Preliminary Report

Glycemic and Lipid Responses to Glucomannan in Thais with Type 2 Diabetes Mellitus^Ä

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Objective: *To evaluate the benefits of glucomannan supplement on glycemic and lipid controls in type 2 diabetic patients.*

Material and Method: *A single-blind, placebo-controlled, crossover trial with two treatments separated by a 2-week washout period was performed in 10 men and 10 women with type 2 diabetes mellitus. Two separated protocols of experiments were sequentially followed. Initially, purified glucomannan (1 g) or placebo was ingested 30 min before 75-g glucose load to evaluate their effects on glucose absorption and insulin secretion in oral glucose tolerance test (OGTT). Later, the glycemic and lipid changes after 4-week intervention with 3 g/day glucomannan comparing to the placebo were determined. The standard OGTT was performed before and after ending of each intervention.*

Results: *Glucomannan taken before performing the OGTT can lower the rise of blood glucose and insulin from 1 to 2 hour in comparison with the placebo, though a statistically significance of insulin was not achieved. Long- term glucomannan supplement significantly reduced the 120-min glucose area under the curve of OGTT. Glucomannan also decreased the rise of low-density lipoprotein cholesterol (LDL-C). Reductions of HOMA-insulin resistance index and body mass index were detected in glucomannan-treated group though the former was shown only in females. No within- and between-group differences of insulin, fructosamine, and other lipids were observed in glucomannan- nor placebo- treated groups.*

Conclusion: *In type 2 diabetes, pre-prandial glucomannan ingestion attenuated a rise of blood glucose without significantly affecting insulin levels. Long-term supplement of glucomannan to the regular diabetic regimen lessened post challenge glucose AUC and impeded the rise of LDL-C. Supplement of glucomannan may be beneficial to the glycemic and lipid controls in type 2 diabetes mellitus.*

Keywords: *Glucomannan, Soluble dietary fiber, Type 2 diabetes, OGTT, Thais*

The prevalence of diabetes mellitus in Thailand was estimated to have increases from 2.4% in 1995 to 3.7% by 2025[1]. Diabetic individuals develop long-term complications both microvascular and macrovascular impairments. Clinical trials have demonstrated the importance of glycemic control in preventing and reducing diabetic complications[2,3]. The ADA recommends glycemic targets for type 2 diabetes at preprandial glucose < 140 mg/dL, 1-to 2- hour postprandial blood glucose < 180 mg/dL, and HbA_1C indicating average glycemia over a six- to eight-week period < 7%[4]. However, a more intensive control of fasting blood glucose at 100-125 mg/dL and 2-h OGTT value of 140-199 mg/dL is suggested for macrovascular disease patients[4].

Most characteristic dyslipidemia in type 2 diabetes are moderate elevation of triglyceride levels,

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low HDL-C values and relatively normal levels of LDL-C carried in highly - atherogenic small, dense LDL particles[5]. The treatment strategies for diabetic dys-lipidemia are firstly, lowering LDL-C to < 100 mg/dL, approximately 30% reduction from pretreatment value, secondly, raising HDL-C to > 40 mg/dL in men or to > 50 mg/dL in women, and lastly, lowering triglyceride to < 150 mg/dL[4,6].

Management of diabetic patients is generally a multifactorial intervention to prevent or delay both acute and chronic complications associated with long-term diseases. Among these, nutrition and lifestyle interventions aimed for weight reduction are empha-sized as the initial step of diabetic management prior to medications[7,8]. The benefits of food with increased fiber contents, low glycemic indexes, and low saturated fat levels in type 2 diabetic patients have been shown and approved[7,9]. Previous studies suggested that soluble fiber was better than insoluble fiber for con-trolling of glucose and lipid metabolisms in type 2 dia-betes[9,10].

