Hindawi
Journal of Chemistry
Volume 2019, Article ID 6821438, 9 pages
https://doi.org/10.1155/2019/6821438

Research Article

Antidiabetic Effects of Soluble Dietary Fiber from Steam Explosion-Modified Black Soybean Hull in Low-Dose Streptozotocin-Induced Type 2 Diabetic Mouses

Meng Shen,[1] **Weihao Wang**,[1,2] **Yunfei Ge**,[1] **Ziyue Kang**,[1] **Juan Wang**,[1] **Zhigang Quan**,[1] **Jinling Xiao**,[1] **and Longkui Cao**[1,2]

[1]*College of Food Science, Heilongjiang Bayi Agricultural University, Xinfeng Lu 5, Daqing 163319, China*
[2]*National Coarse Cereals Engineering Research Center of Heilongjiang Province, Daqing 163319, China*

Correspondence should be addressed to Longkui Cao; caolongkui20190606@163.com

Received 16 May 2019; Revised 9 November 2019; Accepted 28 November 2019; Published 24 December 2019

Academic Editor: Marcone A. L. de Oliveira

Copyright © 2019 Meng Shen et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

This paper studies the antidiabetic effects of soluble dietary fiber (SDF) from steam explosion-modified black soybean hull in low-dose streptozotocin- (STZ-) induced type 2 diabetic mouses. Male C57/BL6 mouses were divided into 4 groups: control (nondiabetic, no SDF intake), model (diabetes only), metformin (metformin: 100 mg/kg body weight), and SDF (SDF: 600 mg/kg body weight). Four weeks post-SDF treatment, treatment of SDF decreased the weight gain of diabetic mouses, normalised the blood glucose level, and reduced the serum cholesterol, serum insulin, leptin, glucagon-like peptide, total cholesterol, triglyceride, low-density lipoprotein cholesterol, arteriosclerosis index, aspartate aminotransferase activity, and malondialdehyde. It also increased high-density lipoprotein cholesterol, adiponectin, glycopeptide peroxidase, superoxide dismutase activity and repaired the pancreatic injury of the diabetic mouses. Our research results show that SDF has the potential for use in type 2 diabetes treatment.

1. Introduction

Diabetes is one of the most common endocrine diseases triggered by insulin secretion deficiency or insulin disorders. Research suggests that by 2030, 552 million people will suffer from diabetes. Each year, diabetes causes more than 2.9 million deaths; this will continue to rise over the next 30 years [1, 2]. Diabetes, especially type 2 diabetes, combined with its complications, such as hyperlipidemia, hypertension, atherosclerosis, and obesity, is a metabolic syndrome and is closely related to human health status [3]. According to surveys, sulphonylurea and biguanide medicines are currently commonly used to regulate blood glucose level to alleviate the symptoms of type 2 diabetes; however, their use is associated with severe side effects, including nausea, vomiting, loss of appetite, abdominal pain, diarrhea, and allergy [4]. Therefore, the identification of natural hypoglycemic components that not only delay the rapid rise of postprandial blood glucose but also reduce the intestinal absorption of sugar from food is required [5].

SDF can reduce blood glucose, blood lipid, blood pressure, and atherosclerosis. Studies have shown that SDF can reduce the postprandial blood glucose, increase insulin (INS) secretion, improve food intake and body weight, and reduce plasma glucose concentration in diabetic rats; it can also reduce the concentration of cholesterol (TC), triglyceride (TG), and low-density lipoprotein cholesterol (LDL-C) in the plasma [6–9]. Previous studies have suggested that dietary fiber obtained from the consumption of fruits, grains, and beans can increase the content of liver glycogen and inhibit the rise of postprandial blood glucose, as well as increase the activity of superoxide dismutase (SOD) in the serum and liver, reduce the content of malondialdehyde (MDA), and reduce the activity of lactic dehydrogenase (LDH) in the serum and liver so as to effectively protect the heart and liver. It has an excellent blood glucose reduction effect [6, 9].

Due to variations in the components and solubility, dietary fiber from different sources has different fermentation degrees in the large intestine of diabetic rats, resulting in different metabolic effects and thus varying degrees of alleviation of the hyperglycemia symptoms in diabetic rats [7, 9]. Studies have found that different types of dietary fiber have different effects on this metabolic syndrome. Soluble guar gum fiber has been found to normalise body weight, blood glucose levels, INS secretion, and TC levels in obese C57/BL6 mouses [8, 10]. Black soybean hull is a by-product of black soybean processing that contains abundant SDF. According to the preliminary studies of this experiment, steam explosion- (SE; moisture content of material: 15%, 1.0 MPa, 80 s) modified black soybean hull showed an increased concentration of SDF (increased from 10.02% (unmodified) to 17.49%) and enhanced physical and chemical properties, such as water retention capacity, expansive force, and oil retention capacity. Studies using *in vitro* hyperglycemia models showed that SDF in SE-modified black soybean hull could effectively improve the inhibitory effect on α-glucosidase and α-amylase activities. Studies recently reported that pectin and xylan polysaccharides reduce glucose intake, fasting blood glucose, and postprandial glucose after a carbohydrate-rich meal through various mechanisms, such as β-cell protection, inhibition of activities of glucose metabolism-related enzymes, or regulation of insulin-related pathways. Guar gum has been reported to lower body weight and TC in obese mice, compared to oat insoluble DF (IDF) [10, 11]. However, there are currently no reports on the role of SDF in the SE-modified black soybean hull alleviation of hyperglycemia and other symptoms in type 2 diabetic mouses. In the present study, we investigated the effects of SDF from steam explosion-modified black soybean hull on body weight, food intake, serum biochemical indices, serum lipids, and histopathological analysis in type 2 diabetic mouses using an experimental mouse model of low-dose streptozotocin- (STZ-) induced diabetes. Our collective findings clearly support the therapeutic potential of SDF from black soybean hull on type 2 diabetes.

2. Materials and Methods

2.1. Materials. Black soybean hulls (Anxuan no. 1) were purchased from Bozhou Runbang Food Sales Co., Ltd. (Anhui, China). Neutral protease, high temperature-resistant α-amylase, glucosidase, and metformin were purchased from Sigma (St. Louis, MO, USA). All other chemical reagents used in this study were of analytical grade.

2.2. Preparation of SDF. Black soybean hulls with a moisture content of 15% were added to a SE cylinder, with a pressure of 1.0 MPa and a time of 80 s to obtain the SE samples. Next, 1.0 g of degreased and SE-modified black soybean hulls was weighed, and distilled water was added at a solid-liquid ratio of 1 : 30 (g/mL) to regulate the pH to 6.0. Subsequently, 100 μL of high temperate-resistant α-amylase was added to the solution. The resulting mixture was maintained at a temperature of 95–100°C and allowed to react for 20 min. The pH was then increased to 7.0 by adding 100 μL of neutral protease solution, and the mixture was allowed to react at 60°C for 30 min. Finally, the pH was decreased to 4.5 by adding 100 μL of amyloglucosidase, and the mixture was allowed to react at 60°C for 30 min. The solution was filtered and concentrated, and then, 95% ethyl alcohol was added to increase the volume by fourfold and allowed to settle for 10 h. The solution was centrifuged at 3200 r/min for 30 min and freeze-dried at −108°C for 8 h to obtain SDF.

