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A high-resistance-starch rice diet reduces glycosylated hemoglobin levels and improves the antioxidant status in diabetic rats

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Abstract

Diabetes mellitus is a common problem in developed countries. An improved postprandial hyperglycemic peak is one of the main therapeutic targets in diabetic patients. The Wistar rats with streptozotocin (STZ)-induced diabetes were divided into cornstarch (control) and Japonica rice groups, which were fed 640 g starch/kg diets for 4 weeks. The area (means ± SD) under the glucose curve of cornstarch was 173.8 ± 6.9 and Japonica rice diet was 154.3 ± 8.7 mmol × min/L, and the area (means ± SD) under the insulin curve of cornstarch was 12.9 ± 0.1 and Japonica rice diet was 12.0 ± 0.6 nmol × min/L. The glycosylated hemoglobin levels, serum fructosamine and cholesterol concentrations in diabetic rats fed the Japonica rice diet were significantly lower than the control group ($P < 0.05$). The decreased malondialdehyde levels and increased superoxide dismutase activity and total radical-trapping antioxidant parameter in plasma were also found in rat fed the Japonica rice diet compared to the control. These results suggested that the diet containing high-resistance-starch Japonica rice might reduce glycosylated hemoglobin levels, serum cholesterol concentrations and raised the antioxidant status in the blood.

Keywords: Antioxidant; Streptozotocin; High-resistance-starch rice; Glycosylated hemoglobin; Total radical-trapping antioxidant parameter

1. Introduction

Diabetes mellitus is a common problem in developed countries. The number of diabetic patients is gradually increasing worldwide, and the prevalence is predicted to double by the year 2010 (Heine, 1999). An improved postprandial hyperglycemic peak is one of the main therapeutic targets in diabetic patients. Starchy foods represent the main candidate for reducing the glycemic and insulinemic responses. However, coincidental with recommendations to increase the intake of starchy foods has been the recognition that the glycemic responses to all starches are not the same, and that starches are not interchangeable (Bornet et al., 1989; Cheng & Lai, 2000; Lerer-Metzger et al., 1996), and those issues has been ongoing for 12 yr in our laboratory (Cheng, 1993; Cheng, Chen, Tung, & Shieh, 1994; Cheng & Yu, 1997; Cheng & Lai, 2000). The passage of undigested starch into the colon may limit the amount of glucose that can be absorbed into the small intestine. Foods with more-highly resistant starch should yield lower glycemic index values than those with less-resistant starch (Annison & Topping, 1994). In addition, high-amylose rice products have been found to induce both lower blood glucose and lower insulin responses compared with similar products with higher amylopectin contents (Cheng et al., 1994; Goddard, Young, & Marcus, 1984; Miller, Pang, & Bramall, 1992). However, strong evidence indicates that the total amount of carbohydrates is more important than the source (sugar or starch) or the type (glycemic index) of carbohydrate. Numerous studies have reported that when subjects are allowed to choose from a variety of starches and sugars, the glycemic response is identical if the total amount of carbohydrates is similar. There is insufficient evidence to recommend diets with a low

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Table 1
Composition of experimental diets

Ingredient	Cornstarch (control) (g/kg diet)	Japonica rice (g/kg diet)
Cornstarch	640	
Japonica rice[A]	–	696
Casein[A]	200	146
Soybean oil[A]	100	98
Mineral mixture[A,B]	35	35
Vitamin mixture[A,B]	10	10
α-Cellulose[A]	10	10
DL-Mthionine[A]	3	3
Choline bitartrate[A]	2	2

[A] Japonica (Tainun No. 67) rice was provided by the Taiwan Agricultural Research Institute. Japonica rice had 0.078 g/100 g protein and 0.003 g/100 g oil. Cornstarch was purchased from Roquette Freres Lestrem USP (Lestren, France). Casein, α-cellulose, the AIN-76 mineral mixture, and the AIN-76 vitamin mixture were procured from ICN Biochemicals (Costa Mesa, CA). Soybean oil, DL-methionine and choline bitartrate were procured from Sigma Chemical (St. Louis, MO).
[B] AIN76.

glycemic index as the primary strategy in food and meal planning for individuals with diabetes (American Diabetes Association, 2002; Salmerón et al., 1997).

