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Effect of resistant starch from corn or rice on glucose control, colonic events, and blood lipid concentrations in streptozotocin-induced diabetic rats

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Abstract

To examine the effect of two types of resistant starch on blood glucose and insulin levels, colonic events, hypolipidemic actions and humoral immune responses, Sprague-Dawley streptozotocin-induced diabetic rats were fed diet containing resistant starch from corn or rice. The marked body weight loss by inducing diabetes was not recovered by feeding resistant starch, even though there are no differences in food intakes compared to the non-diabetic control rats. No significant effect of resistant starch feeding on blood glucose and insulin was found. Even though the length of small intestines, and cecum, colon and rectum together with the tissue weight of cecum were not affected by feeding resistant starch, the intestinal transit time was markedly shortened by both types of resistant starch and resistant starch from corn had a more pronounced effect. The short chain fatty acids in the intestinal contents did not appear to be different among the groups. Nonetheless, both of resistant starch from corn and rice significantly lowered plasma total lipid and cholesterol concentrations compared to the diabetic control. The total liver cholesterol lowering effect was observed with resistant starch from rice. Neither immunoglobulin G nor C_3 were influenced by resistant starch.

Keywords: Resistant starch; Transit time; Lipid metabolism; Diabetic rats

1. Introduction

Resistant starch (RS) is the sum of starch and starch-degradation products not absorbed in the small intestine, because they are resistant to enzyme digestion [1]. RS appeared to have various physiological effects such as the reduction of plasma cholesterol, increase in cecal and large intestinal contents, alteration in microbial populations, and increase in large intestinal short-chain fatty acid production [2]. These effects may lead to the decreased incidence of cecal cancer, atherosclerosis, obesity-related complications in human [3].

In non-insulin-dependent diabetes mellitus, a diet high in simple carbohydrate or digestible carbohydrate might aggravate glycemic control or accelerate the development of cardiovascular diseases [4]. Different carbohydrates can alter glycemic responses in relation to the nature of carbohydrate, food processing, availability to α-amylase, gastric emptying time, gut hormone profiles, and by stimulating colonic fermentation to the production of short-chain fatty acids [5–7]. The relationship between RS and glycemic control has not been elucidated in diabetic human or animal models, even though RS would be a candidate for the ideal glycemic control in diabetic conditions.

Cholesterol lowering through the consumption of non-digestible resistant starch draws much attention lately [8–13]. In rats, it has been shown that RS replaced for digestible starch in the diet can lower serum cholesterol concentrations in normal or hypercholesterolemic rats [14–17]. It has been suggested that retrograded starch may lower the serum cholesterol concentration by several mechanisms,

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including an increase in fecal bile acid excretion [18]. However, not every type of RS can bind bile acids. RS in the form of resistant starch granules (in case of raw potato starch) does not seem to bind to bile acids [17]. Besides, the systemic effect of propionic acid on cholesterol synthesis can be considered as one of the mechanisms of cholesterol lowering effect of RS. It has been proposed that soluble plant fiber lowers plasma cholesterol concentrations by inhibition of hepatic cholesterol synthesis via propionate formed through large-bowl fermentation [19]. And short-chain fatty acid has been shown to suppress cholesterol synthesis in liver and intestine of rats [20]. However, previously it has been argued that *in vivo* production of propionate by diet alone is not high enough to decrease the activity of hydroxy-methylglutaryl-CoA reductase [21]. Even though the exact mechanism of cholesterol-lowering effect of RS is unclear at present, RS appears to be an attractive source of carbohydrate reducing atherogenic potentials without altering its organoleptic properties [16,22].

In the present study, two sources of resistant starch were selected to compare the physiological functions in diabetic rats. Corn is a popular grain and used for starch production due to its stable supply and low cost, and rice is the staple food in many Asian cultures. Different cereal starches have different properties and structures as well as a range of physical and chemical properties and therefore, resistant starch from corn or rice may show the different effects. Resistant starch from corn has been shown to reduce serum cholesterol in rats [23]. In the study of Cheng and Lai of rats under hypercholesterolemic diets, 63% resistant starch from rice has been shown to have a more pronounced effect on cholesterol lowering effect than resistant starch from corn [24].