2.3. Animal Experiments. Seven-week-old male C57/BL6 mouses were purchased from Liaoning Changsheng Biotechnology Co., Ltd. (Shenyang, China). The mouses were adaptively bred at a constant temperature (22 ± 2°C) and a relative humidity of (55 ± 10%), with day and night alternating every 12 hours for two weeks, during which the mouses were provided with access to food and water. The breeding and handling of the animals were carried out in accordance with the regulations of the Chinese society of experimental animals (CALAS).

2.4. Experimental Design. The mouses were randomly divided into nondiabetic (Group 1) and induced diabetic groups. The nondiabetic group ($n = 7$) was intraperitoneally injected with 3 mL/kg of citric acid buffer solution (0.1 mol/L, pH 4.4). The diabetic group was intraperitoneally injected with low-dose 0.1 mol/L of STZ (40 mg/kg, dissolved in 0.1 M, pH 4.4 phosphate buffer solution) [6, 11]. After one week of continuous injections, blood samples were obtained via the tail veins. The fasting blood glucose (FBG) of the mouses was measured using a Roche glucometer (active blood glucose test paper; Roche Diagnostics, Shanghai, China). An FBG concentration >11.11 mmol/L indicated the successful establishment of the type 2 diabetes model. Next, the mouses were randomly divided into 3 groups ($n = 7$): diabetes model group (Group 2, normal saline: 0.2 mL), metformin group (Group 3, metformin: 100 mg/kg 0.2 mL), and SDF group (Group 4, SDF: 600 mg/kg 0.2 mL). According to the pharmacology experimental method, the conversion formula of dose for mouse was based on body weight dose concentration: mouse dose (mg/kg) = human dose (mg/kg) × conversion constant. An adult male weighs 70 kg and consumes 10 g of SDF daily. The daily intake of SDF converted to 1 kg of adult body weight is about 0.14 g. Since the conversion constant of the mouse is 9.1, the daily intake of SDF of 1 kg mouse is about 1.27 g. The preliminary experiment divided the SDF gavage dose into high dose (1200 mg/kg), medium dose (900 mg/kg), and low dose (600 mg/kg). The results showed that when the SDF dose was 600 mg/kg, the physiological status and blood glucose of the mice were the best. Therefore, the dose was chosen to be 600 mg/kg [6, 11].

The experiments were conducted over 4 weeks, during which the mouses had access to food and water. All the gavage reagents were dissolved in normal saline. The feed was obtained from Liaoning Changsheng Biotechnology Co., Ltd. At the end of the 4-week experimental period,

blood samples were obtained from the eyes. The blood samples were collected in 1.5 mL centrifuge tubes and centrifuged at 12000 r/min for 10 min. The resulting serum was stored at −20°C. The pancreas was sectioned into slices and placed in formalin buffer solution for histological observation.

2.5. Analysis of Serum Indices. Commercial kits were used to measure the concentration of insulin (INS), leptin (LEP), adiponectin (ADPN), glucagon-like peptide-1 (GLP-1), malondialdehyde (MDA), total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C), as well as the activity of glutamic oxalacetic transaminase (AST), glutathione peroxidase (GSH-PX), and superoxide dismutase (SOD) (Nanjing Jiancheng Bioengineering Institute, Nanjing, China).

2.6. Calculation of Ambulatory Arterial Stiffness Index (AI). The arterial stiffness index (AI) is calculated as follows:

$$\mathrm{AI} = \frac{\mathrm{LDL} - \mathrm{C\,(content)}}{\mathrm{HDL} - \mathrm{C\,(content)}}. \quad (1)$$

2.7. Histopathological Observations. The pancreases of the mouses were collected, immersed in 10% formalin solution, dehydrated via alcohol gradient, wrapped with paraffin, and cut into 4 μm sections. The samples were then stained with haematoxylin and eosin (HE) to obtain HE-stained samples. The morphological structure of the pancreases was then observed under a microscope.

2.8. Data Analysis. Each experiment was repeated in triplicate, and the experimental data obtained were expressed as the mean ± standard deviation (SD). Statistical analysis was conducted using Microsoft Excel (2003), where $p < 0.05$ was used as the standard for significance.

3. Results and Discussion

3.1. Effect of SDF on Food and Water Intake and Body Weight in Nondiabetic and Diabetic Mouses. Figure 1 shows the effects of SDF on food intake, water intake, and body weight of nondiabetic and diabetic mouses. Figure 1(a) shows that 4 weeks after drug administration, the food intake in the diabetic model group increased by 29.38% ($p \leq 0.05$) compared to the nondiabetic group. Compared to the diabetic model group, the food intake of the metformin and SDF groups decreased by 26.58% and 25.29% ($p \leq 0.05$), respectively, and there was no significant difference between the food intake of the SDF and metformin groups. Figure 1(b) shows that 4 weeks after drug administration, the water intake in the diabetic model group increased by 82.85% ($p \leq 0.05$) compared to the nondiabetic group. Compared to the diabetic model group, the water intake of the metformin and SDF groups decreased by 19.31% and 17.02% ($p \leq 0.05$), respectively, and there was no significant difference between the water intake of the SDF and metformin groups. According to Figure 1(c), 4 weeks after drug administration, the body weight decreased by 36.73% ($p \leq 0.05$) in the diabetic model group compared to the nondiabetic group. Compared to the diabetic model group, the body weight of the metformin and SDF groups increased by 41.81% and 30.93% ($p \leq 0.05$), respectively.

Throughout the experiment, the mouses in the nondiabetic group showed normal behavior including dieting, drinking, and physiological activity. After repeated intraperitoneal injections of low-dose STZ, the diabetic mouses gradually developed polydipsia, polyurination, polyphagia, and emaciation symptoms, with dull hair color, listlessness, slow reactions, and slow movements [12]. These symptoms did not improve in the diabetic model group over the course of the experiment but were significantly alleviated in the drug-induced diabetic group, where the mouses showed improved physiological activity. The diabetic mouses showed INS resistance and decreased blood glucose control ability, possibly due to the low efficiency of food conversion, which resulted in an increased food intake but a decreased body weight [11, 13]. Previous studies have suggested that metformin drugs and SDF can effectively improve food intake, water intake, and body weight of diabetic mouses, which is consistent with the results of this experiment [11, 14]. These findings indicate that SDF can effectively alleviate the symptoms of polydipsia, polyurination, polyphagia, and weight loss caused by glucose metabolism disorders in the diabetic mouse model.

