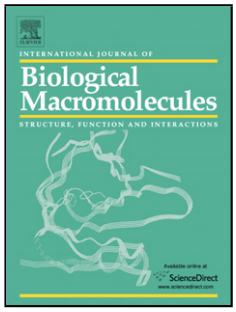

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Author: Mei Liu Yu Zhang Hui Zhang Bo Hu Li Wang Haifeng Qian Xiguang Qi

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Title

The anti-diabetic activity of oat β-D-glucan in streptozotocin–nicotinamide induced diabetic mice

Authors and affiliations: Mei Liu[1)], Yu Zhang[1)], Hui Zhang[1)] *, Bo Hu[1)], Li Wang[1)], Haifeng Qian[1)], Xiguang Qi [1)]

1) State Key Laboratory of Food Science and Technology & School of Food Science and Technology, Jiangnan University, Wuxi 214122, China

*** Corresponding author:** Hui Zhang, Ph.D.

Jiangnan University, State Key Laboratory of Food Science and Technology, 1800 Lihu Avenue, Wuxi, Jiangsu, China. TEL: +86-139-2117-7990 E-mail address: zhanghui@jiangnan.edu.cn

E-mail address: Mei Liu, waitliumei@foxmail.com; Yu Zhang, chumenghen123@gmail.com; Bo Hu, 615159020@qq.com; Li Wang, nmrwl@yahoo.com.cn; Haifeng Qian, 576589831@qq.com; Xiguang Qi, 307957312@qq.com

Abstract

This study was initiated to investigate the mechanism by which oat β-D-glucan (OBG) can control blood sugar levels and improve hepatogenic glycometabolism in streptozotocin-nicotinamide induced mice. After administration of different concentrations and molecular weights of β-D-glucan by oral gavage for 28 days, the body weight, fasting blood glucose, serum insulin, hepatic glycogen, glucose kinase and glucose-6-phosphatase activity of the diabetic mice were measured. In comparison with a negative control group (saline), β-D-glucan, especially medium or high doses of high-molecular-weight β-D-glucan, had a strong hypoglycaemic effect in streptozotocin-nicotinamide-induced mice. The mechanism of this effect may be associated with the high viscosity of the solution, an increase in insulin secretion, a decline in insulin resistance, and especially an improvement in hepatogenic glycometabolism. Moreover, β-D-glucan also markedly repaired and improved the integrity of pancreatic islet β-cell and tissue structures.

Keywords: β-D-glucan, diabetes, hepatogenic glycometabolism

1 Introduction

Diabetes is a chronic disease that is caused by insufficient insulin secretion, the action of insulin or insulin resistance. It can be classified into types 1 (insulin-dependent diabetes) and 2 (non-insulin-dependent diabetes)[1]. Obesity-associated diabetes is mainly found in Western countries[2], whereas the majority of diabetes that is not associated with obesity is found in eastern countries, such as China, Korea and Japan[2, 3]. The difference between non-obese and typical obese diabetes patients is that the reduction in insulin secretion seems to be more important than the increase in insulin resistance for non-obese diabetes[3]. The defect of insulin secretion leads to insulin resistance and results in a decline insulin secretion from the pancreatic β-cells. Insulin resistance and the toxic effects of hyperglycaemia are known partial etiological factors of non-obese diabetes[3]. Insulin resistance results in the long-term hypersecretion of insulin in response to blood glucose, which leads to hyperglycaemia toxicity to β-cells, even

result in apoptosis[3]. In addition, patients with non-obese diabetes have a high risk of cardiovascular disease[4].

β-D-glucan isolated from oat (*Avena sativa L.*), is comprised of linear cell wall homopolysaccharides of D-glucopyranose that are arranged as blocks of consecutive (1→4)-linked β-D-glucan residues separated by single (1→3)-linkages[5, 6]. The physiological activity of β-D-glucan has been attributed to its role in increasing viscosity in the upper digestive tract[7], for example, by helping to increase the expression of plasma peptide Y-Y and intestine peptide Y-Y in obese mice[8]. Some studies have reported the anti-diabetic effects of β-D-glucan. Thondre[9] reported that the glycaemic index (GI) values of chapatis containing 4 and 8 g of β-D-glucan were 43%-47% lower (GI, 30 and 29, respectively) than those of chapatis containing no β-D-glucan (GI, 54). The efficacy of oat β-D-glucan as a potential bioactive hypoglycaemic component is related to its structure, solubility, molecular weight and rheological characteristics[10]. Dong[11] found that oat β-D-glucan was a potential α-glycosidase inhibitor and decreased glucose absorption in the small intestine. However, few studies have focused on the effect of oat β-D-glucan on hypoglycaemic effects, insulin secretion and insulin resistance in non-obese diabetes, the incidence of which has recently begun to increase in East Asian populations. Therefore, the objective of this study was to investigate the mechanism by which oat β-D-glucan can control blood sugar levels and improve pancreatic β-cell function in streptozotocin–nicotinamide induced non-obese diabetic mice and compare the results with those of metformin. In addition, we compared the hypoglycaemic effect and hepatogenic glycometabolism of oat β-D-glucans of different molecular weights.

2.1 Materials and methods

2.1 Preparation and physicochemical properties of β-D-glucan

High-molecular-weight β-D-glucan (OBG500) was extracted from oat bran (purchased from Zhangjiakou, Hebei Province, China) according to the method described by Bhatty[12] and Ahmad[13], except that the solid : liquid ratio was 1:8 at pH 7.0. Samples

of medium-molecular-weight β-D-glucan (OBG40) and low-molecular-weight β-D-glucan (OBG7) were obtained from the initial isolate (OBG500) by controlling the time of acid hydrolysis (HCl, pH 1.0, 80 °C). The isolates were then subjected to exhaustive dialysis for 3 days and concentrated by freeze-drying. The total β-D-glucan content of each sample was determined by the AOAC Official Method 995.16, using an assay kit (Megazyme International, Bray, Ireland). The values of the molecular weights of the β-D-glucan samples were determined by size exclusion chromatography with multi-angle laser light scattering (SEC-LLS), according to Ma[14-16]. The amount of protein was determined using the Kjeldahl method. Moisture was analysed by drying weighed samples in a vacuum oven at 121°C for 5 h and measuring the weight loss. The intrinsic viscosity [η] of each β-D-glucan solution was determined at 37°C with an Ubbelohde capillary viscometer and calculations were made according to the Huggins equation. Rheological measurements were made on 3% (w/v) solutions of oat β-D-glucan. Viscosity was measured by a controlled strain rheometer (TA Instrument, USA) fitted with a cone-and-plate geometry (4°, 20 mm) and the steady shear rate sweep was carried out over shear rates from 0.1 s^{-1} to 100 s^{-1} at 37°C[17].

