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Hypoglycemic Effects and Biochemical Mechanisms of Oat Products on Streptozotocin-Induced Diabetic Mice

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ABSTRACT: Oat products are abundant in β-glucan, which could lower the glycemic index of products or foods. A low glycemic index is beneficial in the control of postprandial glycemia. The study examined the hypoglycemic effects of oat products that had the same percentage of oat β-glucan and were added into the diet fed to streptozotocin-induced diabetic mice for 6 weeks, and potential mechanisms are discussed here. Oat products significantly decreased fasting blood glucose and glycosylated serum protein ($p < 0.05$), but the hypoglycemic effect was not more than that of metformin ($p > 0.05$). Oat products increased glycogen, hormone, and nuclear receptor levels ($p < 0.05$), decreased free fatty acid content and succinate dehydrogenase activity ($p < 0.05$), and inhibited pancreatic apoptosis ($p < 0.05$). The results showed oat products had hypoglycemic effects. Hypoglycemic effects of oat products might be regulating glucose and fat metabolisms, stimulating hormone secretion, activating the nuclear receptor, and protecting organ function.

KEYWORDS: oat products, type II diabetes mellitus, hypoglycemic effects, hypoglycemic mechanism

INTRODUCTION

The beneficial effects of healthy foods on quality of life and cost-effectiveness of health care have prompted the food industry to face the challenge of developing new food products with special nutrition and physiology. Minor crops such as oat, barley, rye, and buckwheat were reported to be rich in soluble dietary fiber measured by an enzymatic−gravimetric method.[1] In addition, dietary fiber plays an important part in people's health.[2] Therefore, more and more people have begun to focus on minor crops that have been underutilized in animal feeding in the past decades.

Type II diabetes mellitus (T2DM) is characterized with insulin resistance and reduced responsiveness of the pancreatic cells to glucose, ultimately leading to hyperglycemia and associated complications.[3] Low glycemic index (GI) diets could plausibly decrease the risk of T2DM by decreasing plasma glucose and insulin responses.[4−7] With extensive studies, food products incorporated with soluble dietary fiber β-glucan predictably reduced the GI, making β-glucan a useful functional food component for reducing postprandial glycemia.[8−10]

Oats are recognized as a "third staple food" in China. Oat products including oat bran, oat flour, and rolled oat are abundant in soluble fiber β-glucan. Moreover, oat products have a low GI, which would be helpful to maintain homeostasis of glucose metabolism in T2DM. Therefore, one purpose of the present study was to investigate the hypoglycemic effects of the three different oat products, each having the same weight of β-glucan (2000 mg L^{-1} body weight) but different GI, on streptozotocin (STZ)-induced diabetic mice. Recent papers on the mechanisms of oat products focused on β-glucan in healthy people;[11] this study paid attention to oat β-glucan and GI in diabetic mice. Another aim was to discuss the hypoglycemic mechanisms of oat products on STZ-induced diabetic mice.

MATERIALS AND METHODS

Reagents and Materials. Quantification of β-glucan was performed with an enzymatic method[12] using a Megazyme kit (Megazyme, Wicklow, Ireland) in triplicate for all of the samples. The main components of oat bran (Jinlvhe Biotechnology Co., Ltd., Shanxi, China), oat flour (Member Food Co., Ltd., Hebei, China), and rolled oat (Guilin Seamild biological Science and Technology Co., Ltd., Guangxi, China) are shown in Table 1A.

STZ was purchased from Sigma Chemical Co. (St. Louis, MO). Metformin was purchased from Pharmaceutical Group Co., Ltd. (Jiangsu, China). Other chemicals were of analytical reagent grade.

Animals. Male KM mice (4 weeks of age, weighing 20.11 ± 0.58 g) were purchased from the Laboratory Animal Center of Henan Province (Zhengzhou, China). Mice were allowed to acclimatize for 3 days in an environmentally controlled room at a temperature of 23 ± 1 °C and a relative humidity of 60% with an alternating 12 h light−dark cycle; the mice had free access to the standard diet (Laboratory Animal Center of Henan Province) and water. The nutrient composition of the standard diet is shown in Table 1B. All procedures using animals were reviewed and approved by the Committee on Care and Use of Laboratory Animals, China Pharmaceutical University.