Oxidative stress has been suggested to play a key role in the pathogenesis of diabetic complications (Baynes & Thorpe, 1999; Ceriello, 2000). Other possible sources include elevated plasma lipids leading to lipid peroxidation and a decreased level of antioxidant defense systems (Halliwell & Gutteridge, 1999). These antioxidants act synergistically in vivo to provide cells with greater protection against radical damage than any single antioxidant can provide by itself. A role of reactive oxygen species (ROS) has been demonstrated as a cause of diabetes induced by chemicals such as streptozotocin (STZ) in experimental animals (Oberley, 1988). However, not only are oxygen radicals involved in the cause, but also the diabetic status itself is associated with increased production of ROS, and this condition in turn has been suggested as one of the pathogenic mechanisms of diabetic complications (Baynes, 1991; Giugliano, Ceriello, & Paolisso, 1996). In this study, Japonica rice or cornstarch was used in each diet that contained the same amounts of starch (640 g/kg) for this experiment (Table 1). The STZ-induced diabetic rats were used to investigate the effects of glycemic responses to the ingestion of Japonica rice and cornstarch on lipid peroxidation, protein glycation, and the antioxidant status.

2. Materials and methods

2.1. Animals

Male Wistar rats (from the Animal Center of the National Science Council, Taipei, Taiwan), weighing about 250 g each, were housed individually in wire-bottomed stainless steel cages in a temperature- and humidity-controlled room (at 22 °C), with a 12-h light–dark cycle and free access to food and water. All procedures of the animal experiments followed published guidelines (National Science Council, 1994).

2.2. Diets

Two different kinds of carbohydrate resources (Japonica rice or cornstarch) contained 640 g/kg starch were used for animal feedings (Table 1). Each diet as analyses of moisture, crude fats, and crude proteins were carried out according to methods of the AOAC (1980) on the subject materials. The husks of the rice grains were removed to yield polished rice. Polished rice flour and cornstarch were precooked at 180 °C for 4 h.

2.3. Glycemic and insulinemic responses in rats without diabetes

Two meals contained 640 g/kg starch were fed to 20 non-diabetic male Wistar rats. Rats were placed in individual cages and trained for 2 week. Rats that consumed all of the meal (within 20 min) were anesthetized with pentobarbital and bled from the tail. Blood samples were taken after 15, 30, 45, 60, 90, and 120 min to measure plasma glucose and insulin concentrations. The total changes in values over 2 h were calculated from the area under the time–concentration curve (AUC) using the trapezoidal rule by adding the each AUC segment (the AUC_{0-15}, AUC_{15-30}, AUC_{30-45}, AUC_{45-60}, AUC_{60-90}, AUC_{90-120}) for glucose and insulin. Plasma glucose and insulin concentrations at 0 min had been determined in the same rats on a separate day under similar experimental conditions.

2.4. Rats with STZ-induced diabetes

After acclimation for 1 week, animals (16 rats) were weighed, fasted overnight, anesthetized with light ether, and then administered 65 mg STZ/kg body weight via an intraperitoneal injection. STZ was dissolved in saline immediately before injection (Junod, Lambert, Stauffacher, & Renold, 1969). One week following the induction of diabetes, the fasting level of blood glucose was measured to detect STZ-induced hyperglycemia. Animals with elevated fasting levels of blood glucose (>15 mmol/L) were randomly divided into two groups of 8 rats each, and fed a different diet for 4 week. Two different kinds of carbohydrate resources (Japonica rice or cornstarch) contained 640 g/kg starch were used for animal feedings (Table 1). Food was withheld for 12 h at the end of week 4.

2.5. Serum and tissue collection and processing

Rats were anesthetized with 1 g/L sodium pentobarbital and dissected. Blood was collected from the abdominal aorta, incubated at room temperature for 45 min and centrifuged at 4000*g* for 15 min. The serum was then stored in a freezer at −70 °C. The liver and kidneys were excised, rinsed in a 9 g/L NaCl solution and stored tightly sealed at −70 °C

until analysis. Tissues were homogenized in 2 vol of 1 mM EDTA and 10 mM Tris buffer (pH 7.4) at 4 °C for 10 s. The homogenate was filtered through cloth, and the filtrate was centrifuged at 105,000*g* for 1 h using a refrigerated centrifuge (Beckman model TL-100). The resultant supernatant was used for further assays.