Glucomannan is a highly soluble dietary fiber derived from the tubers of Amorphophallus konjac. It is made into several food products for traditional cooking in many Asian countries or as food-additive worldwide and available in purified form for fiber sup-plements. Glucomannan lessened the rise in blood glucose and lipids when given as part of a test meal in type 2 diabetes[11,12]. No well-known drug interaction with glucomannan was reported. However, Shima K et al found that glucomannan blunted the intestinal absorption of glibencamide in healthy subjects[13]. In Thailand, glucomannan is easily accessible and costs less than antidiabetic and lipid-lowering drugs. The present study aims to test an immediate effect of konjac glucomannan on 2-hour post challenge glucose level, which is a determinant of glucose intolerance and considered a better predictor of deaths from all causes and cardiovascular disease than the fasting blood sugar[14,15]. To the authors' knowledge, this has not been studied among Thais with type 2 diabetes mellitus. In addition, the effects of 4-week glucomannan consump-tion on glycemic and lipid controls of diabetes will be assessed.

Material and Method
Subjects
Twenty type 2 diabetic patients, 10 men and 10 women, aged 30-70 years, were under dietary con-trol and/or oral diabetic drug or insulin therapy. Bio-chemical screening of liver and kidney dysfunctions were done before recruiting them into the study. Sub-jects were excluded from the study if they were known to have disease of gastrointestinal tract, kidney, heart and any types of cancer. All subjects gave the written informed consent and completed the questionnaires concerning their medical history, cigarette smoking, alcohol drinking, and habitual dietary consumption.

Study design
Two separated protocols of single-blind, placebo-controlled, crossover trial were performed to investigate an immediate and the long-term responses of patients to glucomannan. In the first protocol, ten patients, after 12-hour overnight fast, consumed 2 cap-sules of purified glucomannan (1 g) with 200 mL water at 30 min prior to standard 75-g oral glucose tolerance test (OGTT) while another ten subjects were given 2 capsules of placebo (1 g white rice flour) instead of the glucomannan. A crossover treatment was done after 2-week washout interval. Blood was collected 30 min before having glucomannan or placebo, then at 1 and 2 hours after 75-g glucose load. Each blood sample obtained was analyzed for glucose and insulin.

In the second protocol, ten subjects took 1 g glucomannan while another ten subjects took placebo together with anti-diabetic drug(s) before 3 meals of the day for 4-week duration. The OGTT was performed before and after glucomannan or placebo intervention. The crossover trial was performed after 2-week wash-out period. Blood samples obtained before and after OGTT were analyzed for glucose and insulin. Fasting levels of fructosamine, triglyceride (TG), total choles-terol (TC), high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C) were measured. Serum fructosamine was used instead of HbA_1C to monitor the more rapidly changes of blood glucose that occurred over the past 2-3 weeks[16]. All subjects were instructed not to change their eating or exercise habits throughout the study period. The food records[17] and rating of appetite on visual analogue scale were performed during the run-in phase and twice a week throughout ten-week study period. Drug com-pliance was monitored at the end of each treatment. The experimental protocols were approved by the Human Ethics Committees of the Faculty of Medicine Siriraj Hospital, Mahidol University.

Laboratory assays
Blood glucose, TC, TG, HDL-C, and fructosa-mine were analyzed by colorimetric method (Hitachi 917 automatic analyzer). LDL-C was calculated using

Friedewald formula. Insulin was measured by radio-immunoassay (Linco Research, Inc.). A homeostasis model assessment of insulin resistance (HOMA-IR) was calculated from fasting glucose and insulin concentrations[18].

Statistical analysis

The results are expressed as mean and standard error of mean $\pm$ SEM. A distribution of data was tested by Kolmogorov Smirnov test. Both within- and between- treatment comparison was done with paired t-test. The appetite score and macronutrient compositions of food taken on the different days were compared using the repeated measures ANOVA followed by Bonferroni/Dunn post-hoc comparison test. The SAS StatView program was used for statistical analysis and area under the curve of glucose and insulin was calculated by Areamacros v 1.5 program. A p-value of less than 0.05 was considered statistically significant difference.