Induction of Type II Diabetic Mouse Model and Experimental Treatments. After the 3 day acclimatization period, all of the mice were divided into two groups: group 1 included 15 mice that were given the standard diet throughout the experiment; group 2 included 85 mice that were given a high-sucrose and high-fat diet (10% lard, 10% sucrose, 5% cholesterol, 75% standard diet) for 4 weeks. Then group 1 and 2 mice were fasted for 12 h (free access to water). Each mouse of group 2 was injected with STZ (200 mg kg^{-1} body weight;

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Table 1. Chemical Analysis of Oat Products Used for the Experiment and Nutrient Composition of the Standard Diet

(A) Chemical Analysis of Oat Products Used for the Experiment

content[a] (%)	oat bran	oat flour	rolled oat
β-glucan	13.58 ± 2.58	3.20 ± 0.29	7.31 ± 1.22
dietary fiber	20.52 ± 1.38	5.80 ± 1.11	11.60 ± 3.04
starch	60.12 ± 5.18	75.70 ± 5.60	65.00 ± 4.36
resistance starch	2.09 ± 0.11	1.36 ± 0.02	1.88 ± 0.01
glycemic index	55.00 ± 3.50	77.00 ± 10.20	65.00 ± 6.10

(B) Nutrient Composition of the Standard Diet

component	moisture	crude protein	crude fat	crude fiber	crude ash	amino acid	microelement
value[b] (%)	9.15	23.02	4.22	4.00	7.28	2.28	2.81

[a] Expressed on moisture-free basis; means of duplicate determinations. [b] All determinations were based on GB.

intraperitoneally; in citrate buffer, pH 4.5). Meanwhile, each mousee of group 1 was injected with 0.9% NaCl solution (intraperitoneally) as the negative control (NC). The development of hyperglycemia of mice was confirmed by fasting blood glucose (FBG) estimation after 2 weeks of STZ injection. The animals that maintained FBG above 11.1 mM were considered to be diabetic and selected for studies. Then the diabetic mice of group 2 were subdivided into five groups including model control (MC), positive control (PC), oat bran (OB), oat flour (OF), rolled oat (OR) and were given a high-sucrose and high-fat diet (10% lard, 10% sucrose, 5% cholesterol). Besides, mice in groups MC, PC, OB, OF, and OR were fed 75% standard diet, 74% standard diet and 1% metformin, 71% standard diet and 4% OB, 58% standard diet and 17% OF, and 67.61% standard diet and 7.39% OR, respectively. The choice of oat products was by the same content of β-glucan (2000 mg kg^{-1} body weight). The choice of β-glucan dose (2000 mg kg^{-1} body weight) was decided in the preliminary study.[13] Metformin was used as PC because of its well-known antidiabetic effect.[14] Each group had 15 animals. Every treatment lasted for 6 weeks, during which mice had free access to food and water.

Samples of Blood and Organ Collection. Blood samples were collected from the tail vein of mice after a 12 h fast every 2 weeks. After 6 week of treatment, blood was collected from ocular puncture into a plastic centrifuge tube after an overnight fast. Serum was obtained after centrifugation at 2000*g* for 10 min before analysis. Afterward, all of the mice were anesthetized with ether absolute and sacrificed by decapitation. Immediately, pancreas, liver, and heart were dissected out, then washed in ice-cold saline solution to remove the blood; meanwhile, the pancreas was fixed in 10% neutral buffered formalin for histopathological examination.

Bioassay of Blood and Tissues. Fasting blood glucose (FBG) and glycosylated serum protein (GSP) were evaluated every 2 weeks. FBG was measured with glucose oxidase-peroxidase reagent method by CDC-1 glucometer (Nanjing Cambridge Medical Instrument Co., Ltd., Nanjing, China). Assays of GSP (20091113, 50T), free fatty acid (FFA) content (20091225, 50T), succinate dehydrogenase (SDH, EC 1.3.5.1, 20091225, 50T) used commercial kits that were purchased from Nanjing Jiancheng Bioengineering Institute (Nanjing, China). FFA was evaluated by a UV-1600 ultraviolet and visible spetrophotometer (Shanghai Mapada Instruments Co., Ltd., Shanghai, China); the others were measured by a BA-88A semiautomatic biochemical analyzer (Shenzhen Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, China). Serum fasting insulin (Fins), glucagon-like peptide-1 (GLP-1), and peroxisome proliferator activator receptors γ (PPARγ) were measured by a TECAN GENios microplate reader (Eastwin Life-Sciences, Serve & Push the Development of Chinese Life Science Industry, Beijing, China) using enzyme-linked immunosorbent assay kits (Diagnostic Extracorporeal Reagent, ADL CD-A55105, 96 determinations) of rat anti-mouse insulin, rat anti-mouse GLP-1, and rat anti-mouse PPARγ, respectively, which were purchased from Adlitteram Diagnostic Laboratories, Inc. Finally, the insulin sensitivity index (ISI) was calculated using the formula[15] ISI = (ln 1)/(Fins × FBG).

