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Full Length Research Paper

Antioxidant activities, *a*-glucosidase inhibitory *in vitro* and effects of *Lysimachia paridiformis* Franch. var. *stenophylla* Franch. on alloxan-induced diabetic mice *in vivo*

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To investigate the antioxidant and *α*-glucosidase inhibitory activities *in vitro* and effects of *Lysimachia paridiformis* Franch. var. *stenophylla* Franch (LPF) on alloxan-induced diabetic mice *in vivo*, 96-microplate-based method was used to assay *α*-glucosidase inhibitory activity, and antioxidant activity was determined by the method of DPPH, ABTS and FRAP. The model of diabetic mice was induced by alloxan. Extract of *n*-BuOH (LPFBU, IC_{50} = 20.00 µg/ml) had the highest *α*-glucosidase inhibitory activity in three extracts. LPFBU had the highest antioxidant activity (DPPH: IC_{50}=15.69 µg/ml, ABTS: IC_{50}=11.19 µg/ml and FRAP = 756.40±21.21 µmol TE/g respectively). Compared with diabetic control mice, oral administration of EtOAC extract (LPFEA, 1000,500 mg/kg, respectively) significantly decreased fasting blood glucose, serum TC, TG and MDA levels in diabetic rats, while it significantly increased liver glycogen content and increased SOD levels without statistical significance. The results indicated that LPFBU and LPFEA exhibited the higher activity of *α*-glucosidase inhibitory and a certain antioxidant activity. LPFEA has some good antidiabetic activity and good effects on diabetes complications. It may be useful in prevention and treatment of diabetes and diabetes-associated diseases.

Key words: *Lysimachia paridiformis* Franch. var. *stenophylla* Franch., *α*-glucosidase inhibitory activity, antioxidant activity, diabetes.

INTRODUCTION

Oxidative stress has been suggested to be a contributory factor in development and complication of diabetes (Pidaran et al., 2006). Normally, body organisms are able to control and counter the free radical mediated oxidative damage. However, if radicals produce excessively or are cleared off slowly in the body, they leading to oxidative damage related diseases such as cancer, neuro-degenerative disorders, cardiovascular diseases, atherosclerosis, cataracts and inflammation (Halliwell, 1994; Aviram, 2000; Xiang, 2007). It has been proposed that decrease of lipid peroxidation and free radical formation would reduce diabetes disorders (Ali et al., 2007). Several study have shown that natural anti-oxidants, such as ginger, tea and chokeberry cultivar extracts, have scavenging activity and also decrease in lipid peroxidation activity (Otakar et al., 2010; Ali et al., 2007). Glycosidases are hydrolytic enzymes that play a vital role in digestion of carbohydrates and biosynthesis of glycoproteins (Chao et al., 2010). *a*-Glucosidase inhibitor can competitive inhibition a-glycosidase activity and delay or inhibit the absorption of glucose in the intestinal, effectively reduce the postprandial hyper-glycemia, the postprandial hyperglycemia can reduced effectively (Liu et al., 2010). So, *a*-glucosidase inhibition becomes one of selected and preferred targets for development of antitype II diabetes agents in the current (Ma et al., 2010).

Lysimachia paridiformis Franch. Var. *stenophylla*

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Franch. (LPF), belongs to Primulaceae family (State Administration of Traditional Chinese Medicine "Zhong Hua Ben Cao" Compilation Committee, 1997), is traditional Chinese medicinal herb. LPF is widely used by Miao nationality to cure hemiplegia, fractures and pediatric convulsion (Wen and Li, 2002; Wen and Wen, 2004). Phytochemical research showed that flavonoid glycosides were the main compounds in LPF (Li et al., 2008; Qi et al., 2010; Zhang et al., 2010). Pharmacological investigations showed that flavonoid extracts of LPF possessed analgesia and anti-inflammatory activities (Qi et al., 2010). However, there was no report concerning antioxidant, *a*-glucosidase inhibitory activities *in vitro* and protective effect for alloxan-induced diabetic rats *in vivo*.

