The Comparative Study of Diabetic Specific Formula and Standard Formula on Postprandial Plasma Glucose Control in Type 2 DM Patients

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Objective: *To compare the postprandial plasma glucose level after diabetic specific formula (DSF) and standard formula (SF) administration in type 2 diabetic patients.*

Material and Method: *Thirty type 2 diabetic patients were included in the present randomized, controlled, double-blind, cross-over study. Subjects received DSF and isocaloric SF as a bolus administration of 400 mL while continuing their anti-diabetic medications. Venous blood samples were collected and analyzed to assess plasma glucose levels at pre- and at 30, 60, 90, 120, and 180 min post-administration of the formulas.*

Results: *Postprandial glucose profiles were significantly lower with DSF compared to SF administration determined as a mean glucose concentration at 2-hour post-administration. The glucose area under the curve (AUC) after DSF consumption was 33% lower than the AUC after SF consumption, $p<0.001$.*

Conclusion: *Use of DSF resulted in a significantly lower postprandial rise in plasma glucose concentrations than using SF. It should be the preferred option in diabetic patients who need nutritional support.*

Keyword: *Diabetic specific formula, Postprandial glucose, Type 2 diabetes*

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Prevalence of type 2 diabetes mellitus (T2DM) is increasing due to an increase in the global population, longer life expectancy, lack of exercise, overweight, or obesity. The prevalence of diabetes mellitus in Thailand in people over 35 years of age was 9.6 percent in 2003 and half of all these were newly diagnosed patients[1]. The fourth National Health Examination survey conducted between 2008 and 2009 reported that the prevalence of diabetes mellitus in Thailand in people over 15 years old was 6.9% (7.7% in women and 6% in men)[2]. The management of diabetes mellitus should be a combination of dietary modification, increased physical activity and/or use of hypoglycemic agents. Some diabetic patients and many diabetic hospitalized patients cannot eat enough or cannot tolerate oral diets; therefore, they require nutritional support. In addition, an increasing number of patients receive long-term home enteral tube feeding, including those with diabetes[3]. Several diabetic specific formulas (DSF) were developed for diabetic patients who needed nutritional support either as oral or enteral nutrition. These formulas contain a lower proportion of carbohydrate and a specific nutrient composition for better postprandial glucose control. Such nutrients include fructose, fiber, monounsaturated fatty acids (MUFAs), soy protein, and antioxidants. There is evidence from a systematic review showing that the use of diabetic specific formulas is associated with improvement in short and long term glycemic control compared to the standard formulas[4] and should be considered using in diabetic and hospitalized hyperglycemic patients.

The present study was designed to evaluate and compare the postprandial plasma glucose level after DSF administration to the isocaloric standard formula (SF) administration in T2DM patients.

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Material and Method

Study formulas

The standard formula (SF: Blendera, Thai Otsuka) is the formula for general population who cannot have adequate oral nutrition. Normally, it is used via enteral feeding. The diabetic specific formula (DSF: Gen-DM, Thai Otsuka) using in the present study was developed from Gen Formula, which is a

nutritional product that is proven to reduce LDL-C level significantly after four to eight weeks consumption[5]. The formula was modified by substituting sucrose with fructose and adding fiber to control both cholesterol and postprandial glucose levels. The carbohydrate composition in this formula is much higher than other DSFs, however, the macronutrient composition is the same as the nutritional recommendations for diabetic patients of the American Diabetes Association[6] and other organizations[7-9].

The compositions of the SF and DSF formulas are compared in Table 1. The distribution of macronutrients in both formulas are the same, namely carbohydrate 55%, protein 15% and fat 30%. However, the DSF substitutes sucrose for combination of fructose, polydextrose and fructo-oligosaccharide (FOS) to decrease postprandial glucose absorption.