Liver and heart after excision from the animal were used to estimate the glycogen contents of liver and muscle with commercial kit 20091225, 50T according to the method of the anthrone reagent using the BA-88A semiautomatic biochemical analyzer.

Histopathological Study. The pancreas samples fixed by 10% neutral formalin were dehydrated by successive passing through a gradient of mixtures of ethyl alcohol and water. Then they were embedded in the paraffin after rinsing with xylene. Pancreatic sections (5 μm thick) were stained with hematoxylin and eosin stain for evaluation by light microscopy.

Statistical Analysis. Experiments were performed with 15 mice per group with values presented as the mean ± standard deviation. All of the studies were replicated with 10 representative data shown. Statistical analysis was performed using Student's *t* test for unpaired data or ANOVA and the Tukey—Kamer multiple-comparisons test post hoc. $p < 0.05$ and $p < 0.01$ was considered to be significant differences; $p > 0.05$ was considered to be insignificant difference.

RESULTS

Analysis of Composition for Oat Products. Compositions of oat products were related to glucose metabolism of diabetic mice. Therefore, the main chemical analysis of oat products before the experiment is listed in Table 1A. The data showed that oat bran (OB) was rich in β-glucan, dietary fiber, and resistant starch, which are beneficial to glucose metabolism; however, it was less rich in starch, which was the passive composition to T2DM.

Effects of Oat Products on Fasting Blood Glucose and Glycosylated Serum Protein of Diabetic Mice. Concentrations of fasting blood glucose (FBG) and glycosylated serum protein (GSP) demonstrated the hypoglycemic effects of oat products on diabetic mice after 6 weeks' dietary manipulation (Figure 1). Prior to the treatment, the FBG showed no significant difference ($p > 0.05$) among all of the diabetic mice; however, they were all significantly higher ($p < 0.01$) than normal controlled (NC) mice (Figure 1A). After 6 weeks of administration, FBG of positive controlled (PC) was significantly different ($p < 0.05$) versus NC mice; the difference of oat bran (OB), rolled oat (OR) was significant ($p < 0.05$) to PC; oat flour (OF) mice was significantly