Results

The characteristics of studied patients are shown in Table 1. During the 10-week study period, subjects consumed on average $1,381.81 \pm 30.62$ calories per day consisting of $55.44 \pm 0.64\%$ of carbohydrate, $16.42 \pm 0.27\%$ of protein and $28.15 \pm 0.51\%$ of fat. No significant difference of daily total energy intake and percent of macronutrients throughout the experiment were observed ($p < 0.05$). The appetite scores during treatments with glucomannan and placebo did not differ (5.16 ± 0.27 and 5.32 ± 0.26, respectively, on 1-10 scale, $p < 0.05$). Patient's acceptance of encapsulated glucomannan was very good and the drug compliance during the present study was about 95%. No major adverse effect was detected throughout the experiments except a report of loose stool by a few subjects on glucomannan. Most patients were con-

Table 1. General characteristics of subjects[1]

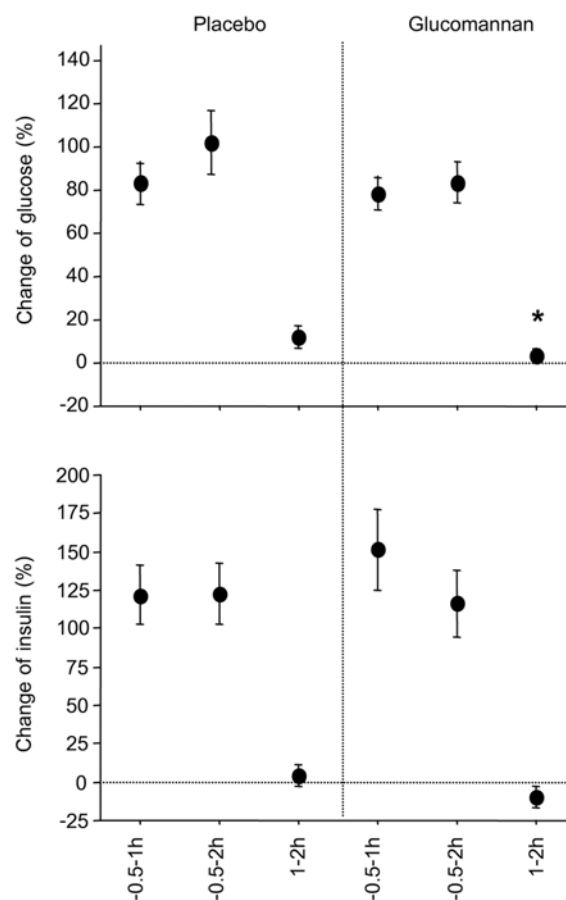| Characteristics	Men (n = 10)	Women (n = 10)
Age (year)	51.40 ± 2.26	51.00 ± 2.16
Body weight (kg)	71.21 ± 4.28	66.25 ± 2.82
Height (cm)	168.50 ± 1.90	155.90 ± 1.85
BMI (kg/m^2)	25.12 ± 1.54	27.41 ± 1.44
Waist circumference (cm)	90.05 ± 4.08	88.70 ± 2.91
Waist hip ratio	0.95 ± 0.01	0.89 ± 0.01

[1] values are mean $\pm$ SEM, n = 20

Fig. 1 Percent changes of blood glucose and insulin concentrations in type 2 diabetic patients taking glucomannan or placebo 30 min before 75 g glucose load (n = 20, * p <0.05 between-group difference)

trolled by oral hypoglycemic drugs except the two patients who were on insulin plus oral anti-diabetic drugs and another three of them were under dietary control. Patients' medication was maintained constant throughout the present study.

The rise of glucose from 1 to 2 hours after taking 1 g glucomannan at 30 min prior to 75-g glucose load was significantly lower than the group of placebo ($3.3 \pm 3.2\%$ and $12.88 \pm 4.95\%$ respectively, p 0.032). However, no significant change of insulin in both treatment groups was observed (Fig. 1).