In diabetic rats, the cholesterol lowering effect of rice was not compared with corn. The principle purpose of the present study was to investigate the effect of resistant corn or rice starch on glycemic control, colonic events, blood lipid concentrations and humoral immune responses in streptozotocin-induced diabetic rats.

2. Materials and methods

2.1. Chemicals

Casein was obtained from Murray Goulbur Co., Australia. Soybean oil was from Cheil Co, Seoul, Korea. Corn starch, rice starch, *t*-butylhydroquinone, cholesterol, cellulose and streptozotocin were purchased from Sigma Chemical Company (St. Louis. MO, USA). Other chemicals were of reagent grade.

2.2. Preparation of resistant starch

Corn or rice starch (amylose contents; 27% by manufacturer, Sigma Co. USA) were dissolved in 4 × volume of boiling water, boiled for 10 min while stirring, removed from heat and simmered for 5 min while stirring. To prepare RS, the suspension was cooled down to 30°C, vacuum-sealed in a retort pouch and autoclaved at 121°C under 15 psi for 1 hr, and then stored at 4°C for 24 hr. After four cycles of autoclaving and cooling, to remove remaining starch not retrograded, the preparation was then dissolved in 10 times volume of water and allowed to stand for 24 hr, and hydrolyzed with 15 g/kg α-amylase (Total unit; 8,000U, from *Bacillus globigii*, Junsei Co. Japan) at 90°C. The sediments were then washed and hot-air dried at 60°C. This enzymatic hydrolytic process was undergone to remove the unchanged starch. The obtained RS of each type was meshed and used in the animal diet. The RS containing diets (30% RS w/w) were prepared on the basis of a modified AIN-93G [25] (Table 1). The estimation of RS contents by the method of Total Dietary Fiber Determination Kit (TDF-100A, Sigma Co., USA) showed that control diet has 25.73 g/kg diet and RS diets contained 161.25 g/kg diet (Table 1).

Table 1
Composition of experimental diets

Ingredient	CONTROL	DIABETES	RS-CORN	RS-RICE
				(unit: g/kg)
Corn starch[1]	529.486	529.486	229.486	229.486
Resistant starch(RS)	—	—		
from corn			300.0	
from rice				300.0
Casein	200.0	200.0	200.0	200.0
Sucrose	100.0	100.0	100.0	100.0
Soybean oil	70.0	70.0	70.0	70.0
Fiber(cellulose)	50.0	50.0	50.0	50.0
Mineral mix[2]	35.0	35.0	35.0	35.0
Vitamin mix[3]	10.0	10.0	10.0	10.0
L-Cystine	3.0	3.0	3.0	3.0
Choline	2.5	2.5	2.5	2.5
t-Butylhydroquinone	0.014	0.014	0.014	0.014
Resistant starch as analyzed	25.73	25.73	161.15	161.15

[1] Commercial corn starch was dissolved in 4 × volume of boiling water, boiled for 10 min while stirring, simmered for 5 min, dried and then meshed.

[2] Mineral mixture (per kg): Calcium carbonate, 357g; Monopotassium phosphate, 196g; Potassium citrate, 70.78g; Sodium chloride, 74g: Magnesium oxide, 24g; Ferric citrate, 6.06g; Zinc carbonate, 1.65g; Manganous carbonate, 0.63g; Cupric carbonate, 0.30g; Potassium iodate, 0.01g; Ammonium paramolybdate, 0.00785g; Powdered sucrose, 269.56215g.