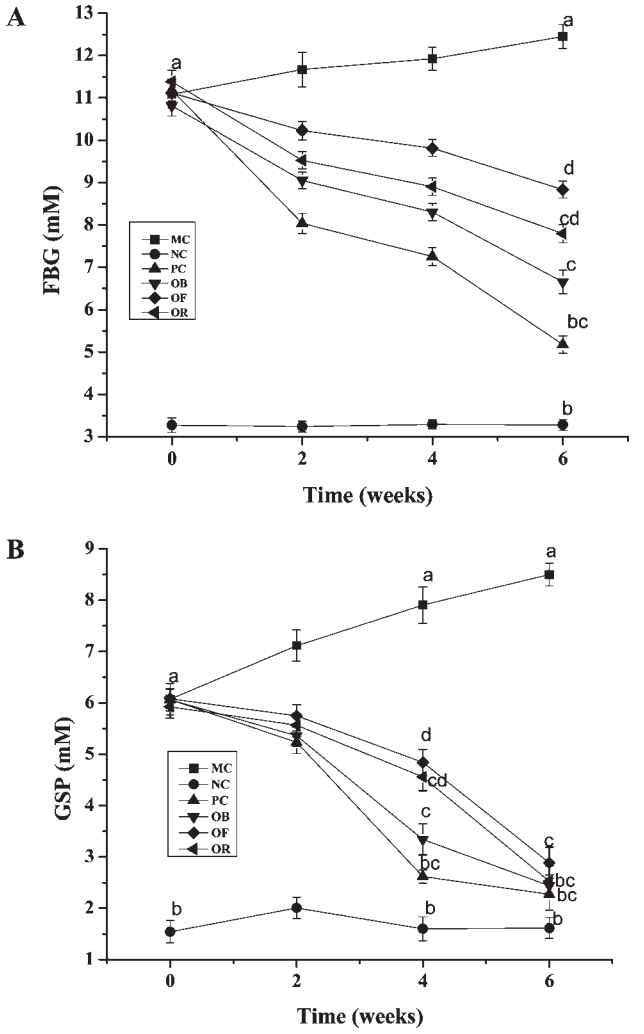

Figure 1. Effects of oat products on fasting blood glucose and glycosylated serum protein of diabetic mice. Values represent the mean ± SD, $n = 10$ each group. Abbreviations: FBG, fasting blood glucose; GSP, glycosylated serum protein; OB, oat bran; OF, oat flour; OR, rolled oat; MC, model control; PC, positive control; NC, negative control. Between groups, values that share the same letters are not significantly different ($p > 0.05$), those that share one letter are significantly different at $p < 0.05$, and those that have different letters are significantly different at $p < 0.01$.

different ($p < 0.05$) in comparison to OR; and they were all significantly different ($p < 0.01$) to model controlled (MC) mice.

Simultaneously, the GSP showed a trend similar to the changes of FBG (Figure 1B). At the beginning, all of the GSP concentrations of diabetic mice were significantly higher than those of the NC mice ($p < 0.01$), but 4 weeks later, the GSP of oat product mice had a significant decrease compared with MC mice. Moreover, OB mice were not significantly different ($p > 0.05$) compared with PC mice until to the end of the experiment, and the difference in both was significant ($p < 0.01$) in comparison to the MC group.

Effects of Oat Products on Glucose Metabolic Pathways of Diabetic Mice. Glycogen metabolism and the tricarboxylic acid cycle are important metabolic pathways of glucose. It showed effects of oat products on glycogen metabolism of liver and cardiac muscle; meanwhile, effects on the key enzyme of the tricarboxylic acid cycle were also shown (Table 2). Hepatic and muscle glycogen contents of all the diabetic mice were significantly ($p < 0.05$) lower than those of normal controlled (NC) mice; however, the groups treated with metformin and oat bran (OB) showed much higher glycogen concentration than model controlled (MC) group ($p < 0.05$). The glycogen content of OB mice was similar ($p > 0.05$) to that of the positive controlled (PC) mice, which suggested OB could effectively promote the synthesis of glycogen. The activities of succinate dehydrogenase (SDH) were very low in diabetic mice from the determination of MC mice, whereas the activities increased after treatment with metformin and oat products; especially the OB mice were significantly different ($p < 0.05$) compared with PC mice.

Effects of Oat Products on Hormone Secretion and Insulin Resistance of Diabetic Mice. Concentrations of insulin and glucagon-like peptide-1 (GLP-1) reflected the effects of oat products on hormone secretions of diabetic mice (Table 3). After 6 weeks, in comparison to model controlled (MC) mice, serum fasting insulin (Fins) contents tended to increase significantly ($p < 0.01$) in mice fed oat bran (OB) and rolled oat (OR) (Table 3). GLP-1 secretions of OR and oat flour (OF) mice increased slightly, but they were not significant compared to MC mice. Yet GLP-1 secretion of positive controlled (PC) mice was significantly different ($p < 0.05$) from that of OB mice, from which it could be found that OB would be a potential activator of GLP-1.

Concentrations of insulin sensitivity index (ISI) as well as free fatty acid (FFA) and peroxisome proliferators' activator receptors γ (PPARγ) indicated the effects of oat products on insulin resistance of diabetic mice (Table 3). ISI was calculated by the formula given under Materials and Methods. ISI in groups OB and OR declined significantly ($p < 0.01$); however, ISI in group OF was significantly ($p < 0.01$) higher versus PC mice. The FFA of PC and OB mice was significantly different versus OR ($p < 0.05$) and was also significantly different versus OF ($p < 0.01$), which indicated that OB and OR could decrease the FFA content to attenuate the toxic action of FFA to cells, and the OB would reach the level of metformin. Furthermore, the PPARγ content of OB mice was significantly different ($p < 0.05$) from that of PC mice, and OR mice were also significantly different ($p < 0.05$) from OB mice, which indicated that OB would be a possible PPARγ agonist and OR played a positive role, too.

