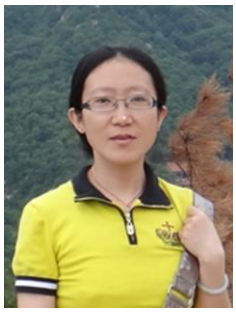

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