Long-term (4-week) intervention by 3 g/day glucomannan significantly reduced the 120-min glucose AUC while the same duration of having placebo showed no significant change (Table 2) Fasting levels of glucose, insulin, fructosamine, TC, TG, HDL-C and

LDL-C on long-term trials with glucomannan or placebo did not show any significant alteration. However, mean LDL-C concentration after receiving glucomannan was less than that of after receiving placebo ($p < 0.01$). An index of insulin resistance (HOMA-IR) before the dietary fiber intervention was 12.12 ± 1.19 and 6.56 ± 0.53 mU/L*mmoL/L in women and men, respectively ($p < 0.0001$ between sexes). Less HOMA-IR values after taking glucomannan than after taking placebo were shown in female but not in male patients (Fig. 2). In addition, the BMI after taking glucomannan was less than after taking placebo (Fig. 3). A direct relationship between the 2-hour blood glucose values and the HOMA-IR was shown in Fig. 4. Serum fructosamine correlated positively with fasting blood glucose, 2-hour blood glucose, TC and LDL-C (Table 3).
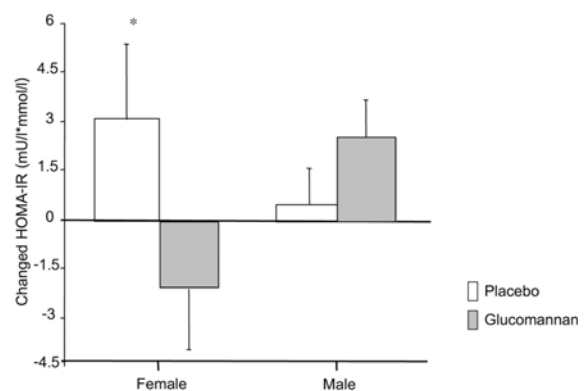

Fig. 2 Changes of HOMA-IR in type 2 diabetic patients after 4-week glucomannan or placebo ($n = 10$ for each sex, * $p < 0.05$ between glucomannan and placebo)

Table 2. The responses of body weight, BMI, glycemic factors and lipids before and after 4-week glucomannan and placebo trials

Variables	Glucomannan[1]		Placebo[1]	
	Pre	Post	Pre	Post
Body weight (kg)	69.50 ± 2.88	69.11 ± 2.82	69.10 ± 2.73	69.43 ± 2.79
BMI (kg/m^2)	26.55 ± 1.17	26.39 ± 1.13	26.40 ± 1.12	26.52 ± 1.13
Fasting glucose (mg/dL)	163.40 ± 13.29	159.15 ± 12.54	158.55 ± 10.78	163.05 ± 9.72
Fasting insulin (µU/mL)	22.70 ± 3.61	25.46 ± 3.53	22.46 ± 3.92	26.51 ± 4.66
Fasting fructosamine (µmol/l)	341.40 ± 24.14	342.80 ± 13.69	323.00 ± 14.09	350.35 ± 19.60[2]
2-hour glucose (mg/dL)	312.95 ± 16.20	308.60 ± 17.96	304.20 ± 15.64	309.65 ± 17.27
Glucose AUC (mg/dl 2h)	218.85 ± 12.44	201.83 ± 14.61[3]	210.25 ± 13.92	199.08 ± 14.83
Insulin AUC (µU/ml 2h)	45.62 ± 9.34	42.68 ± 9.98	42.50 ± 8.54	36.03 ± 8.13
HOMA-IR(mU/l*mmol/L)	9.22 ± 1.71	9.41 ± 0.98	8.50 ± 1.41	10.23 ± 1.63
TG (mg/dL)	264.60 ± 49.56	296.46 ± 86.73	243.36 ± 47.79	235.40 ± 46.02
TC (mg/dL)	191.12 ± 10.04	189.19 ± 12.74	192.66 ± 11.20	198.46 ± 11.20
LDL-C (mg/dL)	92.28 ± 6.95	91.89 ± 7.49[4]	96.14 ± 7.34	104.25 ± 8.11
HDL-C (mg/dL)	52.51 ± 3.09	50.97 ± 3.09	53.28 ± 3.09	52.90 ± 2.70