In this work, antioxidant and *a*-glucosidase inhibitory activities of LPE extracts were assayed and their protective effect for alloxan-induced diabetic rats was studied.

MATERIALS AND METHODS

Plant material and fraction preparation

Air-dried plant of LPF were collected in Guizhou, China, in August 2007. Identified by Professor Fan Liu (Guiyang College of Traditional Chinese Medicine). A specimen was deposited in Institute of Chinese Materia Medica, Henan University. The air dried LPF powder (600 g) was extracted three times with methanol for 3 days at room temperature. After evaporation of solvent *in vacuo*, the concentrated extract was suspended in water and extracted successively with petroleum ether, ethyl acetate and *n*-Buthanol. The solution was concentrated under reduced pressure to yield petroleum ether extract (LPFPE, 2.8 g), EtOAC extract (LPFEA, 11 g) and *n*-BuOH extract (LPFBU, 40 g) respectively. The yields obtained for each fraction with respect to the initial dry material were LPFPE 0.46%, LPFEA 1.79%, and LPFBU 6.50%.

Materials and protocols *in vitro*

Materials in experiments in vitro

α-glucosidase (EC 3.2.1.20), 4-Nitrophenyl-*α*-D-glucopyranoside (PNPG, 026K1516). Acarbose (Lot 16869) from Sigma. dimethyl sulfoxide (DMSO), Gallic acid propyl (PG), butyl-*p*- hydroxyanisole (BHA) and butylated hydroxytoluene (BHT) from Sigma Chemical Co., 1,1- diphenyl-2-picrylhydrazyl (DPPH) from Tokyo Chemical Industry Co.. 2,4,6-tripyridyl- *S*-triazine (TPTZ) from Acros organics. 6-hydroloxy-2,5,7,8-tetramethyl-chroman-2-carboxylic acid (Trolox) from Aldrich. 2,2´-Azino-bis (3-ethylbenzo-thiazoline-6- sulfonicacid) (ABTS) from Fluka.

Antioxidant activity using DPPH assay

DPPH radical scavenging activity was assayed according to the method of Li et al. (2008). 0.1 ml different fractionation of LPF in methanol had been mixed with 3.5 ml DPPH methanol solution (0.06 mmol / L). The solution was measured at 515 nm after 30 min at room temperature with PG, BHA and BHT as positive control. The antioxidant activity was expressed as an IC_{50} value, *i.e.* the concentration in μg/ml that inhibits DPPH· absorption by 50%, and was calculated from the concentration-effect linear regression curve.

Antioxidant activity using ABTS assay

Scavenging activity on ABTS radical of LPF was evaluated in accordance with the literature (kang et al., 2011). The different fractionation of LPF extracts (0.15 ml) were mixed with ABTS radical stock solution (2.85 ml) and incubated at 37°C. The absorbance was observed at 734 nm after 10 min with PG, BHA and BHT as positive control. The percentage inhibition of $ABTS^{\cdot+}$ was calculated using the formula: %Inhibition = $[(A_0-A_1)/A_0]$ x 100, where A_0 was the absorbance of the control and A_1 was the absorbance of the sample and the standard compound.

FRAP reducing activity assay

According to the literature (Thaipong et al., 2006), the LPF (0.2 ml) and fresh prepared TPTZ stock solution (3.8 ml) were mixed and incubated at 37℃ for 30 min. The absorbance was measured at 593 nm. Trolox was used as a reference standard. The standard curve was linear between 25 and 400 μmol/L Trolox. Results were expressed in μmol Trolox equivalents (TE)/g sample.