2.2 Establishment of the non-obese diabetic mouse model

The protocol for the animal experiments was approved by the Animal Research Ethics Boards of Jiangnan University. Male 4-week-old ICR mice were provided by the Experimental Animal Centre of Jiangnan University. All of the mice had free access to water and conventional food. After 1 week of acclimatisation, the group designated to receive medication was fasted for 12 h and then injected intraperitoneally with streptozotocin (50 mg/kg body weight； Sigma-Aldrich, St Louis, MO, USA) on two consecutive days. Nicotinamide (120 mg/kg body weight；Sigma-Aldrich, St Louis, MO, USA) was injected intraperitoneally 15 min before the first intraperitoneal injection of streptozotocin[2]. The non-diabetic mice were injected with saline alone. After 7 days, fasted blood glucose (FBG) levels ≥11 mmol/L were induced successfully.

2.3 Experimental design and biological analysis

The chemically induced diabetic mice were divided into seven treatment groups (abbreviations; daily oral gavage): a negative model group (NM group; saline), a positive model group (PM group; metformin, Sigma-Aldrich, Shanghai, China; 500 mg/kg body weight), a low-dose high-molecular-weight β-D-glucan-treated group (OBG500L group; 500 mg/kg body weight), a medium-dose high-molecular-weight β-D-glucan-treated group (OBG500M group; 1000 mg/kg body weight), a high-dose high-molecular-weight β-D-glucan-treated group (OBG500H group; 2000 mg/kg body weight), a medium-dose medium-molecular-weight β-D-glucan-treated group (OBG40M group; 1000 mg/kg body weight) and a medium-dose low-molecular-weight β-D-glucan-treated group (OBG7M group; 1000 mg/kg body weight). The normal mice served as a normal control group (NC group; saline). The mice were treated daily by oral gavage and individually housed in cages at 25°C and 55% (relative humidity) for 28 days.

Body weight, water intake and dietary intake of all of the experimental groups were determined every week. FBG levels were measured with a glucometer (One touch Ultra®; Johnson and Johnson Co., USA) at the end of every week. An oral glucose tolerance test was performed on day 29, D-glucose (2.0 g/kg body weight) was administered in the above mentioned groups, FBG was determined before and 2 h after glucose administration [18, 19]. At the end of the experiment, the mice were euthanised after an overnight fast with an overdose injection of sodium pentobarbital (150 mg/kg; Sinopharm, China). Serum insulin was assayed using enzyme-linked immunosorbent assay (ELISA) kits (Jianglai Bio, Shanghai, China). The liver glucokinase (GK) and glucose-6-phosphatase (G-6-Pase) activities were also determined with ELISAs (Jianglai Bio, Shanghai, China). Sections of the pancreas from the non-obese diabetic mice and normal mice were cleaned and fixed in 4% neutral formaldehyde solution, embedded in paraffin, serially sectioned, stained with hematoxylin/eosin (H&E), and observed by optical microscope (magnification 400×), according to Yuntao Liu[20] and Saravia[21]. The insulin sensitivity was evaluated by the Homeostatic Model Assessment-Insulin Resistance Index (HOMA-IR) and the Quantitative Insulin Sensitivity Check Index (QUICKI)[22]. HOMA-IR and QUICKI were calculated as:

$$\text{HOMA-IR} = I \times G/22.5,$$

where I is the fasting plasma insulin (μU/mL) and G is the fasting plasma glucose (mM) and

QUICKI = 1/[log (G') + log (I)],

where I is the fasting plasma insulin (μU/mL) and G' is the fasting plasma glucose (mg/dL).

2.4 Statistical analysis

The values were expressed as means ± standard deviation. The data were proceeded using DPS software (version 7.05; Rui Feng Technology Co. Ltd, Hangzhou City, Zhejiang Province, China)[23], and statistically significant were analysed using the analysis of variance (ANOVA) with Duncan's method comparisons test at $P < 0.05$.

3 Results

3.1 Physical and chemical properties of β-D-glucan

The intrinsic viscosity, molecular weight and composition of the β-D-glucan samples are presented in Table 1. All of the samples had a high β-D-glucan content (>80%, dry base) and a low protein content (<4.1%, dry base). The three intrinsic viscosities [η] that resulted from the low-, medium- and high-molecular-weight β-D-glucans differed significantly ($P < 0.05$), meaning that the viscosity of the three β-D-glucan solutions were different in the small intestine. The polydispersity index of β-D-glucan was less than 1.45, meaning that all of the three samples were within a narrow molecular distribution. The viscosity curves of the β-D-glucan solutions are depicted in Fig. 1. As the shear rate increased, the viscosities of all of the β-D-glucan solutions decreased. The viscosity was higher with increasing molecular weight, indicating that the viscosity of the isoconcentration β-D-glucan solution was positively correlated with the molecular weight.

