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Oat β-Glucan Increased ATPases Activity and Energy Charge in Small Intestine of Rats

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ABSTRACT: Dietary oat or oat products may potentially help to fight against high risk of cardiovascular diseases and β-glucan in oat was considered as a central player. The present study aimed to investigate the effects of dietary oat whole meal or β-glucan on insulin sensitivity and energy metabolism of rats. Rats were fed with control diet, oat whole meal based diet, or control diet with supplemented β-glucan for 4 weeks. Oat whole meal and β-glucan increased insulin sensitivity index. Interestingly, supplementation of oat whole meal or β-glucan induced increases in intestinal Na^+K^+-ATPase activity, $Ca^{2+}Mg^{2+}$-ATPase activity, and energy charge, particularly in the distal part of small intestine (ileum). Furthermore, amounts of *Bifidobacterium* and *Lactobacillus* in colon contents were elevated by oat whole meal or β-glucan. These findings provide an insight into that β-glucan increased insulin sensitivity and benefited intestinal health.

KEYWORDS: *Oat β-glucan, ATPase activity, energy charge, intestine, rat*

INTRODUCTION

Naked oat (*Avena nuda*) is a minor grain crop and a good source of protein, fat, minerals, B-complex vitamins, and especially heart-healthy soluble fiber β-glucan for both human beings and animals.[1] Because of the lubricative feature of oat in intestine, it was used as a traditional Chinese medicine hundreds of years ago. It has been proven that oat β-glucan can lower serum lipids in animal test and human clinical trails.[1−6] On January 23, 1997, the USA Food and Drug Administration (FDA) approved a health claim of "a diet high in soluble fiber from whole oats (oat bran, oat meal and oat flour) low in saturated fat and cholesterol may reduce the risk of heart disease" and oat β-glucan was considered to play the major role.[7,8] A previous study by our group demonstrated that oat products (oat bran, oat flour or rolled oat) significantly decreased fasting blood glucose (FBG) and showed hypoglycemic effects on streptozotocin-induced diabetic mice.[9] Researchers suggested that oat β-glucan's effect on lowering cholesterol, blood glucose and insulin was attributed to the increased viscosity of lumen content in intestine.[10−12]

High viscosity of upper gut content (small intestines) was related to prolonged gastric emptying and slower transit time when it passed through small intestine and increased intestinal motility of mice.[13−15] In addition, it has been suggested that the nonabsorbable materials in the small intestine binds water and traps cations, thus, interfering with solute-coupled water absorption and leading to a compensatory rise in ATPase levels and rats fed with high-fiber diet were reported to increase levels of mucosal Na^+K^+-ATPase in the ileum.[16,17]

Importantly, higher viscosity of lumen content, higher extent and frequency of contraction in small intestine may affect ATPase activity and energy charge (EC). However, to our best knowledge, limited information is available in this area. In the present study, we focused on the effects of oat whole meal and β-glucan on insulin sensitivity index, energy metabolism of liver, muscle and small intestine *in vivo*. Furthermore, the microbes from colon content were investigated. Our results indicated an important role for β-glucan in modulating insulin sensitivity index, intestinal peristalsis and probiotics in colon content.

MATERIALS AND METHODS

Chemicals. Nonesterified fatty acid (NEFA), Na^+K^+-ATPase activity and $Ca^{2+}Mg^{2+}$-ATPase activity analysis kits were obtained from Jiancheng Bioengineering Institute (Nanjing, China). Fasting blood insulin (FBI) detection kit was from Jiuding Bioengineering Institute (Tianjin, China). TPY agar, MRS agar and MacConkey agar were purchased from Beijing Land Bridge Technology (Beijing, China). ATP, ADP and AMP standards were obtained from Beijing Solarbio Science and Technology (Beijing, China).

Oat Whole Meal and β-Glucan Preparation. Naked oat (*A. nuda*) was kindly provided by Yanming Liu (Dingxi Dryland Agriculture Center, Gansu Province, China). After removing impurities, the oat kernels were deactivated by steaming for 20 min to kill enzymes and then dried at 38 °C for 24 h to keep the moisture content to 12%. After that, the oat groats were ground into powder with particle diameter <0.8 mm by Perten 3303 Model Mill (Perten Instrument Company, Sweden) and stored at 4 °C. Oat β-glucan was purified using water extraction and alcohol precipitation method as described previously.[18]

Animals. This study was approved by the Animal Ethics Committee of Northwest A&F University. Thirty male SD rats initially weighing 160 ± 10 g (The Fourth Military Medical University, Xi'an, China), were maintained under controlled temperature (23 ± 1 °C), humidity (55%) and air flow conditions with a fixed 12-h light−dark cycle (light 7:00 a.m. to 7:00 p.m.). During 1 week acclimation, the rats were fed with basal diet.