Subjects

The subjects were recruited from the out-patients diabetic clinic of the Department of Internal Medicine, Faculty of Medicine, Chiang Mai University. The inclusion criteria were 1) men or women aged 18 years or older, 2) diagnosis of T2DM at least six months, 3) use stable dose of hypoglycemic agents at least three months before enrolment, and 4) no documented hypoglycemia or hypoglycemic symptoms within three months prior to enrolment. All subjects were required to sign informed consent form. Subjects were excluded if they had any of the following criteria, kidney dysfunction (serum creatinine more than or equal to 2.0 mg/dL), liver dysfunction (aspartate aminotransferase (AST) or alanine aminotransferase (ALT) more than or equal to three times upper limit of normal), diseases related to metabolism such as thyroid dysfunction, gastrointestinal dysfunction such as short bowel syndrome or any colectomy, allergic to any components of the study formulas, could not tolerate the taste of the study formula, enrolled in other clinical study within three months before enrolment, pregnancy, or lactating. The study protocol was approved by the Research Ethics Committees of the Faculty of Medicine, Chiang Mai University and followed the recommendations of the Declaration of Helsinki. All participants signed written informed consents prior to study screening.

Study design

This was a single center, prospective, randomized, double-blind, cross-over study. After informed consents were obtained, history taking and physical examination were performed to ensure that the patients met all the inclusion criteria and had no exclusion criteria. At the screening day, 10-mL venous blood samples were obtained for laboratory determination of complete blood count and chemistries including blood urea nitrogen (BUN), creatinine, glucose, serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), alkaline phosphatase, total cholesterol, triglyceride, HDL-C, LDL-C and thyroid stimulating hormone (TSH), following an overnight fasting at least 8 hours. Urine was also collected for standard urinalysis. The eligible subjects were randomized with an equal probability of receiving either 400-mL (Study formulas 1 sachet provided carbohydrate 55 grams mixed with water up to 400 mL) with caloric density 1 kcal/mL, DSF first or isocaloric SF orally first, on day one. The other formula would be given at the next visit, on day eight, while continuing their anti-diabetic medications. The study formulas were freshly prepared by mixing

Table 1. The composition of 2 dietary formulas used in the present study

	Standard formula (SF)	Diabetic specific formula (DSF)
% energy from protein	15	15
Protein sources	92% soy protein isolate 8% sodium caseinate	50% soy protein isolate 50% sodium caseinate
% energy from carbohydrate	55	55
Carbohydrate sources	68% maltodextrin 32% sucrose	70% maltodextrin 15% fructose 8% polydextrose 7% fructo-oligosaccharide
% energy from lipids	30	30
Lipids sources	79% rice bran oil 21% MCT	100% soy oil

the dry powder with sterile water. Both the subjects and the study nurses who prepare the formula were blinded to the type of the formula given in any particular day. The subjects had to drink the formula completely within 10 minutes. Immediately before and 30, 60, 90, 120, and 180 minutes after consumption, venous blood samples were collected to determine fasting plasma glucose and postprandial plasma glucose responses. Subjects were on normal saline solution (NSS) lock for their advantage.

The presence/absence of adverse events was evaluated during the study and included symptoms of vomiting, diarrhea, constipation, and abdominal distension.

Statistical analysis

Results were expressed as means ± standard deviation (SD) if data were normally distributed (Kolmogorov-Smirnov test), or as medians [25-75% interquartile range] if not normally distributed. Categorical variables were expressed as proportion (percentages). For comparisons of all the continuous parameters in each patient between baseline and at 30, 60, 90, 120 and 180 minutes statistical significance was tested by using repeated measure ANOVA (normally distributed data) or by the Friedman test (non-normally distributed data). A within-subject comparison of post-treatment (180 minute) and baseline values were analyzed by using a paired Student t-test (normally distributed data) or Wilcoxon signed rank test (non-normally distributed data). For comparison of both formulas, Student t-test or a Mann-Whitney U-test was used as appropriate. Categorical variables were compared by the Fisher's exact test. All statistical tests were two-tailed and statistical significance was set at a *p*-value of less than 0.05. Statistical analysis was performed using Stata program, version 10 for Windows.