Effects of Oat Products on Pancreas of Diabetic Mice. Figure 2 shows the pathologic features of pancreas tissues in each group, at the same time the number of islets and islet cells was counted in Table 4. The image for the normal controlled (NC) group illustrated that the islet was a round or oval-shaped cell mass, and the averages of islets and islet cells were 4.00 ± 1.93 and 74.50 ± 2.58 observed under an optical microscope at ×100 and ×400, respectively. The sizes of islets were different, and the volume of islets and the averages of islets and islet cells in the model controlled (MC) group were significantly less ($p < 0.01$) than those in the NC group. The islet and islet cell numbers increased significantly more ($p < 0.01$) in groups of oat bran, oat flour (OF), and positive control than in the MC group, but their numbers in the OF group were similar to those in the MC group ($p > 0.05$).

DISCUSSION

The study adopted a better-controlled and comparative design using STZ-induced diabetic mice in the perspective of physiological nutrition. After a 6 week intake of oat products, concentrations of fasting blood glucose (FBG), glycosylated serum protein

Table 2. Effects of Oat Products on Glucose Metabolic Pathways of Diabetic Mice[a]

	NC	MC	PC	OB	OF	OR
hepatic glycogen (mg g^{-1})	3.25 ± 0.12	0.09 ± 0.08 a	2.02 ± 0.13 b	1.22 ± 0.10 bc	0.30 ± 0.14 ad	0.422 ± 0.13 ae
muscle glycogen (mg g^{-1})	0.41 ± 0.12	0.00 ± 0.11 a	0.31 ± 0.11 b	0.23 ± 0.12 bc	0.09 ± 0.12 ad	0.10 ± 0.11 acd
SDH (U mL^{-1})	77.21 ± 2.15	13.74 ± 3.23	53.63 ± 2.14 a	44.09 ± 2.12 ab	31.27 ± 3.05	36.53 ± 4.13 bc

[a] Values represent the mean ± SD, $n = 10$ each group. Within the same row, the same letter indicates the difference is not significant ($p > 0.05$); one letter the same indicates the difference is significant at $p < 0.05$; and different letters indicate significant difference at $p < 0.01$. Abbreviations: SDH, succinate dehydrogenase; NC, negative control; MC, model control; PC, positive control; OB, oat bran; OF, oat flour; OR, rolled oat.

Table 3. Effects of Oat Products on Hormone Secretion and Insulin Resistance of Diabetic Mice[a]

	NC	MC	PC	OB	OF	OR
Fins (ng mL^{-1})	4.10 ± 0.14	0.66 ± 0.01 a	2.36 ± 0.08 b	1.81 ± 0.02 bc	0.97 ± 0.01 ad	1.29 ± 0.02 abd
GLP-1 (ng mL^{-1})	52.45 ± 2.03	3.96 ± 0.08 a	34.63 ± 2.22 b	27.41 ± 0.71 bc	10.24 ± 0.84 cd	14.10 ± 1.80 cd
ISI	0.06 ± 0.04	2.39 ± 0.04 a	1.06 ± 0.24 c	1.34 ± 0.18 c	2.00 ± 0.08 ab	1.75 ± 0.07 bc
FFA (μM)	350.98 ± 85.47	4349.01 ± 90.26 a	740.87 ± 91.20 b	948.98 ± 101.42 bc	1904.84 ± 89.50 ad	1440.19 ± 88.61 cd
PPARγ (pg mL^{-1})	481.87 ± 33.36	62.66 ± 0.16 a	240.89 ± 9.93 b	128.25 ± 2.45 bc	73.11 ± 0.84 ad	97.12 ± 2.47 bcd

[a] Values represent the mean ± SD, $n = 10$ per group. Abbreviations: Fins, serum fasting insulin; GLP-1, glucagon-like peptide-1; ISI, insulin sensitivity index, 1; FFA, free fat acid; PPARγ, peroxisome proliferators' activator receptors γ; OB, oat bran; OF, oat flour; OR, rolled oat; MC, model control; PC, positive control; NC, negative control. Within the same row, the same letter indicates the difference is not significant ($p > 0.05$); one identical letter indicates the difference is significant at $p < 0.05$; and different letters indicate highly significant difference at $p < 0.01$.