α-glucosidase inhibition assay

The *α*-glucosidase-inhibitory activity was screened by the method of the microplate-based method based on PNPG as substrate according to the literature (Kang et al., 2009; Li et al., 2011). The assay mixture (160 μL) contained 8 μL of a sample in DMSO (or DMSO itself as control), 112 μL phosphate buffer (pH 6.8) and 20 μL enzyme solution (0.2 U/ml *α*-glucosidase in phosphate buffer), mixed and incubated at 37℃ for 15 min. Then, substrate solution (20 μL, 2.5 mM PNPG prepared in the same buffer) was added. The reaction was terminated by adding 80 μL of 0.2 M Na_2CO_3 solution after incubated at 37℃ for 15 min. Enzymatic activity was quantified by measuring the p-nitrophenol released from PNP-glycoside at 405 nm wavelength in a 96 microplate reader. Enzymatic inhibition data were expressed as IC_{50} values (concentration of inhibitor required for 50% inhibition against *α*-glucosidase). The inhibitory rates (%) were calculated according to the formula: $[1-(OD_{test}-OD_{blank})/(\text{control } OD_{test}-\text{ control } OD_{blank})] \times 100\%$. All reactions were carried out with three replications. Acarbose was used as positive control.

Materials and protocols *in vivo*

Materials and animals in experiments in vivo

Male KM normal rats weighted 20±2 g was obtained from the Experimental Animal Center of Henan Province. (Zhengzhou, Hennan, China). (12 h light/dark cycle, 25℃ and humidity 45–65%) and were fed with standard rodent diet and water ad libitum. All animal procedures were approved by the ethical committee in accordance with the 'Institute ethical committee guidelines' for Animal Experimentation and Care. Animals were housed in polycarbonate cages.

Alloxan (Alfa Aesar A Johnson Matthey, USA). Maleicdialdehyde (MDA), superoxide dismutase (SOD) and glycogen were purchased from the Nanjing Jianchen Bioengineering Institute (Jiangsu, China). Total cholesterol (TC) and triglyceride (TG) (Shanghai Beihai Biotechnology engineering Co., Ltd., Shanghai,China). Blood glucose test kit (Shanghai Rongsheng Biotech Co., Ltd., Shanghai, China).

Table 1. Antioxidant activity of the different extracts of *Lysimachia paridiformis* Franch. var. *stenophylla* Franch.

Sample	DPPH radical scavenging capacity IC_{50} (μg/ml)	ABTS radical-scavenging capacity IC_{50} (μg/ml)	ferric reducing antioxidant power $RACT_{50}$ (μmol/g)
LPFPE	NA	109.32±1.52	156.55±0.71
LPFEA	24.38±0.55	10.31±0.30	695.2±7.07
LPFBU	15.69±0.47	10.97±0.33	765.4±21.21
PG[a]	0.90±0.01	0.76±0.01	10675.79± 89.32
BHA[a]	3.20±0.04	1.88±0.01	6633.04 ±114.04
BHT[a]	18.72±0.50	7.77±0.01	1581.68 ±97.41

Note: NA indicate not available because of low activity, BHA, BHT and PG were used as positive control.

Experimental design and treatment schedule

Mice were made diabetic by a single tail vein injection of alloxan monohydrate (80 mg/kg b.w.) after overnight fasting for 12 h. Alloxan was first weighed individually in Eppendorf's tube for each animal according to the weight and then solubilized with 0.2 ml saline (154 mmol/l NaCl) just prior to injection. Fasting for 96 h after alloxan injection, mice with marked hyperglycemia (fasting blood glucose > 11.0 mmoL/L) were included in the study. Experimental mice were divided into twelve groups of ten animals each. Group 1 (normal mice treated with distilled water). Group 2 (diabetic mice treated with distilled water). Group 3-5 received various doses of LPFPE (125, 250 and 500 mg/kg). Group 6-8 received 250, 500 and 1000 mg/kg of LPFEA and Group 9-11 received 200, 400 and 800 mg/kg of LPFBU respectively by intragastric administration. Group 12 was vehicle control (diabetic mice treated with 75 mg/kg acarbose) and then treated with drugs at 24 h later. The duration of treatment was 7 d for diabetic by intragastric administration. Blood was collected from the eyes after fasting 12 h, then the animals were sacrificed by cervical dislocation, the liver, kidney and spleen were removed promptly, and weighed. Blood samples were centrifuged (3000 rpm for 15 min at 4℃) for separatin g the serum. After that, The serum was stored at −20℃ afte r separation for the following biochemic analysis. The tissues were also stored at−20℃ until required.