3.2 Effects of β-D-glucan on body weight and water intake

No statistically significant difference ($P > 0.05$) in body weight was observed between

any of the mice before the induction of non-obese diabetes. During the experiment, the body weight of the diabetic mice increased regularly and was significantly lower ($P < 0.05$) than that of the non-diabetic mice (NC). However, the body weight of the NM group was higher than that of other diabetic mice from week 1 to week 4 and that of the OBG500 groups (OBG500L, OBG500M, and OBG500H) was slight lower than that of the OBG40M and OBG7M groups at week 4 (Table 2). This result may have been due to a higher dietary intake in the NM group than in the other diabetic group at the initial stage of non-obese diabetes, during which the body weight gain was maintained[3]. β-D-glucan can retard gastric emptying, increase satiety and decrease the appetite, which may reduce the rate of body weight gain. Water intake decreased in the following order (at week 4): NM > OBG40M > OBG500L > OBG7M > PM > OBG500H > NC ($P < 0.05$), indicating that medium doses and high doses of the high-molecular-weight β-D-Glucan were most effective in reducing water intake and urinary output than the other β-D-glucan treatments (Fig. 2).

3.3 Effects of β-D-glucan on FBG

Table 3 shows the FBG levels in the non-diabetic and diabetic mice. The differences in FBG between the non-obese diabetic mice (NC) were not statistically significant at week 0 ($P > 0.05$), but the FBG of the NM group was significantly higher ($P < 0.05$) than that of the other groups from week 1 to week 4. Both metformin and β-D-glucan significantly lowered FBG compared with the saline treatment (NM). However, the levels of FBG in the OBG500M and OBG500H groups were significantly lower ($P < 0.05$) than those of the other OBG and NM groups from week 1 to week 3. After 4 weeks, the FBG levels of OBG groups were significantly lower than of the NM group, but did not differ statistically significantly between the groups ($P > 0.05$) (Table 3). The medium (OBG 500M) and high (OBG 500H) doses of high-molecular-weight β-D-glucan were more effective in reducing blood sugar levels than the other β-D-glucan treatments at the initial stage.

3.4 Effects of β-D-glucan on oral glucose tolerance test

In the oral glucose tolerance test, the peak glucose concentration (maximum value at 60 min) in non-obese diabetic mice was significantly lower ($P < 0.05$) in the OBG groups than in the PM group. As the concentration of the same molecular weight of β-D-glucan in the solution increased (Fig. 3a), the peak glucose concentration increased. Fig. 3b shows that the peak glucose concentration decreased as the molecular weight of the β-D-glucan decreased. The results showed that β-D-glucan, especially at a high molecular weight and high dose, significantly reduced the postprandial blood glucose level and improved the glucose tolerance in non-obese type 2 diabetic mice.

3.5 Effects of β-D-glucan on insulin and insulin resistance

The results (Fig. 4a) showed that the level of serum insulin in the NC group at week 4 was significantly higher ($P < 0.05$) than that in the other diabetic groups. Although the levels of serum insulin in the OBG and PM groups decreased significantly ($P < 0.05$), they were still higher than that of the NM group. The levels in OBG7M, OBG40M and OBG500M were only decreased by 18.40%, 34.98% and 22.81%, respectively, and those in OBG500L, OBG500M and OBG500H were only decreased by 16.50%, 22.81%and 18.56%, respectively, whereas that in the NC group was decreased by 35.13%. Consequently, the HOMA-IR index (Fig. 4b) was lower and the QUICKI (Fig. 4c) was significantly higher ($P < 0.05$) in the OBG groups than in the NM group. The QUICKI increased and the HOMA-IR decreased as the concentration of oat β-D-glucan decreased. There was no significant difference ($P > 0.05$) in QUICKI and HOMA-IR between the different MWs OBG groups.

3.6 Effects of β-D-glucan on hepatogenic glycometabolism

A significant increase ($P < 0.05$) in the level of hepatic glycogen was observed in non-obese diabetic mice in the PM group compared with that in the NM and OBG groups

(Fig. 5a), but the differences in the level between the OBG groups were not statistically significant ($P > 0.05$). Although the hepatic glycogen levels in the OBG groups decreased significantly ($P < 0.05$) compared with the PM group, they were still higher than those in the NM group. Fig. 5b shows that the liver GK levels in the OBG groups were significantly higher ($P < 0.05$) than those in the NM group and no statistically significant difference ($P > 0.05$) was observed between the various molecular weight isodose OBG groups. With regard to the different doses of β-D-glucan, the GK level in the OBG500M group was significantly higher ($P < 0.05$) than that in the other OBG500 groups. Fig. 5c shows that the liver G-6-Pase levels in the NC group were significantly lower ($P < 0.05$) than that in the NM group. Moreover, the G-6-Pase levels in diabetic mice decreased in the following order: NM > OBG7M > OBG40M > PM > OBG500M and NM > OBG500H > OBG500L > PM > OBG500M. The decrease was inversely correlated with the molecular weight of the β-D-glucan.

3.7 Changes in the histopathology of the pancreas

The effects of β-D-glucan on pancreatic tissues stained with H&E are shown in Fig. 6. The pancreatic islets of the NC group were densely grouped with integrated membranes. The form and size of the pancreatic islets were significantly altered in the non-obese diabetic mice compared with the normal mice (NC). Hyperaemia and oedema were clearly observed in the pancreatic islets of the NM and OBG7 groups. Moreover, we observed focal necrosis, congestion in the central vein and infiltration of lymphocytes in the pancreas of the NM group, which implied that the destruction and changes in the function of islet cells in the NM group were more serious than those in the OBG and PM groups. However, treatment with PM, OBG500 and OBG40 for 28 days markedly repaired the islet damage and improved the integrity of pancreatic islet β-cell and tissue structures.

4 Discussion

The streptozotocin-nicotinamide-induced diabetic model, established by Nakamura [3]

and Lee [2], demonstrates characteristics that adequately reflect those of non-obese diabetes, the incidence of which has recently been increasing in East Asian populations. The non-obese model not only showed almost normal body weight gain and insulin secretion deficiency but also readily developed insulin resistance when fed with an excess of high-calorie western diet[3]. The body weight of all of the non-obese diabetic mice increased regularly from week 0 to week 4 (Table 2), consistent with the characteristics of non-obese diabetes in which the body weight increases or remains constant at the initial stage of diabetes. However, the typical clinical symptoms of polydipsia, polyphagia and polyuria[1], which become increasingly serious over time in non-obese diabetes, were alleviated by both β-D-glucan and metformin (Fig. 2).