2.6. Analytical methods

2.6.1. Amylose, resistant starch, and glucose contents

The amylose contents in these two diets were measured according to the method of Juliano et al. (1981). Resistant starch was analyzed according to the method of Englyst, Kingman, and Cummings (1992). Five aliquots of these diets (in triplicate assays) were subjected after 1 min of sieving to α-amylase hydrolysis with 300 U of porcine pancreatic α-amylase (Sigma Chemical, St. Louis, MO) with constant stirring (30 rpm) in a phosphate buffer (5 mmol/L; pH 7) for 3 h at 37 °C. Every 5 min, a 0.90-mL sample was mixed with 4.5 mL ethanol (80%) and 0.25 mol/L acetic acid and then stored overnight at 4 °C (Kabir et al., 1998). The next day, samples were centrifuged at 9000*g* for 10 min at 4 °C. The soluble glucose in the supernatant and serum glucose were determined using a glucose analyzer (Helena Laboratory, Sunderland, UK).

2.6.2. Insulin, glycosylated hemoglobin, and fructosamine contents

Serum insulin was determined using a radioimmunological method (Rat Insulin Mercodia kit, American Laboratory Products Company, Uppsala, Sweden). Blood glycosylated hemoglobin was measured using spectrophotometric method (procedure kit, Helena Laboratory). Serum concentrations of fructosamine were determined by a colorimetric method (FRUC kit, Roche Diagnostics, Mannheim, Germany).

2.6.3. Triglyceride and total cholesterol contents

The serum triglyceride concentration was determined as described by Mcgowan, Artiss, Strandbergh, and Zak (1983), and the procedure of Richmond (1973) was used to determine the serum total cholesterol concentration. The liver (1.5 g) was cut and extracted with 20 mL chloroform/methanol (2:1, v/v) according to the method of Folch, Lees, and Sloane-Stanley (1957). After the addition of 4 mL of a 0.5 g/L $CaCl_2$ (w/v) solution, the extracts were collected and stored. The liver lipid extracts were accurately weighed into a glass tube with a screw top, according to the method of Soloni (1971) and were treated according to the method of Carlson and Goldfabr (1977). Triolein was used to plot a standard solution and to determine the liver triglyceride contents. Liver total cholesterol concentrations were determined according to the method of Richmond (1973).

2.6.4. Lipid peroxidation and the total radical-trapping antioxidant parameter (TRAP) assays

Plasma and tissue malondialdehyde levels were measured using the method of Yagi (1984). Lipid peroxidation products were assayed according to an improved thiobarbituric acid-reactive substance (TBARS) fluorometric method with emission at 553 nm and excitation at 515 nm, with 1,1,3,3-tetramethoxypropane used as the standard (Ghiselli, Serafini, Maiani, Azzini, & Ferro-Luzzi, 1995).

2.6.5. Antioxidant enzymes assay

Catalase (CAT) activity in rat hepatocytes was determined as they were harvested (Baudhuin et al., 1964). The remaining supernatant fractions were used to determine the activities of superoxide dismutase (SOD, McCord & Fridovich, 1969) and glutathione peroxidase (GSH-Px, Lawrence & Burk, 1976).

2.6.6. Total protein, hemoglobin, creatinine, and uric acid assays

Total protein was determined using the method of Lowry, Rosebrough, Farr, and Randall (1951), with bovine serum albumin as the standard. Hemoglobin, creatinine, and uric acid were measured using a RANDOX kit (Crumlin, Antrim, UK).

2.6.7. Statistical analysis

All analyses were done in triplicates. Data were analyzed by one-way ANOVA using the SAS general linear model program. Values are reported as the means ± SEM. Group means were considered to significantly differ at $P < 0.05$, as determined by Duncan's new multiple range analysis.

3. Results

3.1. Characterization of the diets

The amylose content in the cornstarch diet is about 2.7-fold of that in the Japonica rice diet (Table 2). Fig. 1 shows the postprandial blood glucose (a) and postprandial blood insulin (b) in normal Wistar rats intragastrically fed two diets during 2 h. The AUC (Fig. 1 and Table 2) for blood glucose in the cornstarch diet is 173 ± 6.9 and is 154 ± 8.7 mmol × min/L in the Japonica rice diet. The AUC for blood insulin (Fig. 1 and Table 2) in the cornstarch diet is

Table 2
Levels of amylose, resistant starch, digestibility, $glucose_{AUC}$, and $insulin_{AUC}$ of starch in the cornstarch and Japonica rice diets[A]