The rats were then randomly divided into 3 groups (n = 10) after the acclimation. The rats in control group and β-glucan group were fed

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with normal laboratory feed, rats in β-glucan group were also administrated with β-glucan (312.5 mg/kg body weight, according to the dosage of FDA) dissolved in 2 mL of distilled water.[7] For the oat whole meal group, 10% oat whole meal was added to the normal laboratory food (β-glucan content: 0.33% of the diet).[7] Diet components are described in detail in Table 1. In addition, the control and oat whole meal groups were administrated with 2 mL of physiological saline. Each treatment lasted for four weeks. All rats had free access to food and water. The feces were collected during the final three days.

Table 1. Animal Diets Composition[a]

diet[a]	control	oat whole meal
oat whole meal (%)	0	10
corn starch (%)	56	46
rice bran (%)	10	10
fish meal (%)	26	26
soybean oil (%)	2	2
bone powder (%)	3	3
yeast powder (%)	2.3	2.3
salt (%)	0.5	0.5
vitamin-mineral mixture (%)	0.2	0.2
total (%)	100	100
energy (kcal/100 g)	538	549

[a]Diet was made according to commercial available rat normal columnar diet. β-Glucan group were fed with normal laboratory feed and rats in β-glucan group were also administrated with β-glucan (312.5 mg/kg body weight) dissolved in 2 mL of distilled water.

Rats were starved for 12 h before being killed at the end of experimental treatment. The rats in each group were anesthetized with 10% chloral hydrate (3.5 mL/kg body weight, intraperitoneal injection). Blood from the femoral artery was collected. Jejunum, ileum and colon samples were washed in phosphate-buffered saline (PBS) before storing at −80 °C. Liver and skeletal muscle samples were stored at −80 °C directly. Samples for ATPase and EC determination were frozen in liquid nitrogen and stored at −80 °C before analysis. Samples for microbial determination were taken from the colon and stored at 0 °C and prepared for analysis within 24 h.

Plasma Biochemical Markers. Blood samples from femoral artery of rats were kept at 4 °C for 90 min. The serum was collected for further analysis by centrifugation at 200*g* for 10 min. The FBI and NEFA were assayed by using commercial kit according to the protocol respectively. FBG was measured with Lifescan One Touch test paper on a Surestep glucose kit (Johnson & Johnson Company). The insulin sensitivity index (ISI) was calculated according to the following formula:[19]

$$\mathrm{ISI} = \ln \frac{1}{\mathrm{FBG\ (mmol/L)} \times \mathrm{FBI\ (mU/L)}}$$

ATPase and EC Measurement. Tissues (liver, muscle, jejunum or ileum), approximately 0.2 g, added with 9 times (volume/tissue weight) ice-cold saline solution were homogenized and centrifuged at 1000*g*, 4 °C for 5 min. The supernatant was used for analysis according to commercial kit protocol, liberated inorganic phosphate was measured. The Na^+K^+-ATPase and $Ca^{2+}Mg^{2+}$-ATPase activity were expressed as micromoles of Pi released per milligram of protein per hour. Protein was examined by Bradford assay. For energy charge determination, frozen liver, muscle, jejunum and ileum of rats were homogenized into powder. Adenosine phosphates were extracted with 2 mL of 0.4 mol/L perchloric acid for 2 min in an ice bath. The extraction mixture was centrifuged at 4 °C for 10 min at 3500*g*. One milliliter of supernatant was collected and for every aliquot 1 mL of 1 mol/L dipotassium hydrogen phosphate was added. The solution was quickly neutralized (pH 6.5) with 1 mol/L KOH. The mixture was centrifuged at 4 °C for 10 min at 3500*g*. The supernatant was collected and filtered through a 0.22 μm filter and used for ATP, ADP and AMP measurements by HPLC (Waters) as described by the previous report.[20] EC = (ATP + 1/2ADP)/(ATP + ADP + AMP).