The viscosity of β-D-glucan is known to play a key role in postprandial hyperglycaemia and insulin responses[24, 25]. Regand[25] reported that 73% of the bioactivity that attenuated the peak blood glucose response could be explained by the viscosity (or $Mw \times C$) of β-D-glucan. Brennan[26] and Thondre[9] partly attributed the hypoglycaemic effect of oat β-D-glucan to its influence on food microstructure, starch digestion and the contact of starch with digestive enzymes. Dong[11] found that oat β-D-glucan could inhibit the activities of intestinal disaccharidases *in vivo* and *in vitro*, which could affect the absorption rate of glucose in the small intestine. In our study, we found that oat β-D-glucan had hypoglycaemic effects in non-obese diabetes by restoring the histology structure of damaged pancreas cells and improving hepatogenic glycometabolism. Interestingly, the hypoglycaemic effects of the high-molecular-weight and high-dose β-D-glucan (OBG500M and OBG500H) were similar to those of the drug (metformin), whereas a decrease in the molecular weight reduced these effects. These results demonstrated that the production of oats with high-molecular-weight β-D-glucan could achieve the same effects as smaller meals, which have a beneficial influence in the dietary treatment of non-obese diabetes[27].

Insulin resistance, especially hepatic insulin resistance, plays a vital role in the initiation and progression of non-obese diabetes. Insulin has a direct influence on hepatic glucose metabolism. Any abnormality in the insulin signal transduction system of liver cells leads directly to insulin resistance[28]. Numerous studies have reported that serum

free fatty acids, which interfere with insulin signalling pathways[18, 29, 30], can be affected by β-D-glucan [31]. The HOMA-IR index and QUICKI mainly reflect the capacity for insulin resistance[22]. The HOMA-IR index was negatively correlated with the level of insulin resistance, whereas QUICKI was positively correlated with the level of insulin[22]. Figs. 4 and Figs. 5 show that oat β-D-glucan could improve the sensitivity to insulin in non-obese diabetes and reduce insulin resistance.

A significant increase in hepatic glucose output was observed in the β-D-glucan groups compared with the NM group, indicating that β-D-glucan can alleviate hepatic insulin resistance. The hypoglycaemic mechanism of metformin, which is often used as an oral insulin sensitiser, may partially depend on the promotion of the release of hepatic glucose[32]. Some reports showed that that β-D-glucan could be absorbed by small intestine and enter the tissues through lymph circulation and lymph circulation [33, 34]. We observed a significant increase in hepatic GK and a significant decrease in hepatic G-6-Pase in the OBG groups, implying that β-D-glucan could transfer more blood glucose into the liver by activating the glycolytic pathway and suppressing the gluconeogenic pathway[18]. Histopathological analysis revealed that administration of β-D-glucan could markedly repair and improve the integrity of pancreatic islet β-cell and tissue structures. Further, high-molecular-weight β-D-glucan protected the pancreatic tissues of diabetic mice more effectively than low- and medium-molecular-weight oat β-D-glucans. However, further investigation is necessary to delineate the exact molecular mechanism underlying the protective effect of β-D-glucan on hepatogenic glycometabolism and pancreatic islets.

In summary, our results provide evidence that oat β-D-glucan has a considerable hypoglycaemic effect in streptozotocin-nicotinamide induced non-obese diabetic mice and that the mechanism of its anti-diabetic effects involves the improvement of glucose tolerance, the increase of insulin secretion, the attenuation of insulin resistance and especially the protection of hepatogenic glycometabolism. The anti-diabetic effects of high-molecular-weight β-D-glucan are greater than those of low- and medium-molecular-weight β-D-glucans. More importantly, our study provides new insight into perspectives of developing oat β-D-glucan, which is helpful to improve hepatogenic glycometabolism, as a hypoglycemic food. However, further studies are necessary to

determine whether the anti-diabetic properties of β-D-glucan are still efficacious in non-obese diabetes mellitus after its long-term oral administration.

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Figure captions

Fig. 1. Viscosity curves of aqueous solutions of oat β-D-glucan of different molecular weights.

Fig. 2. Effect of β-D-glucan on water intake in diabetic and normal mice. Data are expressed as mean ± standard deviation (n=7). Error bars followed by different letters indicate significant difference ($P < 0.05$).

Fig. 3. Effect of (a), different concentrations and (b), different molecular weights of β-D-glucan on glucose tolerance in diabetic and normal mice. Data are expressed as mean ± standard deviation (n=7). Error bars followed by different letters indicate significant difference ($P < 0.05$).

Fig. 4. Effect of β-D-glucan on (a) insulin levels, (b) Homeostatic Model Assessment-Insulin Resistance (HOMA-IR) index, and (c) Quantitative Insulin Sensitivity Check Index (QUICKI) in diabetic and normal mice. Data are expressed as mean ± standard deviation (n=7). Error bars followed by different letters indicate significant difference ($P < 0.05$).

Fig. 5. Effect of β-D-glucan on (a) liver glycogen levels, (b) glucokinase (GK) levels, and

(c) glucose-6-phosphatase (G-6-Pase) levels in diabetic and normal mice. Data are expressed as mean ± standard deviation (n=7). Error bars followed by different letters indicate significant difference ($P < 0.05$).

Fig. 6. Effects of β-D-glucan on the histopathological changes in the tissues of the pancreas in diabetic with haematoxylin/eosin (H&E) staining (magnification 400×).