3.2. Effects of SDF on Blood Glucose Level in Nondiabetic and Diabetic Mouses. As shown in Figure 2, at week 0, there was a significant difference between the FBG values of the nondiabetic group and the diabetic group induced by low-dose STZ, However, there was no significant difference between the FBG values of the diabetic groups. During weeks 1–4, compared to the diabetic model group, the FBG levels of the diabetic groups treated with metformin and SDF decreased significantly ($p \leq 0.05$), with no significant difference between the metformin and SDF groups.

Long-term hyperglycemia causes severe damage to the structure and function of pancreatic β cells, reduces the number of glucose transporter 2 (GLUT-2) on the cell membrane, and reduces the synthesis and release of INS; it also hinders the glucose metabolism in cells, such that the number of abnormally apoptotic pancreatic β cells increases significantly. Cereal extracts rich in β-glucan may reduce the FBG in STZ-induced diabetic mouses [15]. Low-dose STZ-induced diabetes damages pancreatic β cells to a certain degree. During weeks 1–4, the FBG levels of the metformin and SDF diabetic mouses decreased gradually. The secretion of INS by residual β cells was stimulated [16], which improved the sensitivity of peripheral tissues to INS and considerably enhanced the glucose tolerance of diabetic mouses, as well as reduced the demand for INS, contributing to the synthesis of glycogen, thus lowering the blood glucose levels [16–18].

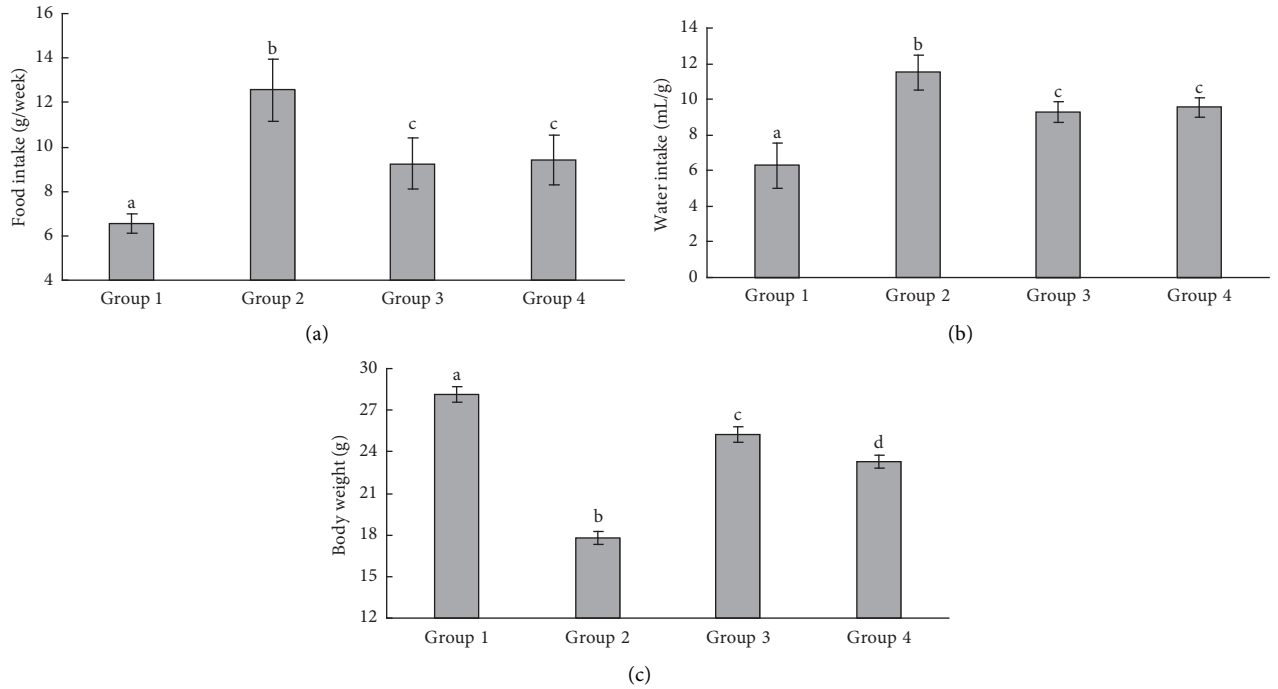


Figure 1: Effect of SDF on food intake, water intake, and body weight in nondiabetic and diabetic mouses. Data are expressed as mean ± standard deviation (SD). Values with different letters are significantly different, $p \leq 0.05$. Group 1: nondiabetic group; Group 2: diabetes model group; Group 3: metformin group; Group 4: SDF group.

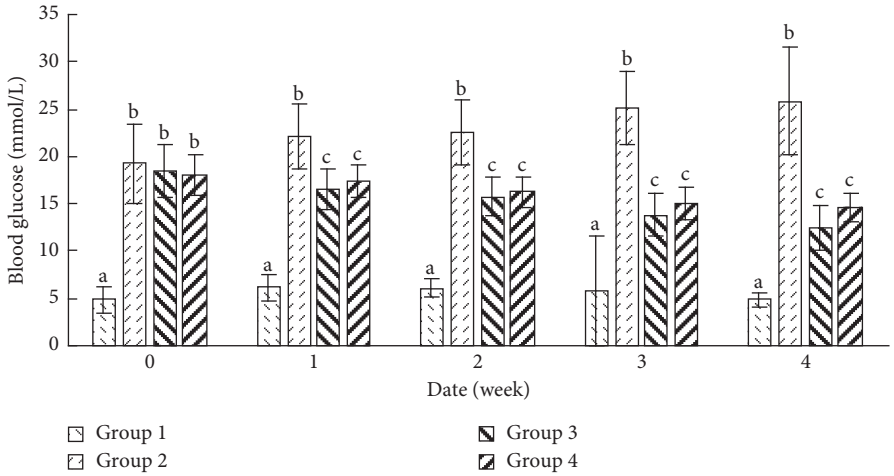


Figure 2: Effects of SDF on blood glucose level in nondiabetic and diabetic mouses. Data are expressed as mean ± standard deviation (SD). Values with different letters on the same histogram are significantly different, $p \leq 0.05$. Group 1: nondiabetic group; Group 2: diabetes model group; Group 3: metformin group; Group 4: SDF group.