Microflora Analysis. The microbial counts were determined as described by Sembries et al.[21] Briefly, approximately 0.2 g of fresh collected content of colon was immediately placed into preweighed tubes and diluted with sterile distilled water. In duplicates, 0.1 mL of each dilution was plated on selective media. MRS agar and MacConkey agar were incubated aerobically at 37 °C for the determination of *Lactobacilli* and *Enterococcus*, respectively (for 48 h). Numbers of *Bifidobacterium* were determined after a 48 h anaerobic incubation of TPY agar at 37 °C.

Statistical Analysis. All results were subjected to ANOVA and presented as mean values ± SE. Differences in mean values between groups were tested using Duncan's multiple range test and were considered significant when $P < 0.05$.

RESULTS

Food Intake, Body Weight, Feces, and Plasma Biochemistry. Food intake of last three days for control group and oat whole meal group was 24.57 and 26.44 g per day for every rat, respectively; rats in β-glucan group had much lower (17.74 g per day for every rat) food intake. β-Glucan fed rats had lower ($P < 0.05$) (Table 2) final body weight and body weight gain than those of control group and oat whole meal group. In contrast, rats of oat whole meal based group had larger ($P < 0.05$) body weight gain than those in control group. In addition, rats of oat whole meal group had higher ($P < 0.05$) wet feces and higher ($P < 0.05$) moisture content of feces while feces of control group and β-glucan group were very similar (Table 2).

Table 2. Body Weight and Feces[a]

group	control	oat whole meal	β-glucan
Body Weight (g)			
initial body weight	157.58 ± 6.61	157.31 ± 4.05	157.49 ± 3.02
final body weight	308.33 ± 9.12	333.44 ± 6.02	276.89 ± 11.98[b]
body weight gain	150.75 ± 6.56	176.13 ± 4.89[b]	119.40 ± 10.87[b]
Feces (g/rat/day), Moisture Content (%), Last Three Days			
wet feces	7.10 ± 0.30	9.15 ± 0.94[b]	5.43 ± 0.22[c]
dry feces	4.32 ± 0.24	5.22 ± 0.52	3.30 ± 0.10[c]
moisture content	34.77 ± 1.01	40.54 ± 0.91[b]	34.12 ± 1.93[c]

[a]Values are presented as means ± SE ($n = 10$). [b]$P < 0.05$ compared with those of control group. [c]$P < 0.05$ compared with those of oat whole meal group in Duncan's multiple range test.

FBG in rats of oat whole meal or β-glucan group was lower than that of control group, but not significant ($P > 0.05$) (Table 3). The FBI level in rats of β-glucan group was lower ($P < 0.05$) than that of the control group, but no significant differences in FBI level was observed between oat whole meal group and control group. The ISI levels in β-glucan group and oat whole meal group were higher ($P < 0.05$) than those in the control group (Table 3), but differences in ISI between the oat whole group and β-glucan group were not significant ($P > 0.05$). Rats in β-glucan group had higher ($P < 0.05$) concentration of serum NEFA when compared with the control group. Taken together, these data indicate a critical role of β-glucan in preventing body weight gain and elevating ISI.

ATPase Activity and EC in Liver, Muscle, and Small Intestine of Rats. Both oat whole meal and β-glucan groups showed slightly lower Na^+K^+-ATPase activity in liver and skeletal muscle than the control group (Figure 1A). Small declines were observed in $Ca^{2+}Mg^{2+}$-ATPase activity in liver

Table 3. Effects of Oat Whole Meal and β-Glucan on FBG, FBI, ISI, and NEFA of Rats[a]

group	control	oat whole meal	β-glucan
FBG (mmol/L)	5.38 ± 0.16	5.13 ± 0.18	5.03 ± 0.14
FBI (mIU/L)	20.77 ± 1.46	17.94 ± 0.62	14.73 ± 0.89[b,c]
ISI	−4.70 ± 0.06	−4.52 ± 0.04[b]	−4.30 ± 0.06[b]
NEFA (mmol/L)	0.523 ± 0.018	0.772 ± 0.268	1.489 ± 0.351[b,c]

[a]Values are presented as means ± SE (n = 8). [b]P < 0.05 compared with those of control group. [c]P < 0.05 compared with those of oat whole meal group in Duncan's multiple range test. FBG: Fasting blood glucose; FBI: Fasting blood insulin; ISI: Insulin sensitivity index; NEFA: Nonesterified fatty acids.