FIGURES

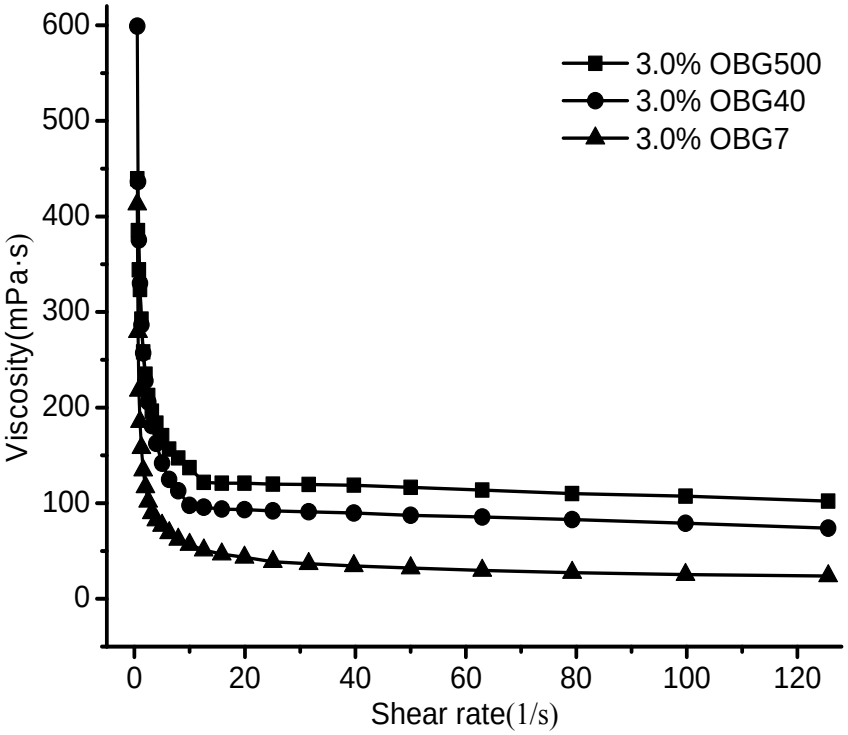

Figure 1

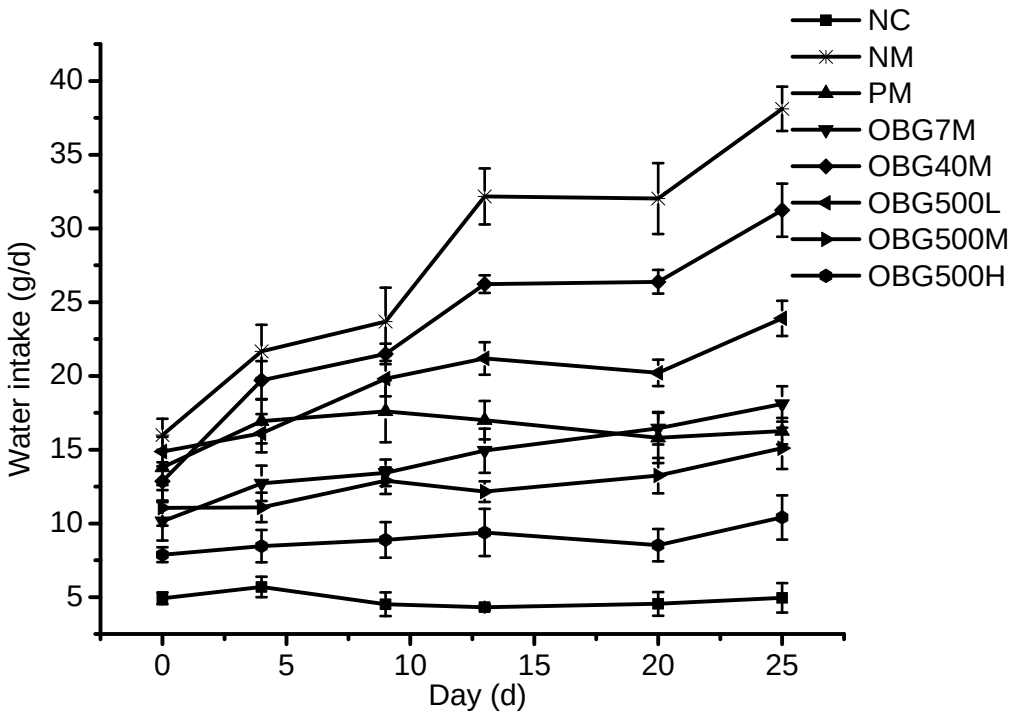

Figure 2

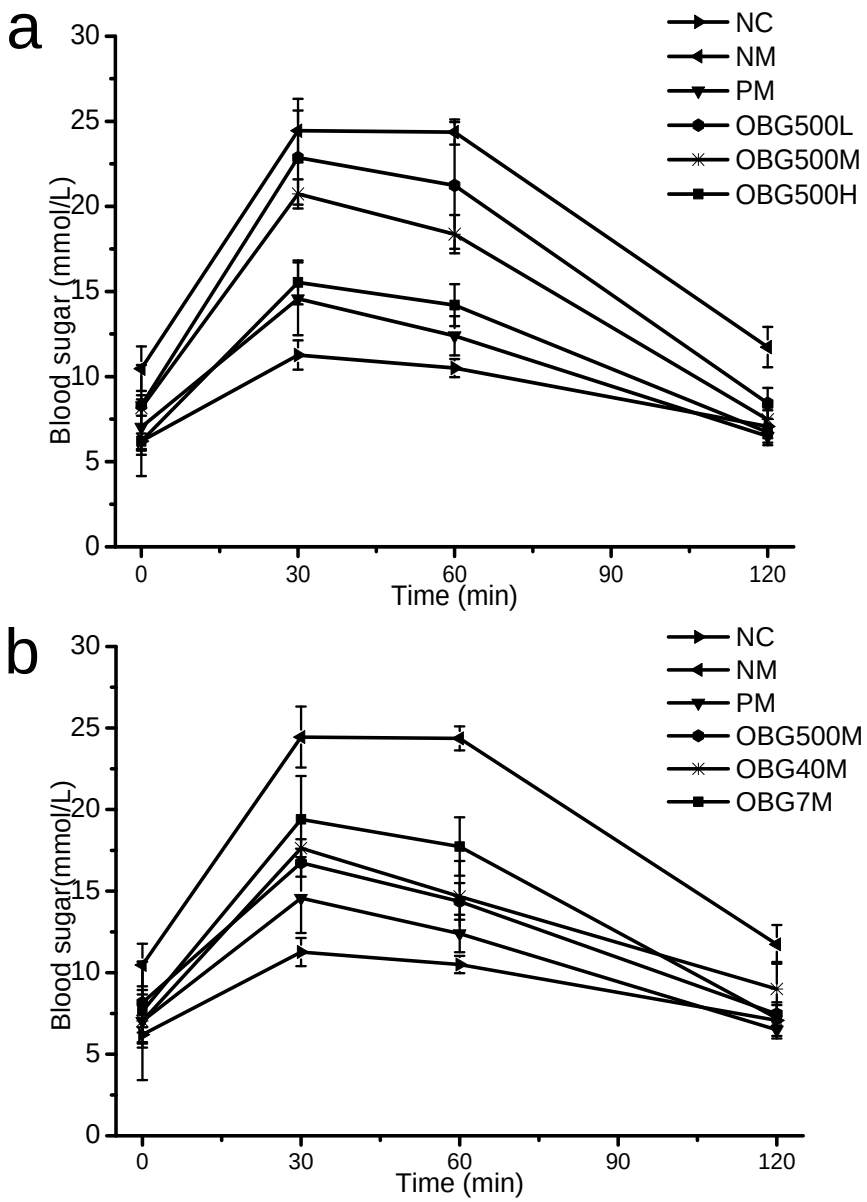

Figure 3

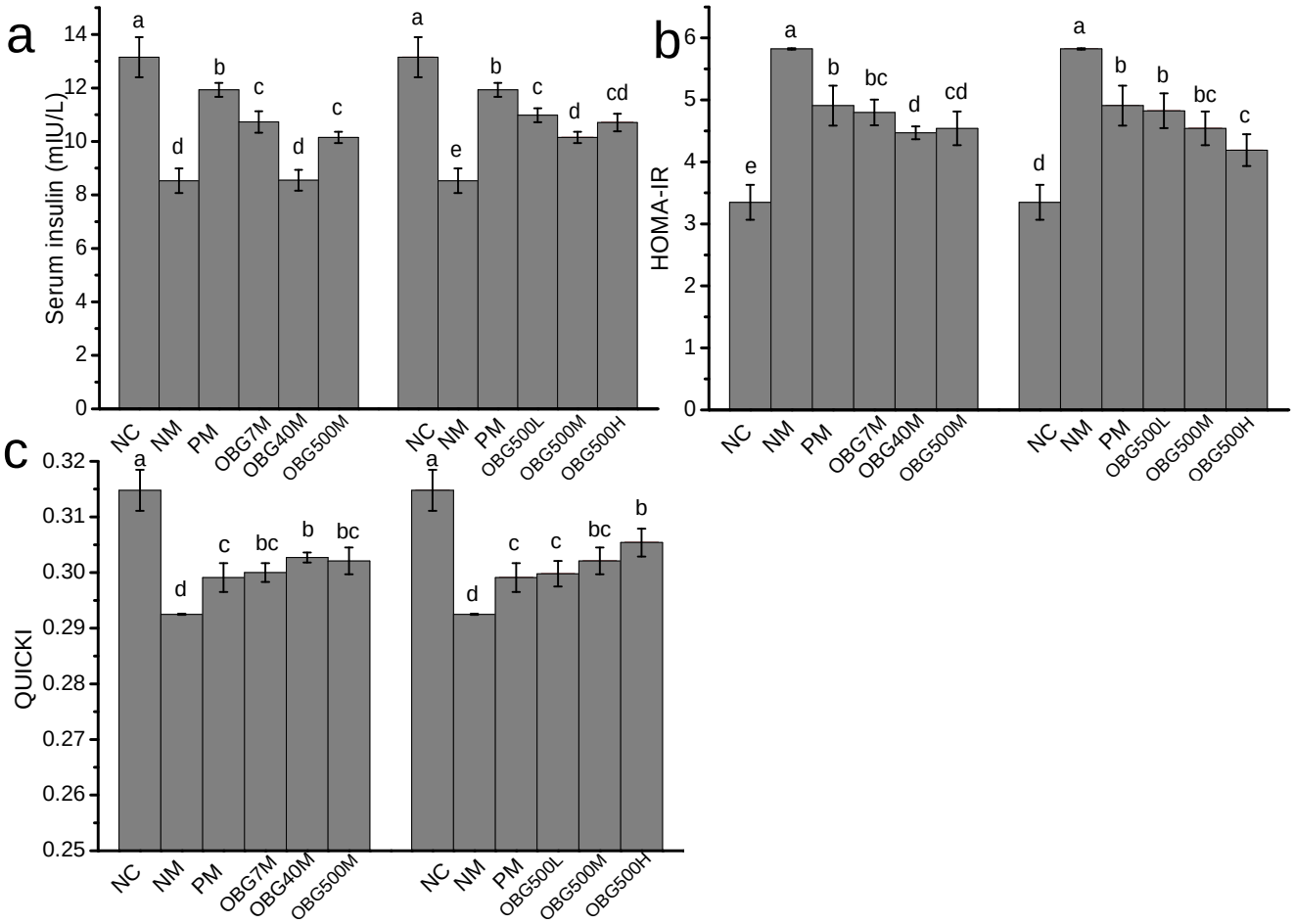

Figure 4

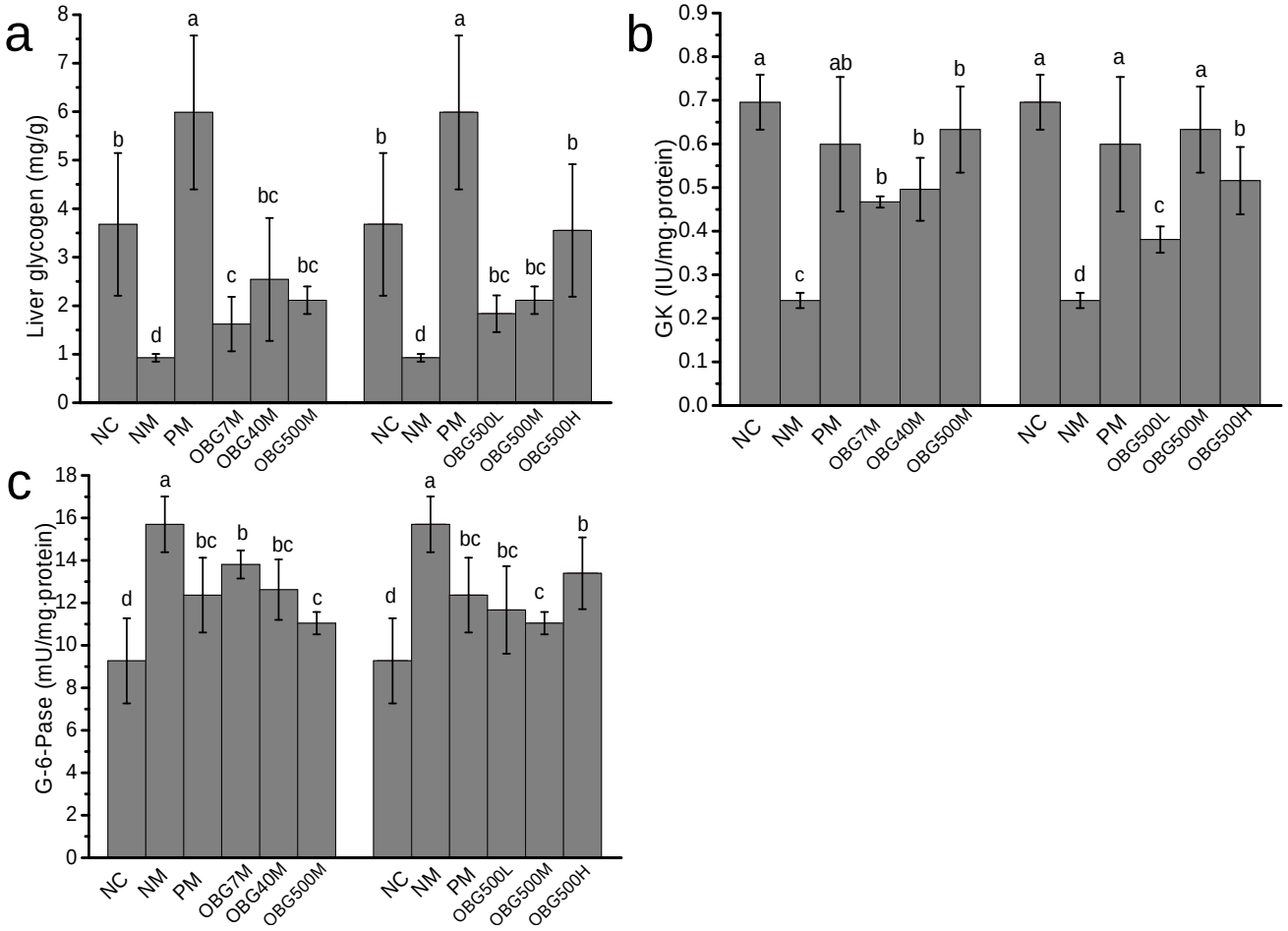

Figure 5

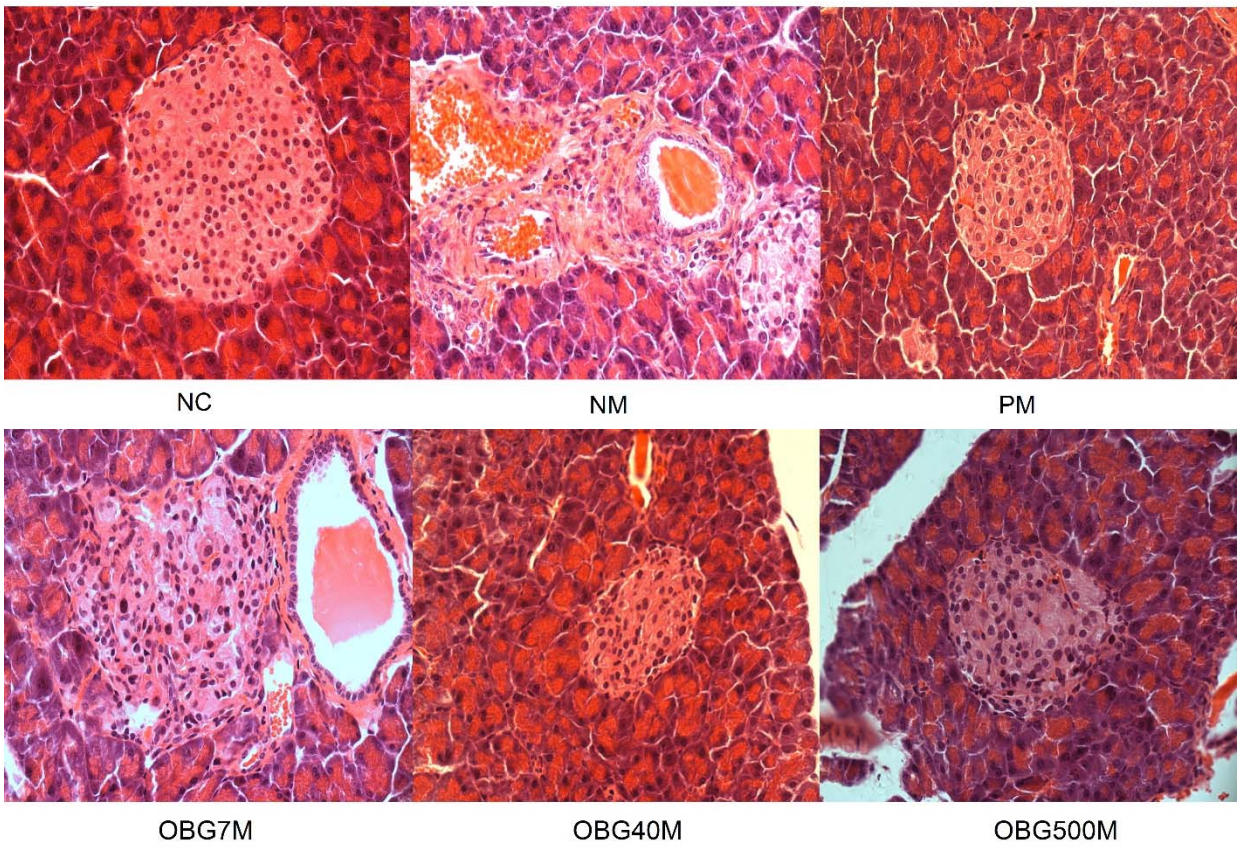

Figure 6

Table 1. Physicochemical properties of the oat β-D-glucan.

Samples	Intrinsic viscosity [η] (mL/g,37 °C)	$Mw \times 10^3$ (g/mol)	β-D-glucan (%, db)	protein(%, db)	Polydispersity index
OBG500	603.9±12.4^a	5,686.6±16.2^a	83.07±1.12^a	4.08±0.08^a	1.082±0.01^c
OBG40	421.9±18.7^b	461.2±1.2^b	82.76±0.24^a	3.99±0.12^a	1.419±0.02^a
OBG7	141.6±17.1^c	68.2±0.2^c	80.53±0.65^b	3.99±0.13^a	1.175±0.05^b