3.3. Effects of SDF on INS, LEP, ADPN, and GLP-1 Concentration in Nondiabetic and Diabetic Mouses. INS is secreted by pancreatic β cells via the hydrolysis of several enzymes. The determination of INS concentration is important in the study of type 2 diabetes [19]. Figure 3(a) shows that 4 weeks after drug administration, the serum INS concentration in the diabetic model group increased by 71.28% ($p \leq 0.05$) compared to the nondiabetic group. Compared to the diabetic model group, the serum INS concentration of the metformin and SDF groups decreased by 29.52% ($p \leq 0.05$) and 11.10%, respectively, and there was no significant difference between the serum INS concentration of the SDF and metformin groups. According to the results, diabetic mouses with low serum INS levels were more sensitive to INS, while those with high serum INS levels were less sensitive to INS [20]. Cereals rich in dietary fiber and pericarps can effectively improve the INS levels of diabetic rats to regulate the INS sensitivity [21]. This indicates that SDF can effectively improve the sensitivity of diabetic mouses to INS and alleviate hyperglycemia in type 2

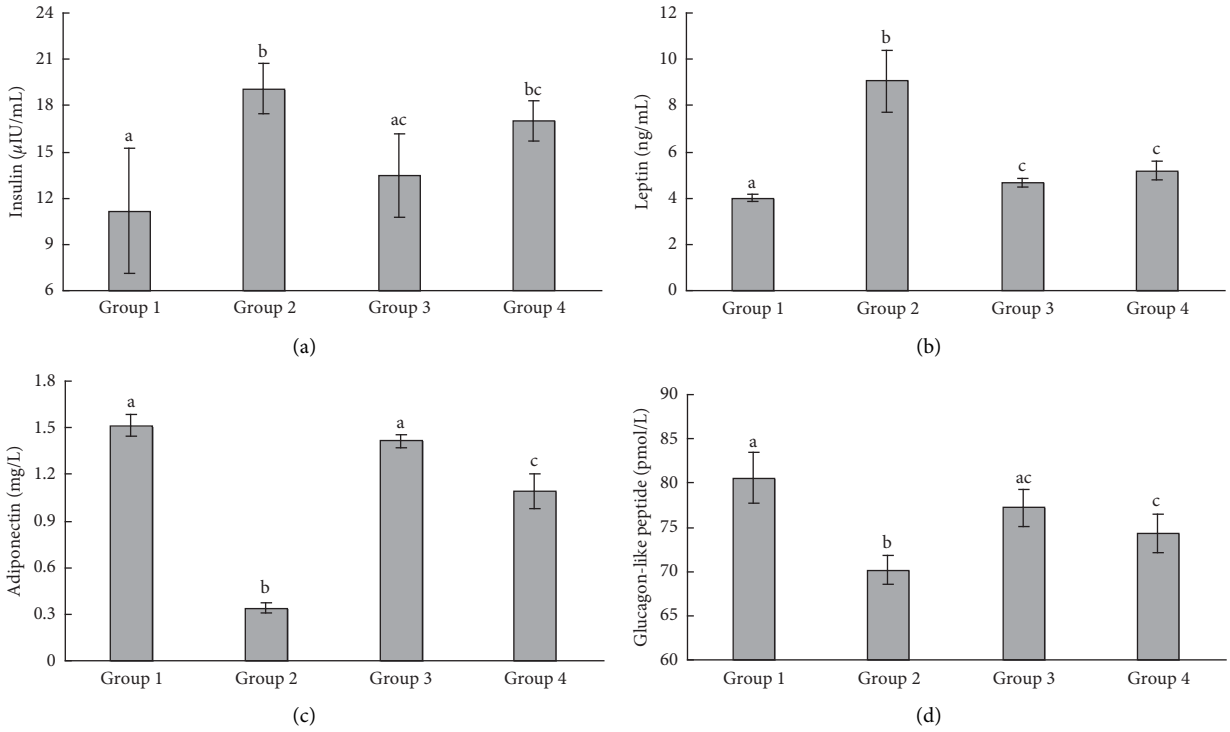


Figure 3: Effects of SDF on the concentration of hormones in nondiabetic and diabetic mouses. Data are expressed as mean ± standard deviation (SD). Values with different letters are significantly different, $p \leq 0.05$. Group 1: nondiabetic group; Group 2: diabetes model group; Group 3: metformin group; Group 4: SDF group. (a) Insulin. (b) Leptin. (c) Adiponectin. (d) Glucagon-like peptide-1.

diabetes, which is consistent with the results of the above-mentioned studies.

LEP is an intracellular protein hormone secreted by adipose tissues. INS can promote the secretion of LEP, which controls the synthesis and secretion of INS via negative feedback [22]. Increased LEP concentration leads to LEP resistance, and the LEP levels in the plasma are usually positively correlated with body weight, especially with regard to changes in adipose tissue. LEP resistance directly results from increased LEP levels in the cycle [22]. As seen in Figure 3(b), 4 weeks after drug administration, the serum LEP concentration in the diabetic model group increased by 5.05 ng/mL ($p \leq 0.05$) compared to the nondiabetic group. Compared to the diabetic model group, the serum LEP concentration in the metformin and SDF groups decreased by 4.36 ng/mL ($p \leq 0.05$) and 3.87 ng/mL ($p \leq 0.05$), respectively; however, there was no significant difference in serum LEP concentration between the SDF and metformin groups. LEP resistance occurs when LEP is either oversecreted or undersecreted, leading to a common metabolic syndrome in mouses [23].

ADPN is an endogenous bioactive polypeptide or protein secreted by adipose cells and an INS-sensitizing hormone that can alleviate INS resistance and atherosclerosis in rats. ADPN level can be used to predict the development of type 2 diabetes and coronary heart disease, which has the potential to alleviate the symptoms of diabetes, atherosclerosis, and inflammation in clinical trials [24]. As seen in Figure 3(c), 4 weeks after drug administration, the serum ADPN concentration of the diabetic model group decreased by 1.18 mg/L ($p \leq 0.05$) compared to the nondiabetic group; the serum ADPN concentrations of the metformin and SDF groups increased by 1.08 mg/L ($p \leq 0.05$) and 0.75 mg/L ($p \leq 0.05$), respectively, compared to the diabetic model group; however, significant difference in serum ADPN concentrations between SDF and metformin groups was observed. Additionally, there was a significant difference in the serum ADPN concentrations between the SDF, nondiabetic, and diabetic groups ($p \leq 0.05$). Clinical studies have shown that patients with diabetes who consume cereal fiber have decreased blood glucose levels and increased ADPN levels, which correlated with the findings presented here [25, 26]. These results suggest that SDF can effectively improve the ADPN secretion levels and promote the secretion of INS in diabetic mouses, thereby improving the symptoms of diabetes.

GLP-1 is a brain-gut peptide secreted by endocrine cells in the ileum that is currently used as a therapeutic target for the treatment of type 2 diabetes. GLP-1 can act on pancreas islet β cells, stimulating the proliferation and differentiation of β cells, inhibiting the apoptosis of β cells, increasing the number of β cells, and promoting the transcription of INS genes, the synthesis, and secretion of INS [27]. According to Figure 3(d), 4 weeks after drug administration, the serum GLP-1 concentration decreased by 12.91% ($p \leq 0.05$) in the diabetic model group and increased by 2.13% ($p \leq 0.05$) and 2.19% ($p \leq 0.05$) in the metformin and SDF groups, respectively, compared to the diabetic model group. There was no significant difference between the serum GLP-1 peptide concentration of the SDF and metformin groups. These

results indicate that SDF significantly increases the plasma GLP-1 concentration in mouses, thus promoting the secretion of INS and alleviating hyperglycemia in diabetic mouses [28–30].