[1] Values in mean $\pm$ SEM, n = 20
[2] Significantly higher than pre placebo value at *p* < 0.05
[3] Significantly lesser than pre glucomannan value at *p* < 0.05
[4] Significantly lesser than post placebo value at *p* < 0.01

Table 3. The relationship between blood fructosamine (X) and fasting glucose, 2-hour glucose, TC and LDL-C[1]

Y	Regression equation	Correlation coefficient (r)	p-value
Fasting glucose (mg/dL)	$Y = 44.616 + 0.343 * X$	0.54	<.0001
2-Hour glucose (mg/dL)	$Y = 161.283 + 0.435 * X$	0.48	<.0001
TC (mg/dL)	$Y = 115.960 + 0.228 * X$	0.37	<.005
LDL-C (mg/dL)	$Y = 61.498 + 0.102 * X$	0.25	<.05

[1] Values in mean $\pm$ SEM, n = 80
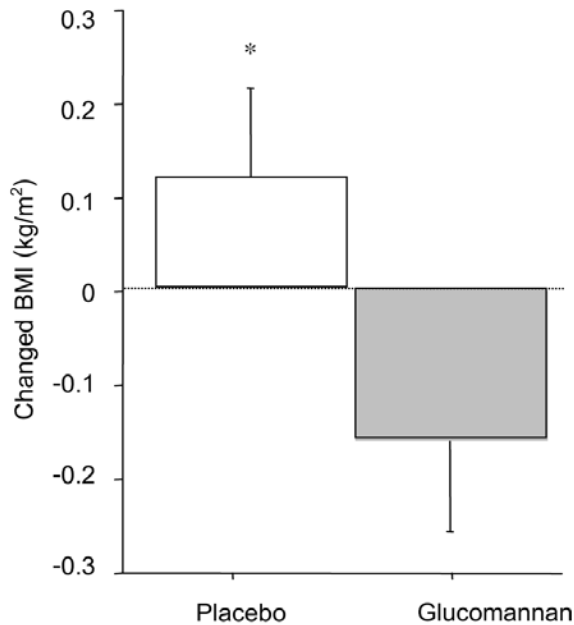
Fig. 3 Changes of BMI in type 2 diabetic patients after 4-week glucomannan or placebo (n = 20, * p < 0.05 between -group difference)
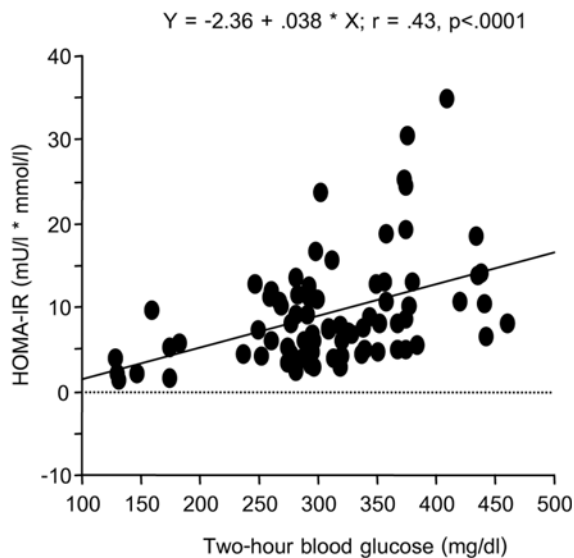

Y = -2.36 + .038 * X; r = .43, p<.0001

Fig. 4 Positive relationships between 2-hour blood glucose concentrations and HOMA-IR (n = 80)

Discussion

Data from the present study showed that in type 2 diabetic patients, glucomannan lowered the rising plasma glucose without alteration of plasma insulin when taken before the OGTT. This result is similar to previous studies in obese and healthy subjects[19,20]. The exact mechanism of how glucomannan can reduce postprandial blood glucose was not investigated in the present study. However, it is believed to be similar to those of other soluble dietary fibers i.e. it increases viscosity of gastrointestinal contents, slows gastric emptying, and acts as a barrier to mucosal diffusion[19,21]. Since the postprandial glucose is an independent risk factor of cardiovascular disease, preprandial glucomannan intake, which is able to reduce the magnitude of postprandial glucose rise, may be helpful in reducing the risk of cardiovascular disease.