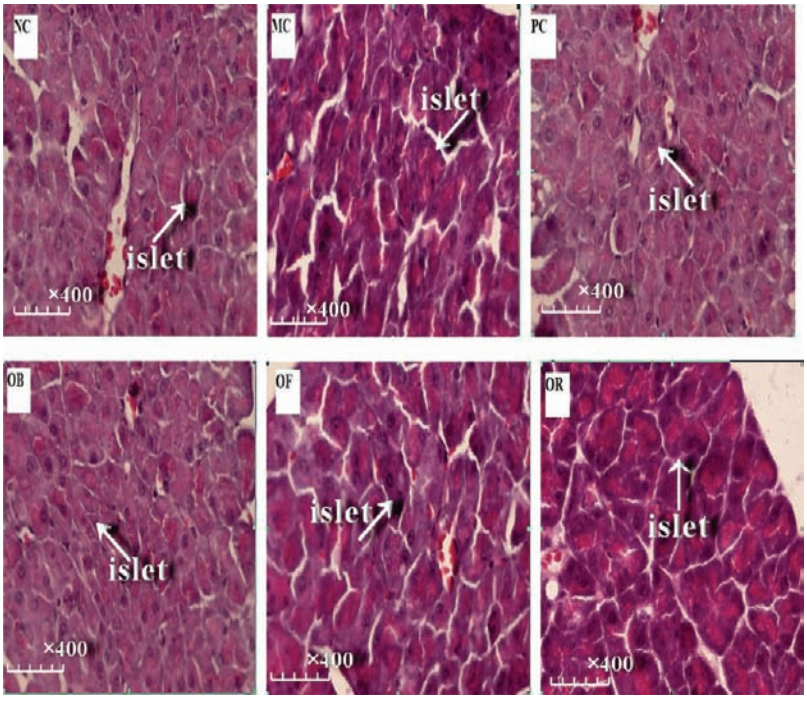

Figure 2. Effects of oat products on pancreatic structure of diabetic mice (hematoxylin and eosin stain, ×400). The arrows indicate the classical islet, and the scale is marked in the picture. Abbreviations: NC, negative control; MC, model control; PC, positive control; OB, oat bran; OF, oat flour; OR, rolled oat.

(GSP), and serum fasting insulin (Fins) declined significantly ($p < 0.05$) and the hypoglycemic effects of oat bran (OB) was the best ($p < 0.01$; Figure 1 and Table 3), which probably related to OB with lowest glycemic index (GI) and abundant fiber in comparison to oat flour (OF) and rolled oat (OR) (Table 1). Meanwhile, the study discussed the potentially hypoglycemic mechanisms underlying STZ-induced diabetic mice. It was shown that oat products stimulated the insulin and incretin hormone secretion (Table 3), regulated the glucose and fat metabolism (Tables 2 and 3), activated peroxisome proliferators' activator receptors γ (PPARγ, Table 3), and protected the normal physiological function of the pancreas (Table 4 and Figure 2). These would be associated with hypoglycemic effects.

Hypoglycemic effects of oat products may be associated with glucose metabolic pathways of diabetic mice.[16] Glycogen metabolism and tricarboxylic acid cycle are the two primarily

Table 4. Effects of Oat Products on Averages of Islets and Islet Cells of Diabetic Mice[a]

	NC	MC	PC	OB	OF	OR
average of islets (×100)	4.00 ± 1.93 a	0.88 ± 1.84 ef	2.13 ± 1.99 b	1.88 ± 1.83 bc	1.00 ± 1.76 de	1.38 ± 1.52 cd
average of islet cells (×400)	74.50 ± 2.58	8.88 ± 2.30 a	26.50 ± 3.25 d	19.63 ± 1.60 cd	10.25 ± 1.98 ab	12.38 ± 2.50 abc

[a] Values represent the mean ± SD, $n = 10$ per group. Within the same row, the same letter indicates the difference is not significant ($p > 0.05$); one identical letter indicates the difference is significant at $p < 0.05$; and different letters indicate significant difference at $p < 0.01$. Abbreviations: NC, negative control; MC, model control; PC, positive control; OB, oat bran; OF, oat flour; OR, rolled oat.

metabolic pathways of glucose, and they play important roles in effective glycemic control. The improvement of glycogenesis (Table 2) of diabetic mice fed oat products for 6 weeks indicated that oat products controlled blood glucose levels of diabetic mice by regulating the glycogen metabolism. Besides, the diabetic mice were more energetic than at the beginning with the treatment of oat products and metformin. Theoretically, glycogen mobilization of oat product mice would alleviate too much glucose to impair the normal function of the liver and muscle. As a result, it would increase glucose utilization of liver and skeletal muscles.