3.4. Effects of SDF on AST, GSH-PX, and SOD Activities and MDA Content in Nondiabetic and Diabetic Mouses. Table 1 shows the effects of SDF on the AST, GSH-PX, SOD, and MDA levels in the serum of type 2 diabetic mouses. Compared to the nondiabetic group, the serum AST activity and MDA content in the diabetic model group increased by 273.22 U/L ($p \leq 0.05$) and 4.85 nmml/mL ($p \leq 0.05$), respectively, while the GSH-PX and SOD activities in the serum decreased by 4.94 U/L ($p \leq 0.05$) and 51.10 U/L ($p \leq 0.05$), respectively. The statistical significance indicates that the liver cells of the mouses in the diabetic model group underwent considerable damage. Compared to the diabetic model group, the GSH-PX and SOD levels in the serum of the metformin and SDF groups increased ($p \leq 0.05$), while the AST and MDA levels decreased ($p \leq 0.05$). The statistical significance indicates that SDF reduces the degree of liver injury in type 2 diabetic mouses.

The liver is an important metabolic organ, where oxidative stress results in several chronic diseases [31]. Oxidative stress is caused by an imbalance between the oxidation and antioxidant levels in the body, triggered by free radicals related to the occurrence and development of diabetes in the body [31, 32]. In the scavenging process of reactive oxide species, SOD enzymes restore superoxide anions into hydrogen peroxide (H_2O_2), and GSH-PX restores H_2O_2 into H_2O. MDA is produced in the process of lipid peroxidation, mainly from the oxidative decomposition of unsaturated fatty acids [32]. Therefore, changes in SOD, GSH-PX, and MDA levels are important indicators for the measurement of the antioxidant system. AST is an indicator of clinical liver function, used to determine whether the liver is damaged. Studies have shown that the intake of foods that ease oxidative stress or improve antioxidation function is an effective way to alleviate type 2 diabetes [33]. The pectin polysaccharide component of SDF has been found to scavenge oxidative free radicals in vitro [34]. Dietary fiber plays an important role in alleviating type 2 diabetes by improving the antioxidant defense system of the liver and reducing lipid peroxidation [35]. After the administration of metformin and SDF in diabetic mouses, the AST activity and MDA content decreased, while GSH-PX and SOD activities increased, suggesting that metformin and SDF reduce the degree of liver injury in type 2 diabetic mouses. These results are consistent with the aforementioned studies.

3.5. Effects of SDF on Serum Lipid Concentration and AI in Nondiabetic and Diabetic Mouses. Table 2 shows the effects of SDF on the TC, TG, LDL-C, HDL-C, and AI levels in the serum of nondiabetic and diabetic mouses. Compared to the nondiabetic group, the concentration of TC, TG, and LDL-C in the serum of the diabetic model group increased by 13.48 mg/mL ($p \leq 0.05$), 0.83 mmol/L ($p \leq 0.05$), and 2.63 mg/mL ($p \leq 0.05$), respectively. The concentration of HDL-C decreased by 22.96 mg/mL ($p \leq 0.05$) and AI increased by 2.06 ($p \leq 0.05$). Compared to the diabetic model group, after the administration of metformin and SDF in nondiabetic mouses, the concentration of TC in the serum decreased by 32.47% ($p \leq 0.05$) and 23.23% ($p \leq 0.05$), respectively; TG decreased by 0.89% ($p \leq 0.05$) and 1.15%, respectively; LDL-C decreased by 28.00% ($p \leq 0.05$) and 16.00% ($p \leq 0.05$), respectively; HDL-C increased by 3.48-fold ($p \leq 0.05$) and 2.04-fold ($p \leq 0.05$), respectively; and AI decreased by 0.87-fold ($p \leq 0.05$) and 0.71-fold ($p \leq 0.05$), respectively.

Type 2 diabetes induced by high-sugar and high-fat diets and low-dose STZ is generally accompanied by dyslipidemia [11]. HDL-C is known as "good" cholesterol. It is an endogenous cholesterol ester involved in reversal transport, which transports cholesterol from the blood to the liver, thereby reducing the risk of coronary heart disease [36]. AI is closely related to atherosclerosis, a warning sign of cardiovascular disease. Normally, an AI level <4 indicates that the degree of atherosclerosis is not serious or is decreasing. The smaller the AI value, the less severe the degree of atherosclerosis and the lower the risk of atherosclerosis [37]. As seen in Table 2, metformin and SDF resulted in reduced TC, TG, LDL-C, and AI levels in the serum of diabetic mouses. SDF effectively reduces the concentration of TC, TG, LDL-C, and AI in the plasma of type 2 diabetic mouses, thus reducing the accumulation of fat in the liver [11].

Obesity was induced in Wistar mouses by high-sugar and high-fat diet. After the intake of dietary fiber from sweet potatoes, the concentration of TC, TG, and LDL-C in the plasma was found to decrease; however, there was no significant difference in the concentration of HDL-C [38]. These results are consistent with those of the above-mentioned studies.

3.6. Pathological Observation and Analysis of Pancreatic Tissues. The pancreatic pathological sections in Figure 4 show the effect of SDF on the pancreases of the nondiabetic and diabetic mouses. The pancreas islets of the nondiabetic mouses were oval or nearly round, with a large area, a complete structure, a regular distribution of β cells with a uniform size and complete arrangement, abundant secretory particles of INS in the cytoplasm, deep staining, no degeneration, and clear islet and β cell boundaries. On the other hand, in the diabetic model group, the pancreas was severely atrophic, the islet area was severely shrunk, and the β cells were severely degenerated, with an uneven distribution, loose arrangement, and irregular and fuzzy boundaries. In both the metformin and SDF groups, the pancreases of the diabetic mouses were significantly improved; the islet area was significantly increased, the number of β cells increased significantly, the secretory granules of the INS in the cytoplasm increased, and the cell boundaries were clear.

During the induction of the type 2 diabetic model, multiple intraperitoneal injections of low-dose STZ selectively degrade islet β cells, causing severe atrophy, reducing the secretion of INS, and eventually increasing the blood

Table 1: Effects of SDF on AST, GSH-PX, and SOD activities and MDA content in nondiabetic and diabetic mouses.