Biochemical analyses

Blood glucose was estimated by commercially available glucose kit based on glucose oxidase method (Jun et al., 2008). Glycogen content, TC, TG, superoxide dismutase (SOD) and malonaldehyde (MDA) levels of the liver were measured following the commercial kit's instructions.

Statistical analysis

All the grouped data were statistically evaluated with SPSS 17.0 software. Statistical comparisons were compared by one-way analysis of variance (ANOVA). The results were considered statistically significant if the *p* values were 0.05 or less. All results are expressed as mean ± standard deviation (SD) for ten mice in each group.

RESULTS

In vitro assay for free radical scavenging activity

The antioxidant activity of LPF with half inhibitory concentration (IC_{50}) and Trolox equivalent ($RACT_{50}$) see Table 1. In DPPH assay, the antioxidant activity of LPFBU (IC_{50}= 15.69±0.47 μg/ml) was higher than that of BHT (IC_{50}= 18.72±0.50 μg/ml) as positive control and higher than that of LPFEA (IC_{50}= 24.38±0.55 μg/ml) and LPFPE. In ABTS assay, the antioxidant activity of LPFEA (IC_{50}= 10.31±0.30 μg/ml) and LPFBU (IC_{50}= 10.97±0.33 μg/ml) were lower than that of three positive control, and far higher than that of LPFPE (IC_{50}= 109.32±1.52 μg/ml). In FARP assay, antioxidant activity of LPFBU ($RACT_{50}$= 765.4±21.21 μmol/g) and LPFEA ($RACT_{50}$= 695.2±7.07 μmol/g) were lower than that of three positive control, and far higher than that of LPFPE ($RACT_{50}$= 156.55±0.71). Results showed that the antioxidant activity of LPFEA and LPFBU were higher than that of LPFPE, and LPFBU had the highest antioxidant activity *in vitro*.

a-glucosidase inhibitory activity in *vitro*

In the Table 2, LPFPE, LPFEA and LPFBU showed stronger inhibitory activity against *a*-glucosidase (IC_{50}= 38.97±0.33, 42.62±0.21 and 20±0.13 μg/ml, respect-tively) than that of acarbose (IC_{50}= 1103.01±12.15 μg/ml) as positive control. Results showed that the *α*-glucosidase inhibitory activities of LPFPE, LPFEA and LPFBU exhibited strong activity in dose dependent manner (Figure 1). *a*-Glucosidase inhibitory activities of different extracts from LPF were reported for the first time. Results showed that LPF might be a good source of natural *α*-glucosidase inhibitor using for the therapy of hyperglycemic and its complication.

Antidiabetic effect of the different extracts of *Lysimachia paridiformis* Franch. var. *stenophylla* Franch. in alloxan-induced diabetic mice

Serum blood glucose

In Table 3, the level of fasting blood glucose of diabetic control rats (Group 2) was significantly increased (p<0.001) compare with normal untreated rats (Group 1) and it indicated the model of alloxan-induced diabetic

Table 2. *α*-glucosidase inhibitory activity of the different extracts of *Lysimachia paridiformis* Franch. var. *stenophylla* Franch.

Sample	Concentration	*a*-glucosidase inhibition	
	(μg/ml)	I%	IC_{50}(μg/ml)
LPFPE	1500	95.41	38.97±0.33
LPFEA	1500	95.42	42.62±0.21
LPFBU	1500	98.95	20±0.13
Acarbose*	1500	57.26	1103.01±12.15

Note: *Acarbose was used as positive control.