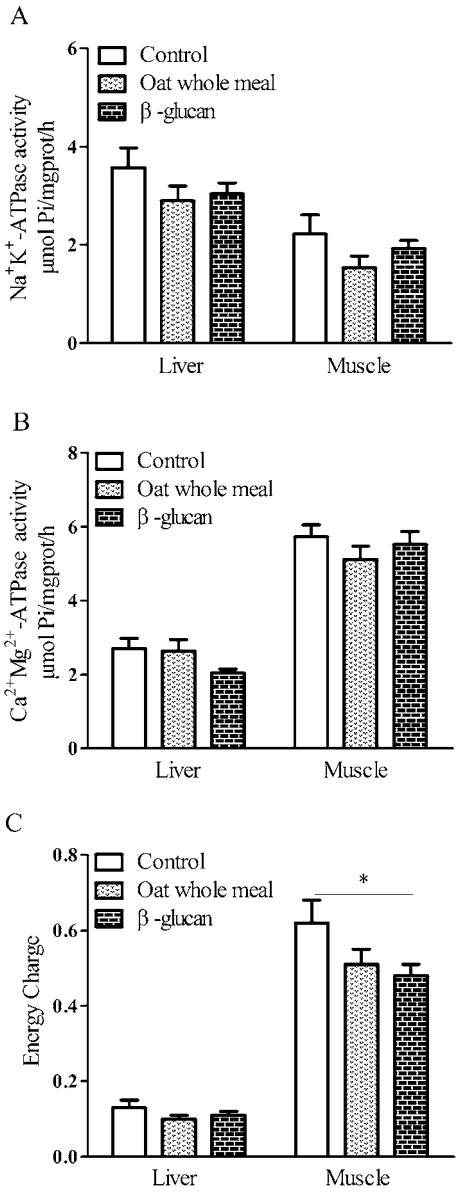

Figure 1. Effect of oat whole meal or β-glucan on: (A) Na^+K^+-ATPase activity in liver and muscle (n = 10); (B) $Ca^{2+}Mg^{2+}$-ATPase activity in liver and muscle (n = 10); (C) EC in liver and muscle (n = 5). The Na^+K^+-ATPase and $Ca^{2+}Mg^{2+}$-ATPase activities were expressed as micromoles of Pi released per milligram of protein per hour. Values are presented as means ± SE *P < 0.05 in Duncan's multiple range test.

and muscle of rats fed with oat whole meal or β-glucan (Figure 1B). The EC in muscle of β-glucan group was decreased significantly when compared with control group (P < 0.05) (Figure 1C) and slight changes were detected in liver of oat whole meal and β-glucan group.

To understand the energy metabolism in the small intestine, ATPase activity and EC were measured after four weeks. Na^+K^+-ATPase activity in jejunum increased gradually from control group to β-glucan group and Na^+K^+-ATPase activity in jejunum of β-glucan group increased significantly (P < 0.05) when compared with control group (Figure 2A). Although