[3] Vitamin mixture (per kg): Nicotinic acid, 3.0g; Ca Pantothenate, 1.6g; Pyridoxine HCl 0.7g; Thiamin HCl, 0.6g; Riboflavin 0.6g; Folic acid, 0.2g; D-Biotin, 0.02g; Vitamin B_{12}, 2.5g; Vitamin E, 15.0g; Vitamin A, 0.8g; Vitamin D_3, 0.25g; Vitamin K (phylloquinone), 0.075g; Powdered sucrose, 974.655g.

2.3. Rats and feeding regiments

Four-week old male Sprague-Dawley rats were maintained on a conventional diet for a period of one week and then divided into 4 groups of eight per group by randomized block design according to the weight. Diabetes was induced

by injecting 50 mg/kg streptozotocin in 0.1 M citrate buffer into intraperitoneal cavities and control rats received only 0.1 M citrate buffer. Blood glucose concentrations of rats were analyzed from tail vein blood at 10 AM and rats with over 16.7 mmoles/L glucose were selected (n=8, each). Rats were maintained individually in cages with wire-mesh bottoms in conditioned rooms (23°C; relative humidity 55%). All rats were provided with food and water *ad libitum* and maintained on each diet for a 3-week period.

2.4. Plasma collection and various organ preparations

Food was removed for 12 hr at the end of the experimental term, and the rats were anesthetized with ethyl ether and dissected. Blood was collected from the heart with heparinzed syringe. The plasma from the blood was collected by centrifugation at 4000 × g for 30 min after settling the blood for 30 min at 4°C. The plasma was then stored at –70°C. The liver, kidneys, thymus, spleen and epididymal fat pads were removed and the wet weights were measured. The length of small intestine and the combined length of cecum, colon and rectum were measured. Cecum were removed from the large intestines and washed with saline, blotted and weighed with or without contents. Livers were removed and stored at −70°C for lipid analysis.

2.5. Plasma glucose and insulin measurement

Plasma glucose was measured using a commercial kit (Asan Pharmaceutical Co., Seoul, Korea). Immunoreactive insulin was determined by radioimmunoassay with RIA kit of Linco Research Inc.(St. Charles, USA).

2.6. Intestinal transit time measurement

The intestinal transit time measurement was carried out after 2 weeks of feeding experimental diets. The rats were fasted for 15 hr, and then fed 10 g of Indigo Carmine containing diet (0.5% in equal mixture of diet and sugar) for 4 hr and then regular diet was supplied. The appearance of dye was checked every 20 min. The interval between the beginning of diet consumption and the first appearance of colored feces was determined as intestinal transit time.

2.7. Short chain fatty acid (SCFA) measurement

Measurement of SCFAs were carried out with gas chromatography (3400CX, Varian., USA). The column was a silica capillary column DB-FFAP (30m × 0.25 μm film, 0.25 mmID, J&W Science, USA). Samples from the fresh cecum contents were stored at −70°C. Immediately before analysis the samples were thawed and added 10% phosphoric acid. This was then vortexed before centrifuging at 10,000 × g at 4°C for 30 min. Finally, 1 μl extracted supernatant was injected into gas chromatography column [26].

2.8. Plasma lipid and immunoglobulin measurement

Plasma total lipids were determined by a modified method of Frings [27]. Plasma total cholesterol and triacylglycerol were measured enzymatically using a commercial kit (Asan Pharmaceutical Co., Seoul, Korea). HDL-cholesterol was measured enzymatically after precipitation of apolipoprotein B containing lipoproteins with dextran sulfate according to the method of Sjoblom and Eklund [28].

Plasma Immunoglobulin G(IgG) and C_3 were measured according to the method of rate nephelometry [29]. IgG was measured as a representative of immunoglobulins and C_3 was determined since it has been shown in humans and in experimental animals, reduced C_3 levels were observed in relation to lowered host defense against infection [30].

2.9. Liver lipid measurement

Total lipids were extracted with chloroform:methanol(2:1,v/v) by a modified method of Folch *et al* [31]. After extraction, total lipid, triacylglycerols and cholesterol were measured utilizing the methods for plasma lipid determination.