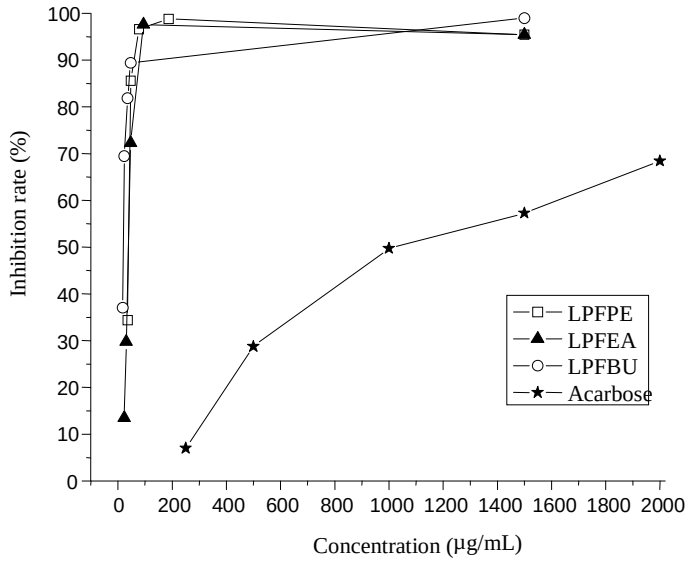

Figure 1. The mass concentration of the different extracts of *Lysimachia paridiformis* Franch. var. *stenophylla* Franch. effect on *α*-glucosidase inhibitory activity.

Table 3. Effect of LPFPE, LPFEA, and LPFBU treatment on fasting blood glucose.

Group	Dose	Before treatment	After treatment
	(mg/kg b.w.)	(mmol/L)	(mmol/L)
Normal control	—	3.33±0.30***	5.50±0.41***
Diabetic control	—	25.98±11.57△△△	30.22±1.48
LPFPE	500	25.08±11.92△△△	26.11±2.72
LPFPE	250	26.08±9.74△△△	25.45±8.67
LPFPE	125	22.73±9.73△△△	22.14±7.81
LPFEA	1000	23.02±7.84△△△	16.22±7.31***
LPFEA	500	22.70±6.45△△△	14.84±7.27***
LPFEA	250	22.10±7.73△△△	26.26±7.06
LPFBU	800	24.97±7.68△△△	25.02±7.53
LPFBU	400	24.88±3.59△△△	27.77±4.52
LPFBU	200	25.24±7.22△△△	29.38±11.17
Acarbose	75	21.88±4.90△△△	22.41±8.11*

Data expressed as means ± s.d (n=10). Acarbose was as the positive control drug. △p < 0.05, △△p<0.01, △△△p<0.001 alloxan-induced diabetic group compared with normal control group. *P＜0.05, **P＜0.01, ***P＜0.001 Treated group compared with alloxan-induced group.

Table 4. Effect of LPFPE, LPFEA, and LPFBU treatment on post-prandial blood glucose.

Group	Dose (mg/kg b.w.)	post-prandial blood glucose mmol/L
Normal control	—	3.85±0.68***
Diabetic control	—	38.43±5.66△△△
LPFPE	500	39.13±5.11△△△
LPFPE	250	41.00±3.73△△△
LPFPE	125	32.62±9.31△△△
LPFEA	1000	35.35±5.66△△△
LPFEA	500	35.39±11.96△△△
LPFEA	250	36.50±4.21△△△
LPFBU	800	30.95±4.63△△△
LPFBU	400	30.06±7.60△△△
LPFBU	200	32.66±9.18△△△
Acarbose	75	32.65±7.12△△△

Data expressed as means ± s.d (n=10). Acarbose was as the positive control drug. △p < 0.05, △△p<0.01, △△△p<0.001 alloxan-induced diabetic group compared with normal control group. *P＜0.05, **P＜0.01, ***P＜0.001 Treated group compared with alloxan-induced group.

Table 5. Effect of LPFPE, LPFEA, and LPFBU on liver glycogen level in alloxan-induced diabetic mice.