Data are expressed as mean ± standard deviation (n=3). Mean values in the same column followed by different superscript letters indicate significant difference ($P < 0.05$).

Abbreviations: db, dry base; *Mw*, weight-average molecular weight; OBG500, OBG40, and OBG7 represent oat β-D-glucan with different MWs.

Table 2. Effect of oat β-D-glucan on the body weight of diabetic and normal mice

Groups	Before injection (g)	Week 1 (g)	Week 2 (g)	Week 3 (g)	Week 4 (g)
NC	31.8±2.1^{a}	38.7 ±2.4^{a}	38.1 ±2.3^{a}	39.4 ±2.7^{a}	40.5 ±2.6^{a}
PM	31.6 ±1.6^{a}	35.8 ±2.9^{ab}	34.1 ±2.2^{bcd}	35.6 ±1.8^{bc}	37.2 ±2.5^{bcd}
NM	31.5 ±1.1^{a}	36.0 ±2.5^{bc}	35.8 ±1.7^{b}	38.4 ±1.5^{a}	39.9 ±1.4^{ab}
OBG500L	31.4 ±1.8^{a}	34.4 ±1.6^{bc}	33.2 ±0.8^{cd}	34.7 ±1.2^{c}	36.1 ±0.9^{cd}
OBG500M	32.1 ±1.3^{a}	35.2 ±2.7^{bc}	32.8 ±2.5^{d}	35.4 ±2.5^{bc}	36.7 ±3.3^{cd}