2.10. Statistical analysis

Statistical analysis was basically performed using the Statistical Analysis System (SAS). Data were expressed as the mean with standard error, and statistically significant differences between subgroup means were evaluated by two-way ANOVA and Duncan's multiple range test.

3. Results

3.1. Body weight gain and food consumption

Even though there were no differences in food intake, body weight was greatly influenced by diabetic states (Table 2). Resistant starch, regardless of corn or rice did not affect this weight loss.

3.2. Organs and epididymal fat pads weights

Epididymal fat pads, thymus and spleen weights were significantly decreased in diabetic control group, resistant starch from corn group and resistant starch from rice group possibly due to the decrease in body weight (Table 3). However, kidney and liver weights were not significantly different among the groups and not affected by feeding resistant starch.

3.3. Blood glucose and insulin levels

Blood glucose levels were increased greatly in diabetic rats (Table 4). Resistant starch from rice group showed

Table 2
Effect of resistant starch from corn(RS-CORN) and resistant starch from rice(RS-RICE) on body weight gain, food intakes and food efficiency ratio (F.E.R.) in streptozotocin-induced diabetic rats fed experimental diets for three weeks

Group	Initial body Weight(g)	Final body weight(g)	Body weight gain(g/period)	Food Intakes (g/period)	F.E.R.
CONTROL	250.6 ± 12.9^{NS}	335.9 ± 52.1^{a}	72.1 ± 42.1^{a}	658.9 ± 42.8^{NS}	0.11 ± 0.06^{a}
DIABETES	251.6 ± 9.4	213.6 ± 40.0^{b}	−26.7 ± 29.2^{b}	617.0 ± 67.6	−0.05 ± 0.05^{b}
RS-CORN	250.9 ± 7.0	213.4 ± 40.1^{b}	−23.2 ± 52.5^{b}	647.2 ± 64.1	−0.01 ± 0.08^{b}
RS-RICE	247.6 ± 8.1	212.5 ± 25.9^{b}	−24.2 ± 16.9^{b}	711.0 ± 67.8	−0.04 ± 0.03^{b}

F.E.R. = g of body weight gains during experimental period/g of food intakes during experimental period.
Data are expressed as means ± standard error.
Values in a column with no common superscript letters are significantly different ($P < 0.05$).
NS means values in a column are not significantly different.

decreasing tendency in blood glucose concentrations, but the mean values were not statistically different from either control or resistant starch from corn group. There were no significant influences of diabetic condition or feeding resistant starch on blood insulin concentrations.

3.4. Length of small and cecum, colon and rectum, cecum weights and intestinal transit time

Neither diabetic condition nor resistant starch affected the length of small and cecum, colon and rectum, cecum weights (Table 5). The diet supplemented with resistant starch from corn decreased intestinal transit time greatly than resistant starch from rice.

3.5. The concentrations of short chain fatty acid from the intestinal contents

The short chain fatty acid contents were not different among the groups (Table 6).

3.6. Plasma lipid concentrations

In diabetic control group, the total lipid contents were significantly increased, and both type of resistant starch decreased the elevated total lipid in blood (Table 7). However, there was neither significant influence of diabetic condition nor resistant starch on triacylglycerols concentrations. Total cholesterol concentrations were significantly lower in rats fed resistant starch from corn or resistant starch from rice compared to the diabetic control group. In diabetic control rats, both total cholesterol and HDL-cholesterol increased greatly. The ratio of HDL-cholesterol to total cholesterol were not significantly different among the groups, indicating a raise in HDL-cholesterol in diabetic control group is not very meaningful because both HDL and total cholesterol were increased.