Group	Dose (mg/kg b.w.)	Liver glycogen (mg/g)
Normal control	—	30.21±1.44***
Diabeti ccontrol	—	4.03±0.71△△△
LPFPE	500	16.48±4.01***
LPFPE	250	25.37±6.05***
LPFPE	125	15.13±3.46**
LPFEA	1000	14.74±5.09**
LPFEA	500	20.13±5.05***
LPFEA	250	26.21±6.37***
LPFBU	800	16.01±3.00***
LPFBU	400	14.08±2.49*
LPFBU	200	10.98±3.50
Acarbose	75	24.35±6.67***

Data expressed as means ± s.d (n=10). Acarbose was as the positive control drug. △p < 0.05, △△p<0.01, △△△p<0.001 alloxan-induced diabetic group compared with normal control group. *P＜0.05, **P＜0.01, ***P＜0.001 Treated group compared with alloxan-induced group.

mice was established. The intragastric administration of LPFEA (1000 mg/kg and 500 mg/kg) to diabetic rats resulted in a significant decrease in level of fasting blood glucose (p<0.001). Daily treatment with LPFEA (1000 and 500 mg/kg) for 10 days led to fall in the level of fasting blood glucose by 70.46 and 65.37%, respectively. The level of fasting blood glucose of diabetic rats is higher than that of normal rats throughout the experimental period.

In Table 4, the level of post-prandial blood glucose of diabetic control was significant increase (p<0.001) compare with normal control. The administration of LPFPE, LPFEA and LPFBU to diabetic rats resulted in no significant decrease in level of post-prandial blood glucose (p>0.05). The treated groups have higher serum glucose level throughout the experimental period compared with normal rats.

Liver glycogen

In Table 5, the content of liver glycogen in serum of normal and diabetic animals was significantly decrease (p<0.001) in diabetes control compared with the normal

Table 6. Effect of LPFPE, LPFEA, and LPFBU treatment on MDA and SOD.

Group	Dose (mg/kg b.w.)	MDA (nmol/ml)	SOD (U/ml)
Normal control	—	4.40±0.44***	171.92±10.18***
Diabetic control	—	11.08±2.39△△△	107.66±16.80△△△
LPFPE	500	8.82±2.12	108.36±49.63
LPFPE	250	6.86±1.64***	72.62±34.15
LPFPE	125	8.00±2.65*	89.69±28.20
LPFEA	1000	7.36±1.75***	105.01±23.44
LPFEA	500	8.10±2.05**	103.17±11.94
LPFEA	250	8.77±1.93	110.48±12.51
LPFBU	800	9.84±1.20	95.32±19.21
LPFBU	400	10.09±0.72	119.33±23.35
LPFBU	200	8.38±1.77*	129.83±17.49
Acarbose	75	7.82±1.41**	123.76±13.78

Data expressed as means ± s.d (n=10). Acarbose was as the positive control drug. △p < 0.05, △△p<0.01, △△△p<0.001 Alloxan-induced diabetic group compared with normal control group. *P＜0.05, **P＜0.01, ***P＜0.001 Treated group compared with alloxan-induced group.

control group. Administration of LPFPE (500, 250, 125 mg/kg), LPFEA (1000, 500, 250 mg/kg), LPFBU (800, 400 mg/kg) and acarbose (75 mg/kg) resulted in a significant increase (p<0.001, p<0.01 and p<0.05, respectively) in the content of liver glycogen. The administration of LPFPE or LPFEA at the dose of 250 mg/kg b.w. and acarbose at the dose of 75 mg/kg b.w. showed a highly significant effect and no significant difference compared withthe normal control group (p>0.05).

MDA and SOD

In Table 6, the content of MDA was significantly increased (p<0.001) in diabetic mice whereas SOD was significantly decreased (p<0.001) in alloxan-induced diabetic mice compared with normal mice. The content of MDA in LPFPE (250 and 125 mg/kg b.w.), LPFEA (1000 and 500 mg/kg b.w.) and LPFBU (200 mg/kg b.w.) treatment group were significantly decreased (p<0.001, p<0.01and p<0.05, respectively) . The content of SOD in treatment group was no significant increase (p>0.05) compared with alloxan-induced diabetic mice.