	AST (U/L)	GSH-PX (U/L)	SOD (U/mL)	MDA (nmml/mL)
Group 1	92.62 ± 2.41^{a}	8.28 ± 0.35^{a}	123.01 ± 2.92^{a}	5.35 ± 0.37^{a}
Group 2	365.84 ± 32.62^{b}	3.34 ± 0.17^{b}	71.91 ± 4.42^{b}	10.20 ± 0.26^{b}
Group 3	145.07 ± 16.86^{c}	6.36 ± 0.34^{c}	102.05 ± 2.25^{c}	8.02 ± 0.20^{c}
Group 4	263.59 ± 13.21^{d}	5.13 ± 0.42^{d}	87.92 ± 2.77^{d}	9.13 ± 0.52^{d}

Data are expressed as mean ± standard deviation (SD). Values in the same column with different letters are significantly different, $p \leq 0.05$. Group 1: nondiabetic group; Group 2: diabetes model group; Group 3: metformin group; Group 4: SDF group. AST: aspartate aminotransferase; GSH-PX: glutathione peroxidase; SOD: superoxide dismutase; MDA: malondialdehyde.

Table 2: Effects of SDF on serum lipid concentration and AI in nondiabetic and diabetic mouses.

	TC (mmol/L)	TG (mmol/L)	HDL-C (mmol/L)	LDL-C (mmol/L)	AI
Group 1	2.75 ± 0.25^{a}	0.57 ± 0.04^{a}	25.95 ± 7.08^{a}	1.87 ± 0.59^{a}	0.07 ± 0.01^{a}
Group 2	16.23 ± 2.73^{b}	1.40 ± 0.25^{b}	2.99 ± 2.26^{b}	4.50 ± 0.65^{b}	2.13 ± 1.46^{b}
Group 3	10.96 ± 1.80^{c}	0.89 ± 0.54^{a}	13.41 ± 3.78^{c}	3.24 ± 0.60^{c}	0.27 ± 0.12^{c}
Group 4	12.46 ± 3.59^{c}	1.15 ± 0.43^{b}	9.10 ± 5.63^{c}	3.78 ± 0.11^{d}	0.61 ± 0.44^{c}

Data are expressed as mean ± standard deviation (SD). Values in the same column with different letters are significantly different, $p \leq 0.05$. Group 1: nondiabetic group; Group 2: diabetes model group; Group 3: metformin group; Group 4: SDF group. TC: total cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; AI: arteriosclerosis index.

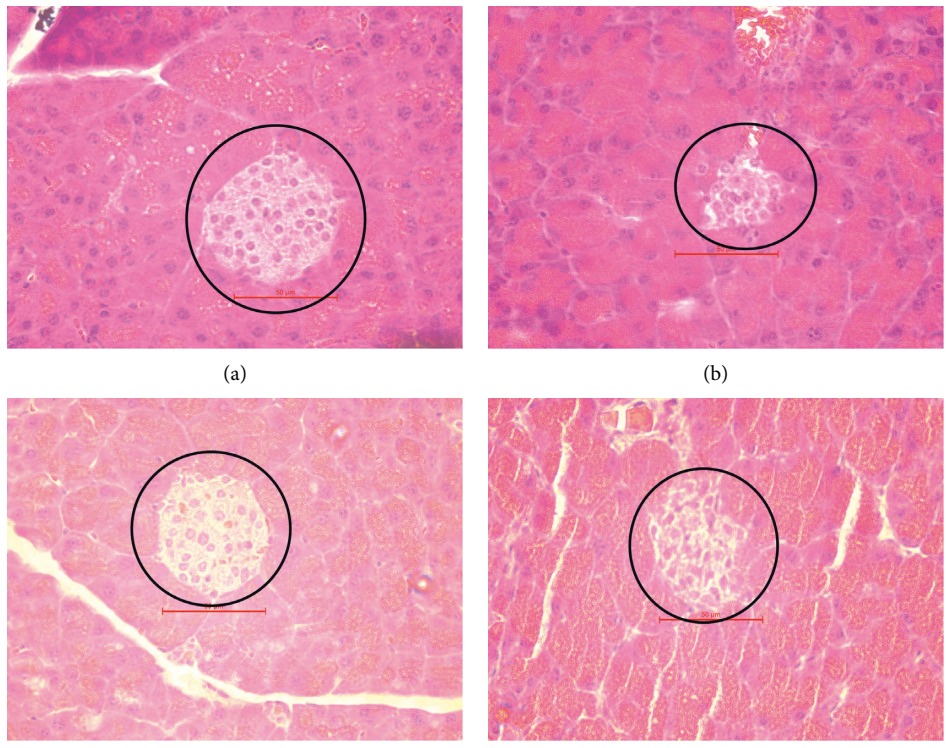


Figure 4: Pathological tissue sections from the pancreas of normal and type 2 diabetic mouses. (a) Nondiabetic group. (b) Diabetes model group. (c) Metformin group. (d) SDF group.

glucose, leading to type 2 diabetes. After the intake of SDF, the islet area and the number of β cells in diabetic mouses were found to increase, showing varying degrees of repair on the pancreas islet and β cells. Ma and Mu suggested that SDF significantly repairs pancreas islet and β cells, thus improving the secretion of INS and glycogen synthesis and relieving the symptoms of diabetes [11]. The results presented here are consistent with those of the abovementioned studies.

4. Conclusions

The intragastric administration of SDF in type 2 diabetic mouses was found to effectively regulate the physiological indexes of food intake, water intake, body weight, and blood glucose levels, as well as promote the secretion of INS, increase the sensitivity to INS, enhance the antioxidant enzyme capacity and lipid metabolism of the liver, and repair pancreatic injury in diabetic mouses to effectively inhibit

pancreas islet lesions. Therefore, SDF can be used as an effective supplementary reagent during the prevention and treatment of type 2 diabetes. However, due to the complex components of SDF, the prevention and the treatment of type 2 diabetes do not share the same mechanism. Consequently, studies on the proteomic differences between samples from the pancreas, liver, kidney, spleen, and other organs are needed to determine the underlying mechanism by which SDF alleviates diabetic hyperglycemia.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Acknowledgments

This research was supported by grants from the National Key Research and Development Program of China "Technologies for Regulation and Activity Maintenance of Active Components of Coarse Cereals During Processing and Their Application" (no. 2017YFD0401203), the active components of miscellaneous grains and beans were preserved during processing and storage; Heilongjiang Farms & Land Reclamation Administration Technology Project "Pilot Test and Conversion of Coarse Cereal Processing Technology" (no. HNK135-05-02); Special Fund Project for the Central Guided Local Development of Science and Technology "Generalization of Soybean, Coarse Cereal and Other Varieties and Conversion of Their Processing Technologies" (no. ZY16C07); and Graduate Student Innovation Project of Heilongjiang Bayi Agricultural University (no. YJSCX2018-Y56). The authors thank Pr. Longkui Cao and researcher Weihao Wang for their guidance on the experimental design and Yunfei Ge, Ziyue Kang, Juan Wang, Zhigang Quan, and Jinling Xiao for their help in the experimental process.

References

[1] D. R. Whiting, L. Guariguata, C. Weil, and J. Shaw, "IDF diabetes atlas: global estimates of the prevalence of diabetes for 2011 and 2030," *Diabetes Research and Clinical Practice*, vol. 94, no. 3, pp. 311–321, 2011.