Type of starch	Cornstarch (control)	Japonica rice
Amylose[B] (g/kg)	494.02 ± 1.84^{a}	181.01 ± 0.67^{b}
Resistant starch[B] (g/kg)	8.01 ± 0.09^{b}	24.11 ± 0.08^{a}
Digestibility in vitro[B] (%/30 min)	48.77 ± 1.85^{a}	44.19 ± 0.96^{b}
$Glucose_{AUC}$[C] (mmol × min/L)	173.88 ± 6.97^{a}	154.34 ± 8.72^{b}
$Insulin_{AUC}$[C] (nmol × min/L)	12.96 ± 0.16^{a}	12.00 ± 0.64^{b}

[A] Values are the means ± SEM, $n = 8$; different superscript letters in a row indicate significantly different means, $P < 0.05$.

[B] Amylose was measured by the method of Juliano et al. (1981); resistant starch was measured by the method of Englyst et al. (1992); digestibility in vitro was measured by the method of Bornet et al. (1989).

[C] $Glucose_{AUC}$, area under the glucose curve in non-diabetic rats, which was calculated from Fig. 1a; $Insulin_{AUC}$, area under the insulin curve in non-diabetic rats, which was calculated from Fig. 1b.

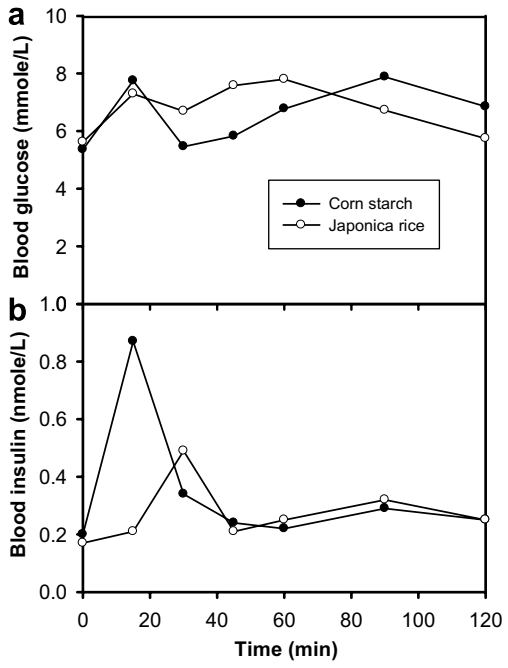

Fig. 1. Postprandial blood glucose (a) and postprandial blood insulin (b) in normal Wistar rats intragastrically fed 2 g of a mixed diet containing 640 g starch/kg as either cornstarch or Japonica rice diet during 2 h. Rats were anesthetized with pentobarbital and bled from the tail; blood samples were taken after 0, 15, 45, 60, 90, and 120 min to measure plasma glucose and insulin concentrations ($n = 9$).

12.9 ± 0.1 and is 12.0 ± 0.6 mmol × min/L in the Japonica rice diet. From calculations, the glycemic (calculated from Fig. 1a) and insulinemic responses (calculated from Fig. 1b) using the AUC present in normal rats fed the Japonica rice diets were significantly lower compared with those of rats fed the cornstarch diets ($P < 0.05$; Table 2). The cornstarch diets were also found to have higher *in vitro* digestibility and lower resistance-starch compared to the Japonica diet (Table 2).

3.2. Weight gain, food intake, and blood glucose in STZ-induced diabetic rats

The use of 45 mg of STZ per kg of body weight by intraperitoneal administration effectively induced diabetes in those rats, as evidenced by the increased amount of blood glucose (from 5 to 15 mmol/L) and fructosamine (from 0.2 to 0.37 mmol/L) compared with normal rats and the blood insulin concentrations decreased from 124 (in normal rats) to 93 mmol/L (in diabetic rats) (data not shown). However, the daily food intake, weight gain and concentrations of blood glucose after the experiment did not show significant differences between the two groups in STZ-induced diabetic rats (Table 3).

3.3. Fructosamine, glycosylated hemoglobin, triglyceride, and cholesterol in STZ-induced diabetic rats

The fructosamine concentrations, glycosylated hemoglobin, and serum cholesterol showed significantly different and lowered in Japonica rice diets compared to those in the cornstarch diets ($P < 0.05$; Table 3) after 4 week feedings. Though the lower serum triglyceride was found in diabetic rats fed the Japonica rice diets, however, there were no significant differences between these two meals.