TC and TG

In Table 7, the content of TG and TC in diabetic control was significantly increase (p<0.01 and p<0.05, respectively) compared with normal control groups. The content of TG in administration of LPFPE, LPFEA, LPFBU and acarbose was significantly decreased (p<0.001, p<0.01 and p<0.05, respectively). The content of TC in administration of LPFEA (1000, 500 and 250 mg/kg b.w.), LPFBU (800 and 400 mg/kg b.w.) and acarbose (75 mg/kg b.w.) was significantly decreased (p<0.001, p<0.01and p<0.05, respectively).

DISCUSSION

In recent years, studies have shown that there was exist phenomenon that free radical decompensate, the content of MDA was significantly increased and SOD was significantly decreased in animal models of diabetes mellitus and clinic diabetic sufferer. The level of free radicals and lipid peroxidation *in vivo* was associated with blood sugar elevated in patient, when the free radicals increased, because of its special role in cell toxicity, it may damage the body to produce a series of roles, in which the pancreatic β cell damage is one of the important factors that trigger diabetes and directly affects the further development of disease (Candlish and Das, 1996; Ye et al., 2000). *a*-Glycosidase enzymes is located in the small intestinal villi, it is a key enzymes that carbohydrate is digested to the last step (Gao et al., 2008). Varieties of plants extract are known to have *α*-glucosidase inhibitory activity and can effectively regulate postprandial glucose level of diabetes patient (Kang et al., 2010; Ma and Lue, 2010). The experimental results show that LPFPE, LPFEA and LPFBU have good *α*-glucosidase inhibitory activity and definite antioxidant activity *in vitro*, LPFBU has the strongest *α*-glucosidase inhibitory and antioxidant activity, and then LPFEA. Experiments in diabetes mice show that, LPFEA (1000, 500 mg/kg b.w.) significantly decreased fasting blood glucose, LPFEA (500, 250 mg/kg b.w.) and LPFBU (800,

Table 7. Effect of LPFPE, LPFEA, and LPFBU treatment on TG and TC.

Group	Dose (mg/kg b.w.)	TG (mmol/L)	TC (mmol/L)
Normal control	—	0.94±0.18**	2.67±0.38*
Diabetic control	—	1.40±0.24△△	3.87±0.53△
LPFPE	500	0.73±0.18***	2.98±0.53
LPFPE	250	0.64 ±1.19***	3.48±0.53
LPFPE	125	0.73±0.28***	3.68+0.80
LPFEA	1000	0.77±0.37***	2.84±0.60*
LPFEA	500	0.79±0.23***	1.65±0.46***
LPFEA	250	0.98±0.26**	1.83±0.30***
LPFBU	800	1.04±0.22*	2.19±0.49***
LPFBU	400	0.96±0.25**	1.76±0.51***
LPFBU	200	1.09±0.35*	3.38±0.61
Acarbose	75	0.53±0.10△***	3.07±0.69

Data expressed as means ± S.D (n=10). Acarbose was as the positive control drug. △p < 0.05, △△p<0.01, △△△p<0.001 alloxan-induced diabetic group compared with normal control group. *P＜0.05, **P＜0.01, ***P＜0.001 Treated group compared with alloxan-induced group.

400 mg/kg b.w.) significantly increased liver glycogen and significantly decreased in serum TG, TC content, LPFEA (1000, 500 mg/kg b.w.) and LPFBU (200 mg/kg b.w.) significantly decreased the MDA content, increased serum levels of SOD, but no significant difference.

In summary, LPFPE, LPFEA and LPFBU have strong α-glucosidase inhibitory activity and certain antioxidant activity. IPFEA (500 mg/kg b.w.) can decrease fasting blood glucose, serum TG and TC content to improve concurrent hyperlipidemia of ALX-induced diabetic mice, and correct the abnormal lipid metabolism. Through improved liver glycogen synthesis, decreased glycogen break down and lower blood sugar. By reducing the content of MDA and increasing activity of SOD to enhance antioxidant capability and protect the body to further oxidative damage from free radicals in diabetic mice, which play the role of hypoglycemic effect. Therefore, LPF has effective prevention and treatment on the development for diabetes and its complications.

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