[2] P. Bigoniya, R. Nishad, and C. S. Singh, "Preventive effect of sesame seed cake on hyperglycemia and obesity against high fructose-diet induced Type 2 diabetes in rats," *Food Chemistry*, vol. 133, no. 4, pp. 1355–1361, 2012.

[3] F. Nagatomo, N. Gu, H. Fujino, I. Takeda, K. Tsuda, and A. Ishihara, "Skeletal muscle characteristics of rats with obesity, diabetes, hypertension, and hyperlipidemia," *Journal of Atherosclerosis and Thrombosis*, vol. 16, no. 5, pp. 576–585, 2009.

[4] A. Douros, S. Dell'Aniello, O. H. Y. Yu, K. B. Filion, L. Azoulay, and S. Suissa, "Sulfonylureas as second line drugs in type 2 diabetes and the risk of cardiovascular and hypoglycaemic events: population based cohort study," *British Medical Journal*, vol. 362, p. k2693, 2018.

[5] D. A. Brockman, X. Chen, and D. D. Gallaher, "Hydroxypropyl methylcellulose, a viscous soluble fiber, reduces insulin resistance and decreases fatty liver in Zucker diabetic fatty rats," *Nutrition & Metabolism*, vol. 9, no. 100, pp. 1–12, 2012.

[6] Y. Zhang, *Study of Dietary Diabetes Fibre's Mellitus Hypoglycemic Effect on Type 2 Rats and its Mechanism*, Nanchang University, Nanchang, China, 2011.

[7] M. Galisteo, R. Morón, L. Rivera, R. Romero, A. Anguera, and A. Zarzuelo, "Plantago ovata husks-supplemented diet ameliorates metabolic alterations in obese Zucker rats through activation of AMP-activated protein kinase. Comparative study with other dietary fibers," *Clinical Nutrition*, vol. 29, no. 2, pp. 261–267, 2010.

[8] R. K. Toivonen, R. Emani, E. Munukka et al., "Fermentable fibres condition colon microbiota and promote diabetogenesis in NOD mice," *Diabetologia*, vol. 57, no. 10, pp. 2183–2192, 2014.

[9] K. Weitkunat, S. Schumann, K. J. Petzke, M. Blaut, G. Loh, and S. Klaus, "Effects of dietary inulin on bacterial growth, short-chain fatty acid production and hepatic lipid metabolism in gnotobiotic mice," *The Journal of Nutritional Biochemistry*, vol. 26, no. 9, pp. 929–937, 2015.

[10] F. Isken, S. Klaus, M. Osterhoff, A. F. H. Pfeiffer, and M. O. Weickert, "Effects of long-term soluble vs. insoluble dietary fiber intake on high-fat diet-induced obesity in C57BL/6J mice," *The Journal of Nutritional Biochemistry*, vol. 21, no. 4, pp. 278–284, 2010.

[11] M. Ma and T. Mu, "Anti-diabetic effects of soluble and insoluble dietary fibre from deoiled cumin in low-dose streptozotocin and high glucose-fat diet-induced type 2 diabetic rats," *Journal of Functional Foods*, vol. 25, pp. 186–196, 2016.

[12] A. Mohammed, N. A. Koorbanally, and M. S. Islam, "Ethyl acetate fraction of Aframomum melegueta fruit ameliorates pancreatic β-cell dysfunction and major diabetes-related parameters in a type 2 diabetes model of rats," *Journal of Ethnopharmacology*, vol. 175, pp. 518–527, 2015.

[13] I. Cordero-Herrera, M. Á. Martín, F. Escrivá, C. Álvarez, L. Goya, and S. Ramos, "Cocoa-rich diet ameliorates hepatic insulin resistance by modulating insulin signaling and glucose homeostasis in Zucker diabetic fatty rats," *The Journal of Nutritional Biochemistry*, vol. 26, no. 7, pp. 704–712, 2015.

[14] Z. C. Tu, Z. Li, and G. Chen, "Hypoglycemic effect of soluble soybean dietary fiber," *Food Science*, vol. 30, no. 17, pp. 294–296, 2009.

[15] A. Uskoković, M. Mihailović, S. Dinić et al., "Administration of a β-glucan-enriched extract activates beneficial hepatic antioxidant and anti-inflammatory mechanisms in streptozotocin-induced diabetic rats," *Journal of Functional Foods*, vol. 5, no. 4, pp. 1966–1974, 2013.

[16] Y. W. Wang, Y. J. Wang, and C. Y. Li, "Effect of dietary fiber on blood sugar in diabetic patients," *Food Industry Technology*, vol. 22, no. 5, pp. 25–27, 2001.

[17] H. C. Hou and J. S. Li, "Physiological function and clinical application of dietary fiber," *Journal of Clinical Medicine*, vol. 8, no. 5, pp. 12–15, 2004.

[18] X. F. Wu, "Development and application of dietary fiber," *Journal of Shanxi College of Traditional Chinese Medicine*, vol. 5, no. 2, pp. 58-59, 2004.

[19] N. Wang, D.-Y. Guo, X. Tian et al., "Niacin receptor GPR109A inhibits insulin secretion and is down-regulated in type 2 diabetic islet beta-cells," *General and Comparative Endocrinology*, vol. 237, pp. 98–108, 2016.

[20] T. Kadowaki, "Insights into insulin resistance and type 2 diabetes from knockout mouse models," *Journal of Clinical Investigation*, vol. 106, no. 4, pp. 459–465, 2000.

[21] U. B. Pajvani, C. J. Shawber, V. T. Samuel et al., "Inhibition of Notch signaling ameliorates insulin resistance in a FoxO1-dependent manner," *Nature Medicine*, vol. 17, no. 8, pp. 961–967, 2011.

[22] V. A. Barr, D. Malide, M. J. Zarnowski, S. I. Taylor, and S. W. Cushman, "Insulin stimulates both leptin secretion and production by rat white adipose tissue," *Endocrinology*, vol. 138, no. 10, pp. 4463–4472, 2011.

[23] M.-J. Lee and S. K. Fried, "Multilevel regulation of leptin storage, turnover, and secretion by feeding and insulin in rat adipose tissue," *Journal of Lipid Research*, vol. 47, no. 9, pp. 1984–1993, 2006.

[24] H. M. Zhao, Z. L. Lei, and J. Guo, "Advances of the contradictory relationship between plasma adiponectin level and type 2 diabetes mellitus and cardiovascular disease," *Chinese Journal of Cell Biology*, vol. 40, no. 5, pp. 820–826, 2018.

[25] L. Qi, E. Rimm, S. Liu, N. Rifai, and F. B. Hu, "Dietary glycemic index, glycemic load, cereal fiber, and plasma adiponectin concentration in diabetic men," *Diabetes Care*, vol. 28, no. 5, pp. 1022–1028, 2005.