3.7. Liver lipid concentrations

Table 8 shows the results of the effect of feeding RS on liver total lipid, triacylglycerols and total cholesterol concentrations in diabetic rats in comparison with non-diabetic control rats. Even though hepatic total lipid contents did not differ from diabetic controls, the reduction of total lipid contents from the nondiabetic controls was observed. Hepatic triacylglycerols were decreased in diabetic rats and resistant starch did not decrease triacylglycerol levels below the level of diabetic control rats. Resistant starch from rice decreased liver cholesterol when it was compared to the control rats. However, there were no significant differences in all of the three diabetic groups. The total lipid accumulating effect by diabetic conditions was observed in this work, however, resistant starch did not abolish this effect.

Table 3
Effect of resistant starch from corn(RS-CORN) and resistant starch from rice(RS-RICE) on organ weights in streptozotocin-induced diabetic rats fed experimental diets for three weeks

Group	Kidney	Liver	Epididymal fat pads	(unit:g of wet weights) Thymus	Spleen
CONTROL	2.72 ± 0.19^{NS}	9.86 ± 0.90^{NS}	3.41 ± 1.47^{a}	0.32 ± 0.17^{a}	0.72 ± 0.18^{a}
DIABETES	2.87 ± 0.30	9.22 ± 1.16	1.09 ± 0.84^{b}	0.11 ± 0.06^{b}	0.42 ± 0.14^{b}
RS-CORN	2.75 ± 0.38	8.73 ± 1.04	0.97 ± 0.60^{b}	0.16 ± 0.11^{b}	0.39 ± 0.17^{b}
RS-RICE	2.90 ± 0.27	9.18 ± 1.26	1.15 ± 0.76^{b}	0.12 ± 0.06^{b}	0.40 ± 0.14^{b}

Data are expressed as means ± standard error.
Values in a column with no common superscript letters are significantly different ($P < 0.05$).
NS means values in a column are not significantly different.

Table 4
Effect of resistant starch from corn(RS-CORN) and resistant starch from rice(RS-RICE) on plasma glucose and insulin levels in streptozotocin-induced diabetic rats fed experimental diets for three weeks

Group	Plasma glucose (mmoles/L)	Insulin (pmoles/L)
CONTROL	7.8 ± 1.4^{b}	442.6 ± 333.0^{NS}
DIABETES	15.9 ± 6.9^{a}	338.9 ± 109.8
RS-CORN	15.6 ± 8.1^{a}	419.9 ± 515.2
RS-RICE	12.9 ± 3.2^{ab}	579.7 ± 305.4

Data are expressed as means ± standard error.
Values in a column with no common superscript letters are significantly different ($P < 0.05$).
NS means values in a column are not significantly different.

Table 6
Effect of resistant starch from corn(RS-CORN) and resistant starch from rice(RS-RICE) on short chain fatty acids in cecum contents in streptozotocin-induced diabetic rats fed experimental diets for three weeks

			(unit:μmoles/g cecum contents)	
Group	Acetic acid	Propionic acid	Butyric acid	Total short chain fatty acids
CONTROL	13.3 ± 2.4^{NS}	5.2 ± 0.8^{NS}	3.9 ± 0.8^{NS}	22.5 ± 2.9^{NS}
DIABETES	16.2 ± 6.0	5.4 ± 1.5	3.6 ± 1.6	25.1 ± 7.4
RS-CORN	14.8 ± 3.5	5.1 ± 1.1	3.2 ± 0.6	23.1 ± 4.6
RS-RICE	12.8 ± 4.8	4.5 ± 1.1	3.0 ± 0.3	21.1 ± 4.5

Data are expressed as means ± standard error.
NS means values in a column are not significantly different.

3.8. Plasma immunoglobulin G and C_3 concentrations

No significant interaction between diabetes and resistant starch supplementation was found in plasma IgG and C_3 concentrations (Table 9).