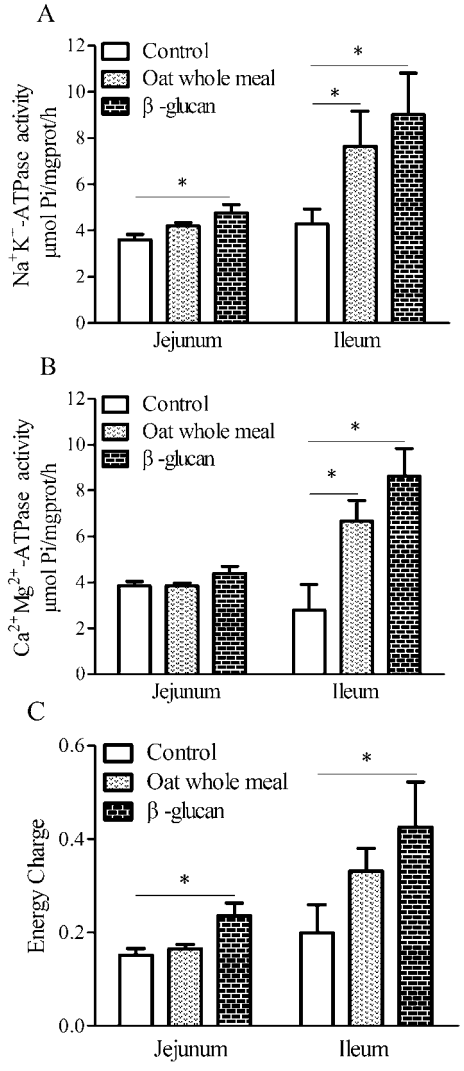

Figure 2. Effect of oat whole meal or β-glucan on: (A) Na^+K^+-ATPase activity in jejunum and ileum (n = 10); (B) $Ca^{2+}Mg^{2+}$-ATPase activity in jejunum and ileum (n = 10); (C) EC in jejunum and ileum (n = 5). The Na^+K^+-ATPase and $Ca^{2+}Mg^{2+}$-ATPase activities were expressed as micromoles of Pi released per milligram of protein per hour. Values are presented as means ± SE *P < 0.05 in Duncan's multiple range test.

there was an increase in $Ca^{2+}Mg^{2+}$-ATPase activity of jejunum in β-glucan group, changes were not significant (Figure 2B). Na^+K^+-ATPase activity and $Ca^{2+}Mg^{2+}$-ATPase activity in ileum were stimulated significantly (P < 0.05) in both oat whole meal and β-glucan groups when compared with that in control group.

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β-Glucan had positive effect on the induction of EC in jejunum and ileum. EC in both jejunum and ileum of β-glucan group increased significantly ($P < 0.05$) when compared with control group (Figure 2C). In ileum, EC of oat whole meal group was much higher ($P < 0.05$) than that in control group and no significant changes happened in jejunum between oat whole group and control group ($P > 0.05$).

Microbes in Colon. Because water-soluble β-glucan can be fermented by microflora in large intestine, we next explored the changes of microflora in colon content. *Bifidobacterium*, *Lactobacillus*, and *Enterococcus* were investigated (Figure 3).

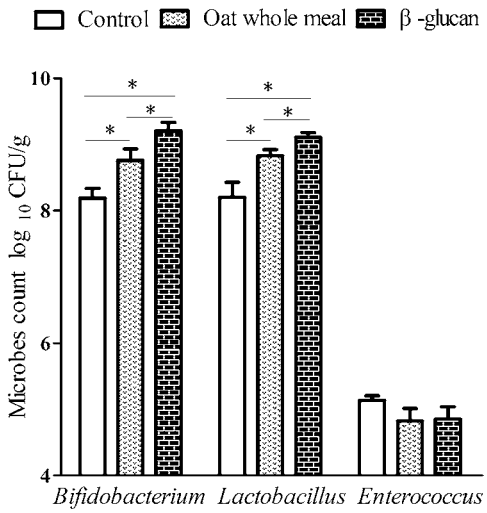

Figure 3. Microbes count in colon content of rats fed with different diets ($\log_{10}$ CFU/g). Values are presented as means ± SE ($n = 5$). *$P < 0.05$ in Duncan's multiple range test.

There were significant increases in *Bifidobacterium* ($P < 0.05$) and *Lactobacillus* ($P < 0.05$) amount after rat consumed oat whole meal. Rats administrated with β-glucan had higher population of *Bifidobacterium* ($P < 0.05$) and *Lactobacillus* ($P < 0.05$) compared with rats fed the control or oat whole meal-based diet. However, numbers of *Enterococcus* were not significantly different between all groups.

DISCUSSION

In the present study, rats fed with β-glucan had lower body weight gain compared with rats in oat whole meal group or control group. It is well recognized that β-glucan provided less energy as a substitute for nutrients in diets.[22] In addition, the reduced body weight in β-glucan group may be attributed to retarded transport of digestive enzymes to the substrates and increased thickness of unstirred layer on the absorbing surface.[23] The molecular weight of β-glucan plays an important role in reducing body weight, rats consumed high-fat diet containing β-glucan with different molecular weights had significantly lower body weight than rats fed with high-fat diet, and higher molecular weight of β-glucan brought body weight to a much lower level and even lower than those of normal diet fed rats.[24] But oat fiber or oat bran increased animals' body weight in some instances.[25,26] In the present study, rats fed with oat whole meal had larger final body weight and higher body weight gain. The body weight increasing effect of oat fiber, oat bran or oat whole meal in animal experiments may be attributed to protein, fat, and vitamin complex in them. Protein content in oat whole meal based diet is 1.5 g higher than control for 100 g diet.[1]