Succinate dehydrogenase (SDH) is a key enzyme of the tricarboxylic acid cycle. The chemically induced model of diabetes is characterized by a partial or total deficiency of insulin, which results in a decline of SDH activity.[17] In Table 2, SDH values of model controlled (MC) mice showed a significant decline ($p < 0.01$) over the entire experiment. As for the positive controlled (PC) mice, SDH activities of oat product mice also increased, which suggested that oat products could increase glucose utilization of diabetic mice. The increment of SDH activities can accelerate the oxidation of succinate. This suggested that oat products could improve the important enzyme activity of the tricarboxylic acid cycle, enhancing the glucose utilization of diabetic mice.

In addition, the hypoglycemic effects of oat products may be associated with hormone secretion and insulin resistance of diabetic mice. T2DM is increasingly viewed as a disease of insulin deficiency due not only to intrinsic pancreatic β-cell dysfunction but also to reduction of β-cell mass.[18] Table 3 shows that the insulin secretions of diabetic mice were deficient compared with the negative controlled (NC) mice; the histological results (Figure 2 and Table 4) also showed that the average numbers of islets and islet β-cells in the MC group were less than those in the NC group, but the mean numbers were somewhat elevated in oat products groups. Similarly, the concentrations of Fins of OB mice also rose to the level of Fins in the PC group, which suggested the pancreatic function of OB mice returned nearly to the normal level (Table 3). Glucagon-like peptide-1 (GLP-1) is a polypeptide hormone secreted from L cells in the gastrointestinal tract in response to the ingestion of nutrition.[19] GLP-1 is viewed as a potent agent for the therapeutic option in T2DM.[20,21] The study investigated serum GLP-1 in response to oat product intake, which would enhance the secretion of insulin from pancreatic cells.

T2DM is characterized by a high concentration of serum free fatty acid (FFA).[22] This study showed that MC mice had higher FFA levels than NC mice. However, FFA concentrations of diabetic mice treated with OB and OR showed a significant reduction ($p < 0.05$), which was not reported before (Table 3). Excessive quantities of FFA will inhibit β-cell function,[23] lipotoxicity may play a role in causing β-cell dysfunction and apoptosis of diabetes mellitus,[24,25], and lowering of FFA concentration may be potentially beneficial. The study found the histological improvement of pancreatic islet and pancreatic cell and the rise of the insulin sensitivity index (ISI), which suggested that the oat products were favorable to β-cell function and insulin sensitivity (Figure 2 and Tables 3 and 4).

PPARγ is a key regulator of glucose metabolism and the action of insulin.[26] Growing data demonstrate that PPARγ agonists improved insulin secretion in T2DM subjects.[27] By lowering FFA circulation, PPARγ agonists could inhibit excessive apoptosis and increase β-cell mass. A possible mechanism of the insulin resistance reduction of oat product mice was that oat products directed FFA away from the liver and cardiac muscle and stimulated adipose tissue to absorb FFA. As a result, oat products could decrease the serum FFA and improve the ISI and PPARγ to decrease insulin resistance level.

In summary, the study showed that oat products had hypoglycemic effects on STZ-induced diabetic mice, and the oat bran was the best as it was highest in β-glucan and lowest in glycemic index compared with oat flour and rolled oat. Furthermore, the results indicated that oat products increased glucose utilization to maintain glucose metabolism balance by increasing the secretion of insulin and GLP-1 and the important enzyme activity such as SDH in the pathway of the tricarboxylic acid cycle or promoting synthesis of tissue glycogen. Correspondingly, oat products decreased the FFA level and improved ISI and PPARγ, which would prevent pancreatic cell apoptosis and fatty degeneration to alleviate the insulin resistance level. Potentially, oat β-glucan or oat products rich in β-glucan would be functional food components as GLP-1 accelerators or PPARγ agonists.

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ABBREVIATIONS USED

T2DM, type II diabetes mellitus; GI, glycemic index; STZ, streptozotocin; NC, negtive control; FBG, fasting blood glucose; MC, model control; PC, positive control; OB, oat bran; OF, oat flour; OR, rolled oat; GSP, glycosylated serum protein; FFA, free fatty acid; SDH, succinate dehydrogenase; Fins, fasting insulin; GLP-1, glucagon-like peptide-1; PPARγ, peroxisome proliferators activator receptors γ; ISI, insulin sensitivity index.

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