OBG500H	31.5 ±1.2^{a}	33.7 ±1.6^{c}	32.8 ±1.9^{d}	34.0 ±2.5^{c}	34.8 ±2.7^{d}
OBG40M	31.4 ±0.8^{a}	34.9 ±1.5^{bc}	34.4 ±1.5^{bcd}	35.6 ±1.6^{bc}	38.4 ±2.5^{abc}
OBG7M	31.6 ±1.4^{a}	36.5 ±2.8^{ab}	35.2 ±2.2^{bc}	37.2 ±2.1^{ab}	37.4 ±2.1^{bcd}

Data are expressed as mean ± standard deviation (n=7). Mean values in the same column followed by different superscript letters indicate significant difference ($P < 0.05$). Abbreviations: L, low-dose; M, mid-dose; H, high-dose; OBG500, OBG40, and OBG7 represent oat β-D-glucan with different MWs.

Table 3. Effects of oat β-D-glucan on fasting blood glucose in diabetic and normal mice

Groups	Week 0 (mmol/L)	Week 1 (mmol/L)	Week 2 (mmol/L)	Week 3 (mmol/L)	Week 4 (mmol/L)
NC	6.0±0.8^{b}	6.5±1.0^{c}	5.6±0.8^{d}	5.1±0.9^{e}	6.4±0.9^{c}