Previous studies showed both decrease[12,19] and unchanged[11] of fasting blood glucose after a long-term usage of glucomannan. Huang et al reported the glucomannan-induced decreases of both fasting and postprandial glucose levels in type 2 diabetes[22]. In the present study, postchallenge hyperglycemia in type 2 diabetic patients shown by the 2-h AUC of blood glucose was reduced after 4-week glucomannan supplement (3 g/day). Vuksan et al also reported a decrease in serum fructosamine after 3-week of high dose glucomannan (~15g/day) in biscuit form[11]. However, the authors only noticed a significant increase of serum fructosamine in diabetic patients after four weeks of placebo intervention but not in the glucomannan intervention. In addition, a change of serum fructosamine level was in the same direction as those of the fasting and 2-hour blood glucose - the indices of glycemic control, and those of the lipids TC and LDL-C. In comparison with the glycated hemoglobin (HbA$_1$C), blood fructosamine is more related to dietary sugar intake and may be a marker of exposure to dietary carbohydrate, particularly simple sugars[23]. This may reflect a benefit of glucomannan on postprandial hyperglycemia induced by carbohydrate-rich meal among Thai diabetic patients.

Our data show that the 2-hour blood glucose levels can predict an insulin resistant state in diabetic patients (Fig. 4). In addition, mean HOMA-IR decrease in female subjects taking glucomnnan but increase in the placebo-taking females. However, the authors did not find similar results in the male subjects. The glycemic responses to glucomannan may vary according to the differences in metabolic background, age and sex of diabetic patients as well as the dosage and duration of glucomannan consumption. The impacts of sex on insulin secretion, insulin action, hepatic insulin extraction, and glucose effectiveness, resulting in a more marked deterioration of glucose tolerance in women

than men, have been recently reported[24].

Dyslipidemia is frequently found in patients with type 2 diabetes. LDL-C though does not obviously increase in diabetes, retains its properties to increase atherogenesis. LDL-C has been shown to be the strongest predictor of coronary heart disease followed by HDL-C [25]. Previous studies reported the reduction of LDL-C with unchanged TG levels in diabetic patients receiving glucomannan supplement[11, 12]. Although the within-treatment difference of LDL-C concentrations in glucomannan-treated group was not found in the present study, the between-treatment difference compared to the placebo was detected. Glucomannan supplement in type 2 diabetes was found with significantly increased fecal neutral sterol and bile acid contents thus improving blood lipid levels[12]. Compared to other gel-forming fibers, glucomannan has three-to five folds stronger lipid-lowering effect[26].

Both obesity and diabetes mellitus increase the risk of coronary heart disease. Increasing trends of severity of cardiovascular risk factors and prevalence of morbidity conditioned across increasing levels of obesity measurements were reported[27]. In addition, most drugs used in the treatment of diabetes tend to increase body weight, which leads to an increase in insulin resistance and consequently a decline in glycemic control[28]. It is accepted that weight loss should be a primary recommendation for overweight type 2 diabetic patients and each 1 kg of weight loss in the patient is associated with 3-4 months prolonged survival[29]. The authors found that taking glucomannan in the long-term can lower the BMI whereas taking the placebo did not. This may be another benefit of glucomannan in diabetic treatment. It has been suggested that at least 1240 mg daily dose of glucomannan as a single fiber supplement should be used to get a significant weight reduction in healthy subjects[30].

In conclusion, pre-prandial glucomannan ingestion attenuated the postchallenge glucose elevation. Long-term use of glucomannan reduces postprandial glucose increment and impedes an elevation of serum fructosamine and LDL-C. The authors suggest that it would be beneficial to type 2 diabetes mellitus if glucomannan supplement is included into the diabetic treatment regimen.