Results

Thirty-eight subjects were screened and eight were excluded (six subjects had abnormal thyroid function test and two were unable to be followed-up after screening). Thirty subjects (23 female and 7 male) aged between 39 and 85 (mean 60.93±11.71) years with a body mass index (BMI) of 20.3 to 54.8 kg/m^2 (median 26.4) fulfilled the entry criteria and were recruited to the study (Table 2). Every subject had been on oral hypoglycemic agents and one had been on insulin injection for glycemic control. Plasma glucose levels showed no significant difference between the two formulas administration at baseline and at 30 minutes. However, 2-hour plasma glucose levels increased significantly less post-DSF than post-SF consumption (166.69±40.88 vs. 195.83±52.58 mg/dL, $p<0.001$). There was a significant difference in plasma glucose levels between the two groups at 60, 90, and 180 minutes after formula administration as well. Plasma glucose levels at pre- and post-formula administration are shown in Table 3. The glucose area under the curve (AUC) over 3 hours after the DSF consumption was 7,569.6 mg.min/dL compared to after SF consumption of 11,187.3 mg.min/dL. The AUC ratio was 0.6766, which is 33% lower in the DSF when compared with the SF formula, $p<0.001$. The maximal changes in postprandial plasma glucose (at 90 min in both formulas) relative to baseline plasma glucose were significantly lower in the DSF group when compared with the SF group (191.99±43.44 vs. 218.73±48.36, $p<0.001$). At postprandial 180 minutes, plasma glucose returned to baseline levels in the DSF group, but was still higher than baseline in SF group. The AUC data were shown in Fig. 1.

No subjects had any gastrointestinal symptoms with both formula consumption and

Table 2. Baseline characteristics of the included subjects

Characteristic (n = 30)	Mean ± SD	Median (Q1-Q3)
Age (years)	60.93±11.71	58 (53, 70)
Body weight (kg)	67.76±15.83	63.55 (58.8, 74.6)
Height (cm)	156.27±7.05	156 (152, 160)
Gender: female	23 (76.7%)	
SBP (mm.Hg)	134.87±14.67	133 (127, 144)
DBP (mm.Hg)	76.50±11.93	74.5 (68, 84)
Pulse rate (beats/min)	76.53±11.44	76 (69, 83)

SBP = systolic blood pressure; DBP = diastolic blood pressure

Table 3. Plasma glucose levels at pre- and post-formula administration (mean ± SD)

Time	Plasma glucose levels (mg/dL)		*p*-value between groups
	DSF	SF	
Baseline	119.03±36.97	117.87±29.02	0.796
30 min	155.93±43.72	157.77±38.66	0.747
60 min	186.70±43.55	205.51±44.33	0.001**
90 min	191.99±43.44	218.73±48.36	<0.001**
120 min	166.69±40.88	195.83±52.58	<0.001**
180 min	122.33±48.56	145.44±53.73	<0.001**

** Significant difference

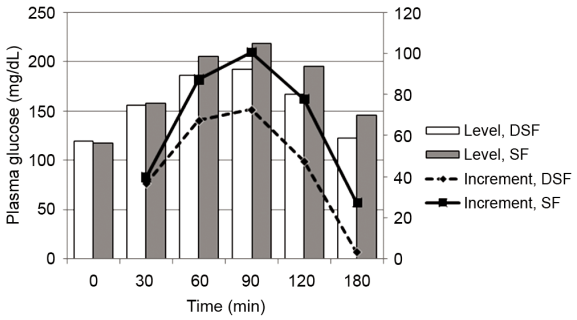

Fig. 1 Incremental area under the plasma glucose response curve.

nobody withdrew from the study due to adverse events or formula intolerance. No one experienced hypoglycemic symptoms during the study.

Discussion

Nutrition is the cornerstone of diabetes management and nutritional guidelines have been established by the European Association for the Study of Diabetes[6], the American Diabetes Association[7], the Canadian Diabetes Association[8] and the Nutrition Subcommittee of Diabetes UK[9]. Although country specific guidelines differ slightly in the optimal macronutrient composition, their primary goal is to achieve and maintain near-normal postprandial and fasting blood glucose levels to prevent or delay complications[10].