4. Discussion

The purpose of present study was to examine the effects of the consumption of resistant from corn or rice in streptozotocin induced diabetic rats on various parameters related to glycemic control, hypercholesterolemia, and colonic events. Part of the starch, after heating and subsequent cooling, can be converted into an indigestible fraction called resistant starch. The hydrothermic treatment followed by keeping disorganized starch macromolecules at low temperature (4°C) can be repeated to increase RS transformation. This evolution of starch is defined as highly resistant starch in recrystallized forms of amylose and amylopectin [32]. RS has been proposed as a new category of dietary fiber possessed with some metabolic functions against abnormality caused by over supply of energy or fat [33]. One of the significant effects of resistant corn or rice starch is the lowering of serum cholesterol concentrations as shown in the present study. Dietary high-amylose cornstarch compared to low-amylose starch decreased blood cholesterol concentrations by 30-36% [34]. The range of cholesterol lowering was from 8% to 23% in subsequent studies [14–17]. Triacylglycerol concentration changes were more variable than cholesterol with the range of 0-42% [14–16]. The reason why there is no triacylglycerol lowering effect of RS is not clear from this study. In contrast to the rat studies, the cholesterol response to RS is not consistent in human.Some investigators found the significant reduction in serum cholesterol concentration [34]. But in other studies with hyperlipidemic or normolipidemic subjects, raw or retrograded RS did not lower serum lipid concentrations [35–37].

Liver total lipid and cholesterol lowering effect was observed with resistant starch from rice. Resistant starch from corn exerted only total lipid lowering effect. It was speculated that hypocholesterolemic effect of RS was caused by the interference of intestinal cholesterol and bile acid absorption [12]. However, it has been also suggested that other mechanisms involving the alterations in the bilary bile acid profile or repressed hepatic lipogenesis appear to be involved in the hypolipidemic effect of resistant starch [38].

The length of small intestine, and those of cecum, colon and rectum were not changed by RS. Furthermore, cecum weights were not significantly different in RS groups. In other study of meal-fed rats, RS also did not affect the

Table 5
Effect of resistant starch from corn(RS-CORN) and resistant starch from rice(RS-RICE) on length of small and large intestines, cecum weights, and intestinal transit time at 2 weeks of feeding in streptozotocin-induced diabetic rats fed experimental diets

Group	Small intestine length(cm)	Cecum, Colon, Rectum length (cm)	Cecum wights(g)		Transit time at 2 weeks of feeding (minute)
			with contents	without contents	
CONTROL	109.1 ± 9.2^{NS}	18.6 ± 2.3^{NS}	2.1 ± 0.3^{NS}	1.5 ± 0.3^{NS}	705.0 ± 124.2^{a}
DIABETES	107.7 ± 8.6	18.0 ± 3.6	2.4 ± 0.3	1.8 ± 0.5	657.1 ± 66.4^{a}
RS-CORN	110.2 ± 10.2	18.8 ± 2.4	2.3 ± 1.0	1.8 ± 0.8	483.5 ± 45.6^{c}
RS-RICE	107.1 ± 11.6	18.8 ± 3.3	2.2 ± 0.5	1.7 ± 0.6	570.0 ± 44.6^{b}

Data are expressed as means ± standard error.
Values in a column with no common superscript letters are significantly different ($P < 0.05$).
NS means values in a column are not significantly different.

Table 7
Effect of resistant starch from corn(RS-CORN) and resistant starch from rice(RS-RICE) on plasma lipid concentrations in streptozotocin-induced diabetic rats fed experimental diets for three weeks

Group	Total Lipid	Triacylglycerols	Total Cholesterol	(unit:mg/100 ml plasma) HDL-Cholesterol	HDL/total Cholesterol (%)
CONTROL	174.1 ± 21.9^{b}	52.1 ± 11.8^{NS}	54.9 ± 7.2^{ab}	29.1 ± 4.1^{b}	53.9 ± 10.4^{NS}
DIABETES	210.0 ± 41.2^{a}	50.3 ± 12.9	72.1 ± 26.0^{a}	44.8 ± 9.7^{a}	66.2 ± 15.2
RS-CORN	165.6 ± 28.9^{b}	51.9 ± 15.4	48.6 ± 11.7^{b}	25.9 ± 7.3^{b}	53.7 ± 12.3
RS-RICE	171.6 ± 25.8^{b}	53.5 ± 25.4	49.8 ± 14.4^{b}	31.8 ± 9.8^{b}	64.4 ± 10.1