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การตอบสนองของระดับน้ำตาลและไขมันต่อกลูโคแมนแนนจากหัวบุกในคนไทยที่มีโรคเบาหวานชนิดที่สอง

สุพรพิมพ์ เจียสกุล, สมเกียรติ แสงอุไร, วรรณี นิธิยานันท์, วันทนีย์ เกรียงสินยศ, สุวัฒณี คุปติวุฒิ, ทัสมา หะริณธนาวุฒิ

วัตถุประสงค์: ศึกษาประโยชน์ของผงบุกต่อการควบคุมระดับน้ำตาลและไขมันในผู้ป่วยเบาหวานชนิดที่สอง

วัสดุและวิธีการ: แผนการทดลองแรกให้อาสาสมัครผู้ป่วยเบาหวานชนิดที่สองชาย 10 คน และหญิง 10 คน กินผงบุก หรือ ยาหลอก 1 กรัม 30 นาทีก่อนดื่มกลูโคส 75 กรัม เก็บตัวอย่างเลือดก่อนกินผงบุก/ยาหลอก และที่ 1-2 ชั่วโมง หลังดื่มกลูโคสเพื่อวัดระดับกลูโคส และอินซูลิน แผนการทดลองที่สอง อาสาสมัครกินผงบุก/ยาหลอกก่อนอาหาร 3 มื้อ (3 กรัมต่อวัน) นาน 4 สัปดาห์ ทดสอบความทนน้ำตาลก่อนเริ่มและเมื่อสิ้นสุด 4 สัปดาห์ เว้นช่วงระหว่างการทดลองที่ สลับระหว่างผงบุกและยาหลอก 2 สัปดาห์ วัดระดับกลูโคส และอินซูลินในเลือดที่เก็บทุกตัวอย่าง วัดระดับฟรุกโทซามีน และไขมันในตัวอย่างเลือดที่เก็บก่อนการทดสอบความทนน้ำตาล

ผลการศึกษา: การกินผงบุก 1 กรัมก่อนทดสอบความทนน้ำตาล ทำให้ระดับน้ำตาลและอินซูลินในเลือดระหว่าง 1-2 ชั่วโมง เพิ่มน้อยกว่าเมื่อกินยาหลอก โดยไม่พบความแตกต่างที่มีนัยสำคัญของอินซูลิน การกินผงบุกก่อนอาหาร ทุกมื้อนาน 4 สัปดาห์ทำให้น้ำตาลในเลือดที่เพิ่มระหว่าง 120 นาทีของการทดสอบความทนน้ำตาลลดลงอย่างมี นัยสำคัญ เมื่อเทียบกับก่อนได้รับผงบุก ระดับ LDL-C หลังได้รับผงบุกต่ำกว่าหลังได้รับยาหลอก ผู้ป่วยหญิงที่ได้รับ ผงบุกมีค่า HOMA-insulin resistance index ลดลง ผู้ป่วยทั้งกลุ่มที่ได้รับผงบุกมีดัชนีมวลกายลดลง ไม่พบการ เปลี่ยนแปลงของอินซูลิน, ฟรุกโทซามีน และไขมันที่ศึกษาชนิดอื่นระหว่างก่อนและหลังได้รับผงบุก หรือ ระหว่างได้รับ ผงบุกและยาหลอก

สรุป: การกินผงบุกก่อนอาหารลดการดูดซึมน้ำตาลโดยไม่มีผลเปลี่ยนแปลงอินซูลินในเลือด ผู้ป่วยเบาหวานที่กินผงบุก ก่อนอาหารทุกมื้อ เมื่อครบ 4 สัปดาห์พบระดับน้ำตาลหลังอาหารเพิ่มน้อยลง LDL-C ไม่เพิ่มขึ้น ดัชนีมวลกายลดลง และในผู้ป่วยหญิงภาวะดื้ออินซูลินลดลงด้วย การกินผงบุกเสริมกับการรักษาอื่น ๆ อาจมีประโยชน์ต่อการควบคุมระดับ น้ำตาลและไขมันในผู้ป่วยเบาหวานชนิดที่สอง