Table 3
Food intake, weight gain, blood glucose, fructosamine, glycosylated hemoglobin serum triglyceride, serum cholesterol, liver triglyceride, and liver cholesterol in rats with streptozotocin-induced diabetes fed the cornstarch and Japonica rice diets for 4 week[A]

	Cornstarch (control)	Japonica rice
Food intake (g/d)	31.49 ± 0.94	32.02 ± 2.13
Weight gain (g)	30.60 ± 2.72	34.70 ± 3.33
Initial blood glucose (mmol/L)	15.44 ± 0.43	15.38 ± 0.40
Final blood glucose (mmol/L)	19.75 ± 1.04	19.48 ± 0.90
Fructosamine (μmol/L)	379.43 ± 16.53^{a}	355.77 ± 18.29^{b}
Glycosylated hemoglobin (%)	5.81 ± 0.36^{a}	5.49 ± 0.18^{b}
Serum triglyceride (mmol/L)	0.97 ± 0.06	0.89 ± 0.10
Serum cholesterol (mmol/L)	2.60 ± 0.10^{a}	2.40 ± 0.11^{b}
Liver triglyceride (mmol/g liver)	0.45 ± 0.06	0.41 ± 0.03
Liver cholesterol (mmol/g liver)	0.41 ± 0.03	0.41 ± 0.03

[A] Values are the means ± SEM, $n = 8$; different superscript letters in a row indicate significantly different means, $P < 0.05$.

3.4. Oxidative stress parameters in STZ-induced diabetic rats

Diabetic rats fed the Japonica rice diet showed significantly different ($P < 0.05$; Table 4) and had a lower MDA concentrations in serum and liver than those of the cornstarch groups. However, the higher SOD activities and lower GSH-Px activities from serum, liver, and kidneys showed significantly different ($P < 0.05$; Table 4) in Japonica rice diet than those of the cornstarch diet. The TRAP in

Table 4
Oxidative stress parameters evaluated in rats with streptozotocin-induced diabetes fed the cornstarch and Japonica rice diets for 4 week[A]

	Cornstarch (control)	Japonica rice
Serum		
MDA[B] (nmol/L)	1.15 ± 0.09^{a}	1.01 ± 0.04^{b}
GSH-Px[B] (nmol/min/g Hb)	257.09 ± 7.88^{a}	247.85 ± 8.26^{b}
CAT[B] (k/s/gHb)	0.42 ± 0.06^{a}	0.35 ± 0.02^{b}
SOD[B] (U/gHb)	1453.0 ± 83.5^{b}	1531.9 ± 114.4^{a}
Liver		
MDA[B] (nmol/mg protein)	0.11 ± 0.02^{a}	0.07 ± 0.01^{b}
GSH-Px[B] (nmol/min/mg protein)	400.75 ± 15.74^{a}	381.6 ± 16.92^{b}
CAT[B] (k/s/mg protein)	0.20 ± 0.03^{b}	0.24 ± 0.03^{a}
SOD[B] (U/mg protein)	16.1 ± 1.38^{b}	22.13 ± 1.08^{a}
Kidney		
MDA[B] (nmol/mg protein)	0.26 ± 0.03	0.20 ± 0.01
GSH-Px[B] (nmol/min/mg protein)	435.35 ± 57.72	480.32 ± 35.93
CAT[B] (k/s/mg protein)	0.04 ± 0.004	0.04 ± 0.004
SOD[B] (U/mg protein)	11.31 ± 0.97^{b}	13.65 ± 0.95^{a}

[A] Values are the means ± SEM, $n = 7$; different superscript letters in a row indicate significantly different means, $P < 0.05$.

[B] CAT, catalase; GSH-Px, glutathione peroxidase; MDA, malondialdehyde; SOD, superoxide dismutase.

Table 5
Plasma total radical-trapping antioxidant parameter (TRAP), uric acid, and creatinine evaluated in rats with streptozotocin-induced diabetes fed the cornstarch and Japonica rice diets for 4 week[A]

	Cornstarch (control)	Japonica
Plasma TRAP (μM)	88.86 ± 5.50^{b}	121.49 ± 12.46^{a}
Uric acid (mmol/L)	0.52 ± 0.04^{a}	0.51 ± 0.02^{b}
Creatinine (μmol/L)	135.25 ± 15.99^{a}	80.44 ± 7.14^{b}

[A] Values are the means ± SEM, $n = 7$; different superscript letters in a row indicate significantly different means, $P < 0.05$.

diabetic rats fed the Japonica rice diet showed significantly different ($P < 0.05$; Table 5) and had 37% higher than that of the cornstarch diet group. The creatinine and uric acid concentrations in rats fed the Japonica rice diet showed significantly different ($P < 0.05$) and had 41% lower than those of the cornstarch groups.