[26] L. Qi, R. M. van Dam, S. Liu, M. Franz, C. Mantzoros, and F. B. Hu, "Whole-grain, bran, and cereal fiber intakes and markers of systemic inflammation in diabetic women," *Diabetes Care*, vol. 29, no. 2, pp. 207–211, 2006.

[27] D. M. Kemp and J. F. Habener, "Synergistic effect of dimethyl sulfoxide on glucagon-like peptide 1 (GLP-1)-stimulated insulin secretion and gene transcription in INS-1 cells: characterization and implications," *Biochemical Pharmacology*, vol. 64, no. 4, pp. 689–697, 2002.

[28] C. L. Adam, P. A. Williams, M. J. Dalby et al., "Different types of soluble fermentable dietary fiber decrease food intake, body weight gain and adiposity in young adult male rats," *Nutrition & Metabolism*, vol. 11, no. 36, pp. 1–12, 2014.

[29] H. V. Lin, A. Frassetto, E. J. Kowalik et al., "Butyrate and propionate protect against diet-induced obesity and regulate gut hormones via free fatty acid receptor 3-independent mechanisms," *PLoS One*, vol. 7, no. 4, Article ID e35240, 2012.

[30] C. F. Hansen, M. Bueter, N. Theis et al., "Hypertrophy dependent doubling of L-cells in Roux-en-Y gastric bypass operated rats," *PLoS One*, vol. 8, no. 6, Article ID e65696, 2013.

[31] A. O. Ademiluyi and G. Oboh, "Attenuation of oxidative stress and hepatic damage by some fermented tropical legume condiment diets in streptozotocin-induced diabetes in rats," *Asian Pacific Journal of Tropical Medicine*, vol. 5, no. 9, pp. 692–697, 2012.

[32] M. Dhibi, F. Brahmi, A. Mnar et al., "The intake of high fat diet with different trans fatty acid levels differentially induces oxidative stress and non alcoholic fatty liver disease (NAFLD) in rats," *Nutrition & Metabolism*, vol. 8, no. 1, p. 65, 2011.

[33] H. Nyblom, H. Nordlinder, and R. Olsson, "High aspartate to alanine aminotransferase ratio is an indicator of cirrhosis and poor outcome in patients with primary sclerosing cholangitis," *Liver International*, vol. 27, no. 5, pp. 694–699, 2007.

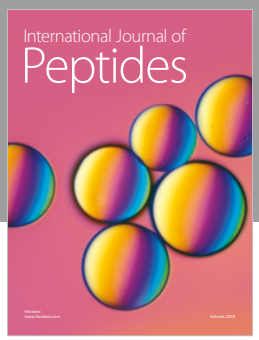
[34] I. Mateos-Aparicio, C. Mateos-Peinado, and P. Rupérez, "High hydrostatic pressure improves the functionality of dietary fibre in okara by-product from soybean," *Innovative Food Science & Emerging Technologies*, vol. 11, no. 3, pp. 445–450, 2010.

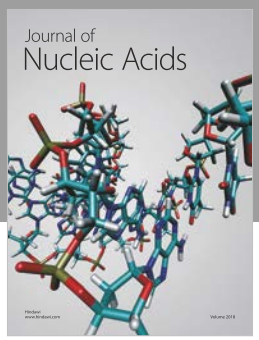
[35] F. D. Li, *Intervention Effects of Okara, Okara Protein and Dietary Fiber on Type 2 Diabetes and the Mechanism*, Huazhong Agricultural University, Wuhan, China, 2015.

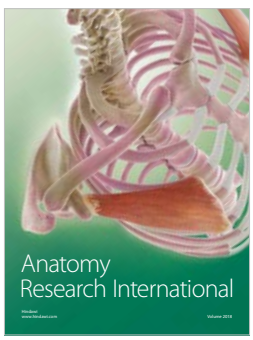
[36] H. F. Bakkeren, F. Kuipers, R. J. Vonk, and T. J. C. Van Berkel, "Evidence for reverse cholesterol transport in vivo from liver endothelial cells to parenchymal cells and bile by high-density lipoprotein," *Biochemical Journal*, vol. 268, no. 3, pp. 685–691, 1990.

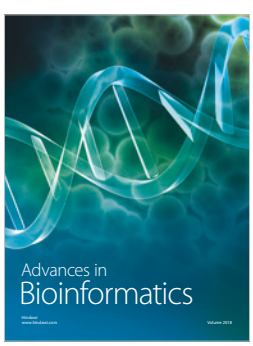
[37] S. M. Aurelian, D. M. Cheta, and D. Onicescu, "Microvesicles-potential biomarkers for the interrelations atherosclerosis/type 2 diabetes mellitus," *Romanian Journal of Morphology and Embryology*, vol. 55, no. 3, pp. 1035–1039, 2014.

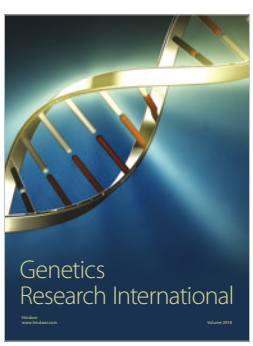
[38] X. M. Wang, *Study on the Extraction of Sweet Potato Dietary Fiber and its Prevention, Treatment Effect on Obesity of Wistar Rat*, China Agricultural University, Beijing, China, 2013.

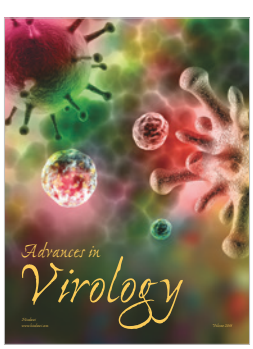


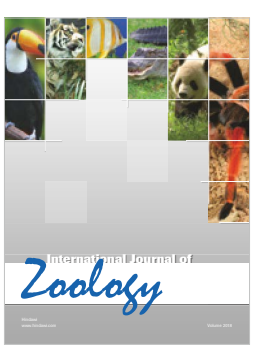


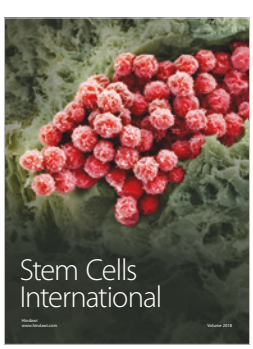


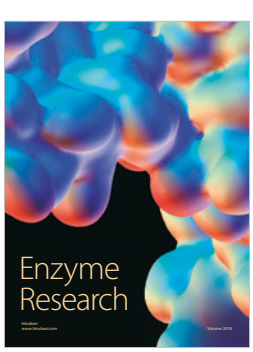


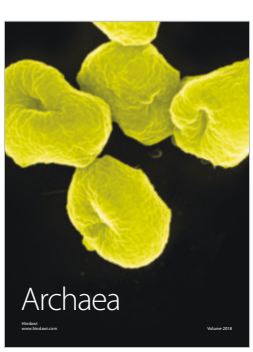













Hindawi

Submit your manuscripts at
www.hindawi.com