Data are expressed as means ± standard error.
Values in a column with no common superscript letters are significantly different ($P < 0.05$).
NS means values in a column are not significantly different.

length of the small intestine [34]. A rather short experimental period of this study may have resulted in no significant effect of RS on the length of intestines. The present study showed that the intestinal transit time seemed to be decreased by RS, and resistant starch from corn was found to be more effective. Carbohydrates reached large intestines appear to provide a potential energy to colonic microorganisms. Many *in vivo* studies have shown that RS may be readily fermented by colonic microflora [35], but the differences in fermentability were found [36]. It has been claimed that rice starch can be completely fermented by gut bacteria [37]. However, this may not have any relevance to this study, since it has been turned out that there were no significant differences in short chain fatty acids from the intestinal contents. Further clarification of the short chain fatty acid production under intact condition is required to elucidate the exact colonic events followed by the ingestion of RS in diabetic rats. Under fasting condition like in this study it may differ in SCFA production from intact condition. However, one can not rule out the possibility of the minimal effect of special type of RS on SCFA formation, since Kishida *et al.* have observed no differences in SCFA concentrations in high amylose cornstarch compared to the normal cornstarch fed rats [39]. Furthermore, in diabetic conditions gut physiology might be altered, and therefore, it may be difficult to compare to the normal state.

Even though it is hard to extrapolate the beneficial effect of resistant starch in drug-induced diabetic rats to human type 2 diabetic conditions, it is feasible that complications of diabetes such as hyperlipidemia can be controlled using resistant starch. Specially, resistant starch from rice seemed to have a higher potential.

In summary, it has been shown that resistant starch from corn or rice at the level of 16%(w/w) could lower plasma cholesterol concentrations in diabetic rats. Additionally, resistant starch decreased the elevated plasma lipid contents by diabetic condition. Resistant starch from rice was able to lower liver cholesterol contents. The decreased intestinal transit time was observed by feeding both types of resistant starch.

Table 8
Effect of resistant starch from corn (RS-CORN) and resistant starch from rice(RS-RICE) on liver lipid concentrations in streptozotocin-induced diabetic rats fed experimental diets for three weeks

Group	Total Lipid	Triacylglycerols	(unit: mg/g wet liver) Total Cholesterol
CONTROL	29.4 ± 10.4^{a}	9.7 ± 6.0^{a}	6.2 ± 1.4^{a}
DIABETES	25.2 ± 10.8^{ab}	2.7 ± 2.8^{b}	5.0 ± 1.4^{bc}
RS-CORN	18.1 ± 3.8^{b}	2.7 ± 1.5^{b}	5.4 ± 0.8^{ab}
RS-RICE	17.9 ± 3.2^{b}	3.1 ± 3.1^{b}	4.7 ± 1.4^{c}

Data are expressed as means ± standard error.
Values in a column with no common superscript letters are significantly different ($P < 0.05$).

Table 9
Effect of resistant starch from corn(RS-CORN) and resistant starch from rice(RS-RICE) on plasma Immunoglobulin G(IgG) and C_3 in streptozotocin-induced diabetic rats fed experimental diets for three weeks

Group	IgG	(unit:mg/100 ml plasma) C_3
CONTROL	51.1 ± 23.2^{NS}	39.5 ± 5.4^{NS}
DIABETES	59.3 ± 9.9	34.5 ± 9.3
RS-CORN	61.0 ± 5.3	35.4 ± 10.5
RS-RICE	55.6 ± 7.7	35.9 ± 7.5

Data are expressed as means ($n = 10$) ± standard error.
NS means values in a column are not significantly different.

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