4. Discussion

The present study showed that the high-resistance-starch in Japonica rice diet reduces glycemic and insulinemic responses of normal rats and reduces serum cholesterol, fructosamine, creatinine concentrations, glycosylated hemoglobin and MDA levels, and increases TRAP of STZ-induced diabetic rats compared to the cornstarch diets.

The reduced fructosamine and glycosylated hemoglobin in STZ-induced diabetic rats can be partially explained by high-resistance-starch contents in Japonica rice diets. Japonica rice starch is digested more slowly, then alters lipid metabolism. The reduction in blood glucose in rats fed a high-resistance-starch diet might also have been due to the dietary carbohydrates of Japonica rice being digested and absorbed at slower rates and to lesser extents in the animal's small intestine (Cummings & Roberfroid, 1997). There is consistent evidence in the scientific literature that diets with more-highly resistant starch should yield lower glycemic index values than those with less-resistant starch (Annison & Topping, 1994). Thus, the passage of undigested starch into the colon limits the amount of glucose that can be absorbed into the small intestine. The amylopectin also induces insulin resistance (Wiseman, Higgins, Denyer, & Miller, 1996). Even a relatively small proportion of uncooked amylose (270 g/kg) is sufficient to achieve a maximal attenuating effect on postprandial insulin concentrations as compared with 0 g amylose/kg total starch. Following cooking, however, a much-higher proportion of amylose (600 g/kg total starch) is needed to achieve a similar effect (Brown, Storlien, Brown, & Higgins, 2003).

Resistant rice starch may be fermented to produce propionate, which reduces serum and hepatic cholesterol in hyperchlosterolemic rats (Cheng & Lai, 2000). Thus, this results in reduced serum lipids leading to decreased lipid peroxidation and increased levels of antioxidant defense systems. In the current study, feeding the Japonica diet produced lower serum and liver MDA levels, higher serum and liver SOD activities, and higher plasma total radical-trapping antioxidant properties compared to the cornstarch diet on diabetic rats (Tables 3 and 4). We speculated that these changes might be adaptations to significant decreases in plasma cholesterol, fructosamine concentrations, and glycosylated hemoglobin levels. The present results confirmed that intravenous administration of 45 mg STZ per kg of body weight effectively induces diabetes in rats. There is consistent evidence that the total antioxidant status in type 1 or 2 diabetes mellitus is lower than that of age-matched controls, and this might be attributed to lower levels of vitamin C and vitamin E (Maxwell et al., 1997) or other factors including niacin (as niacinamide), the minerals zinc, chromium and vanadium (Cunningham, 1998). The assay of TRAP was proposed to evaluate the plasma antioxidant capacity, by taking into consideration known and unknown antioxidants present in the plasma as well as their mutual interactions (Ghiselli et al., 1995). A reduced TRAP value has previously been reported in patients with non-insulin-dependent diabetes mellitus (NIDDM) (Danjo et al., 2003). The current study suggests that in diabetic rats, feeding the Japonica rice diet resulted in the plasma TRAPs being significantly higher than those in the cornstarch group. A recent study showed that blockade of hyperglycemia-induced ROS production reverses the pathways implicated in diabetic angiopathy: activation of protein kinase C, formation of advanced glycation end products, and elevating of the sorbitol content in cultured endothelial cells (Nishikawa et al., 2000). This suggests that enhanced ROS production in diabetes may be a common pathway linking diverse pathogenic mechanisms of diabetic vascular complications.

5. Conclusions

The present data demonstrate that Japonica rice has potential antihyperglycemic effects. In addition, Japonica rice feeding had significant effects on elevation of total plasma radical-trapping activity, which evaluates the plasma antioxidant capacity due to known and unknown antioxidants present in the plasma; in addition, their creatinine concentrations were also significantly reduced. These findings support the conclusions that a Japonica rice diet as the carbohydrate source has a potential effect in improving the antioxidant status compared to a cornstarch diet in rats with STZ-induced diabetes.

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