As previously documented, oat bran or β-glucan have generally been reported to decrease glucose and insulin responses in normoglycemic and diabetic subjects.[10,27−29] First suggested mechanisms for these results are that oat β-glucan can increase viscosity of intestinal content, slow digestion of carbohydrate, thereby reducing postprandial hyperglycemia and insulin secretion.[30,31] Second, β-glucan may also delay gastric emptying rate due to high viscosity of the fiber, concomitantly causing an extended sensation of fullness. Third, β-glucan could be fermented up to 100% by microflora in large bowel and was not found in feces, leading to release of short-chain fatty acids, lowering postprandial glucose levels.[10,28−32] There are some insoluble dietary fibers in oat whole meal which cannot be easily fermented by microflora when they pass through small intestine and large bowel. These insoluble fibers have high water holding capacity and contribute to increased stool amount and higher moisture content.[1,32] Furthermore, oat products increased the amount of probiotics, while coliforms were decreased.[34] Our study demonstrated that *Bifidobacterium* and *Lactobacillus* population increased significantly in rats which consumed oat whole meal or β-glucan when compared with control group. There was a slight decrease of FBG, while ISI was increased significantly in both oat whole meal group and β-glucan group. Moisture content showed almost the same level in control group and β-glucan group, while higher level was observed in oat whole meal group.

Supplemented β-glucan caused decreased EC and slight decrease in Na^+K^+-ATPase activity in skeletal muscle of rats. This could be explained, at least in part, by lower blood insulin level resulting in declined Na^+K^+-ATPase activity in skeletal muscle, while insulin was known to activate the Na^+K^+-ATPase by dephosphorylation.[33,34]

Intake of β-glucan or a diet containing oat whole meal was associated with elevated levels of Na^+K^+-ATPase, $Ca^{2+}Mg^{2+}$-ATPase and EC in ileum of rats. There are two hypotheses for β-glucan enhancing the ATPase activity and contractional movement in intestine. (1) High viscosity of the intestinal contents. Animal studies indicated a significant depolymerisation of oat β-glucan during digestion in the small intestine of rats and chicks.[35,36] Migration of microflora from large to small intestine caused by high fiber diets may contribute to fermentation, but physical barriers and residence time may cause fermentation in the small intestine to be limited and viscosity to remain elevated at the distal end of the intestine.[37] As the mammalian enzymes were unable to hydrolyze β-glucan, intact β-glucan was observed in the small intestine of healthy man,thus increasing the viscosity throughout the small intestine. The maximum β-glucan concentration was found in the ileum, where most of the starch has been absorbed.[35,36] Viscosity of oat β-glucan is concentration dependent and affected by pH. The high level of oat β-glucan and neutral pH in ileum may cause high viscosity or gel-formation, followed by increasing contractional movements, while contractional movements in control group were limited.[38,39] (2) The elevation of Na^+K^+-ATPase, $Ca^{2+}Mg^{2+}$-ATPase levels in ileum might be an effect similar to that found in rats fed the nonabsorbable material, polyethylene glyco1,[17] where intestine levels of ATPase are elevated. It has been suggested that the nonabsorbable material binds water and traps cations (chiefly sodium), thus interfering with solute-coupled water absorption and leading to a compensatory rise in ATPase levels.[16]

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Moreover, rats fed with the high-fiber diet had increased levels of mucosal Na^+K^+-ATPase in the ileum.[16] The oat β-glucan might also act by trapping water and cations with a similar result when it passes through small intestine.

In conclusion, oat whole meal or β-glucan could increase ISI and elevate the population of *Bifidobacterium* and *Lactobacillus* in colon of rats. Oat β-glucan most likely activated Na^+K^+-ATPase activity, $Ca^{2+}Mg^{2+}$-ATPase activity, and EC in distal small intestine of rats.

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Notes

The authors declare no competing financial interest.

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ABBREVIATIONS USED

FDA, food and drug administration; EC, energy charge; FBG, fasting blood glucose; FBI, fasting blood insulin; ISI, insulin sensitivity index; NEFA, nonesterified fatty acid; PBS, phosphate-buffered saline.

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