Standard medical care in diabetes 2014 stated that current evidence suggests that there is no ideal percentage of calories from carbohydrate, protein and fat for people with diabetes. Macronutrient distribution should be based on individualized assessment of current eating patterns, preferences, and metabolic goals. A variety of eating patterns (combinations of different foods or food groups) are acceptable for the management of diabetes[11]. However, the Associations recommend substituting low-glycemic load foods for higher-glycemic load foods to modestly improve glycemic control. In people with T2DM, the Mediterranean-style, MUFA-rich eating pattern may benefit glycemic control and CVD risk factors and it can be recommended as an effective alternative to a lower-fat, high carbohydrate diets[11].

The results of the present study illustrated that type of carbohydrate was important in improving glycemic control. Compared to SF, DSF, which had the same proportion of carbohydrate but contains fructose instead of sucrose and added fibers, was associated with a 33% reduction in plasma peak and AUC of glucose response. The present study demonstrated that change in carbohydrate content in the nutrition formula also helped improving glycemic response in diabetic patients. In other studies, the DSF which demonstrated reduced postprandial glycemic response after consumption was high-MUFA, low-carbohydrate formula[12-14]. Diabetic specific formulas (DSF) is typically higher in fat (40-50% of energy, with a high proportion of MUFAs), with lower carbohydrate content (35-40% of energy) and up to 15% of energy from fructose and added soluble fibers. These nutrients could improve glycemic management by delaying gastric emptying (fat and fiber), delaying intestinal absorption of carbohydrate (soluble fiber), and producing lower glycemic responses (fructose)[4]. Voss et al[10] demonstrated that DSF was associated with reduction in plasma glucose response (P-AUC) of nearly twice that of the standard formula.

In contrast to most diabetic specific formulas, this formula contains 30% of energy from fat (soy oil), 15% protein (50% soy protein isolate and 50% caseinate) and 55% carbohydrate. However, the type of carbohydrate has been changed from 32% sucrose to 15% fructose, 8% polydextrose and 7% fructo-oligosaccharide. Fructose is included to facilitate glucose clearance by the liver through the formation of fructose-1-phosphate, a fructose intermediate metabolite that reduces the inhibition of glucokinase, thus blunting the postprandial rise in plasma glucose[15,16]. The presence of soluble fibers in the DSF (compared with no fiber in the SF) may also play a role in the decreased glucose response. High-soluble fiber-containing foods improve glycemic control at least in part of delayed glucose absorption[17]. Animal studies had demonstrated that fermentable fibers, such as short-chain fructo-oligosaccharides, raised plasma levels of GLP-1[18,19]. Short-chain fatty acids, the byproducts of carbohydrate fermentation, may also play a role in the secretion of GLP-1[10].

Bouma et al[20] noted that postprandial plasma glucose was more related to HbA1C than preprandial plasma glucose. Evidence suggests that acute hyperglycemia may increase cardiovascular risk by variety of mechanisms, leading to the production of oxidative stress[21]. Markers of cardiovascular risk had been associated with elevated postprandial glucose levels. Postprandial glucose had been linked to inflammation and endothelial dysfunction[22] and adhesion molecules[23]. The Risk Factors in Impaired

Glucose Tolerance for Atherosclerosis and Diabetes (RIAD) study demonstrated that post-challenge hyperglycemia related more strongly to carotid intima-media thickness than did fasting hyperglycemia[24]. Furthermore, Ning et al[25] noted that postprandial plasma glucose was related to insulin resistance and increased mortality due to cardiovascular events.