PM	11.5±2.4^{a}	14.2±2.4^{a}	9.02±2.4^{bc}	8.2±1.1^{d}	10.3 ±1.0^{bc}
NM	12.0±1.0^{a}	13.5±1.0^{ab}	16.6±2.0^{a}	15.9±1.2^{a}	17.1 ±1.9^{a}
OBG500L	11.1±1.0^{a}	14.1±2.5^{a}	12.2±2.0^{b}	13.1±2.1^{bc}	11.0 ±0.9^{b}
OBG500M	11.8±1.2^{a}	10.3±2.5^{abc}	10.6±1.9^{bc}	10.6±1.9^{d}	11.2 ±2.1^{b}
OBG500H	10.7±1.1^{a}	9.5±1.7^{bc}	8.4±0.4^{cd}	10.8±1.5^{cd}	9.8 ±0.8^{bc}
OBG40M	12.1±1.0^{a}	11.9±3.2^{ab}	11.1±2.4^{bc}	14.1±2.5^{ab}	13.1±1.9^{b}
OBG7M	11.2±0.9^{a}	12.6±4.2^{ab}	15.8±3.0^{a}	13.3±1.6^{abc}	11.2 ±1.9^{b}

Data are expressed as mean ± standard deviation (n=7). Mean values in the same column followed by different superscript letters indicate significant difference ($P < 0.05$).

Abbreviations: L, low-dose; M, mid-dose; H, high-dose; OBG500, OBG40, and OBG7 represent oat β-D-glucan with different MWs.