Utarwuthipong et al[26] did the study to compare the effects of a diet containing soybean oil (SBO), rice bran oil (RBO), palm oil (PO) or a RBO/PO (3:1) mixture (20% of the energy intake of the diet) in 16 hypercholesterolemic women. They demonstrated that total cholesterol and LDL-C levels were significantly reduced during SBO, RBO and RBO/PO consumption, while HDL-C was significantly decreased by SBO consumption. There was a significant reduction in sdLDL-C only after SBO consumption, whereas it was significantly increased following PO consumption. The sdLDL-C oxidation lag time was significantly increased during PO, RBO/PO, and RBO consumption, but significantly reduced following SBO. Therefore, the consumption of this DSF in the long term might reduce total cholesterol, LDL-C, sdLDL-C, and sdLDL-C oxidation lag time and might further reduce the risk of atherosclerosis. Soy oil has a low component of saturated fat, a moderate amount of monounsaturated fat and a high composition of polyunsaturated fat. The composition of those three fatty acids in soy oil is 1.0: 2.1: 4.6, respectively. Soy oil also has an adequate amount of linoleic acid and alpha-linolenic acid, which are both essential fatty acids[5].

A potential limitation of the present study was that it focused on short-term postprandial response. The present study evaluated plasma glucose over a three hours postprandial period, which was a short-term comparison of glycemic responses between two nutritional formulas. However, the results can be an indicator of longer-term glucose control.

Conclusion

The present study showed that the use of diabetic specific formula (containing standard proportions of carbohydrate with some fructose and fibers) was associated with an improvement in glycemic control compared to the use of standard formulas. A long term study is warranted to confirm the benefits of improving glycemic control, lipid control and/or reducing macrovascular or micro-vascular complications in type 2 diabetic patients who were maintained on diabetic specific formula.

What is already known on this topic?

Compared with standard formula, diabetic specific formulas (DSFs) demonstrate a lower postprandial glycemic response after consumption. DSFs are typically higher in fat (40-50% of energy, with a high proportion of MUFAs), with a lower carbohydrate content (35-40% of energy) and up to 15% of energy from fructose and added soluble fibers. These formulas could improve glycemic management by delaying gastric emptying, delaying intestinal absorption of carbohydrate, and producing lower glycemic responses. Voss et al demonstrated that DSF was associated with a reduction in plasma glucose response (P-AUC) of nearly twice that of the standard formula.

What this study adds?

The carbohydrate composition of this DSF is much higher than other DSFs. However, the macronutrient composition is the same as the nutritional recommendations for diabetic patients of the American Diabetes Association and other organizations. The compositions of the formula are carbohydrate 55%, protein 15%, and fat 30%. However, the DSF substitutes sucrose for a combination of fructose, polydextrose, and fructo-oligosaccharide (FOS) for decreasing postprandial glucose absorption. The results of the present study illustrate that changes in carbohydrate content in the nutrition formula but with the same amount of carbohydrate also helps to improve glycemic response by reduction in two-hour postprandial glucose and AUC of glucose response in diabetic patients.

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Potential conflicts of interest

None.

References

1. Aekplakorn W, Stolk RP, Neal B, Suriyawongpaisal P, Chongsuvivatwong V, Cheepudomwit S, et al. The prevalence and management of diabetes in Thai adults: the international collaborative study

of cardiovascular disease in Asia. Diabetes Care 2003; 26: 2758-63.
2. Aekplakorn W, editor. The report of Thailand population health examination survey IV, Thailand 2008-9. Nonthaburi: The Graphico Systems; 2009. [in Thai]
3. Jones BM, Stratton RJ, Holden C, Russell C, Glencorse G, Micklewright A. Trends in artificial nutritional support in the UK 2000-2003: Annual report of the British Artificial Nutrition Survey (BANS). Redditch, U.K.: BAPEN; 2005.
4. Elia M, Ceriello A, Laube H, Sinclair AJ, Engfer M, Stratton RJ. Enteral nutritional support and use of diabetes-specific formulas for patients with diabetes: a systematic review and meta-analysis. Diabetes Care 2005; 28: 2267-79.
5. Tanphaichitra V, Stitchantrakul W, Leelahagul P, Pakpeankitvatana R. The results of Gen-formula supplement on plasma lipid levels in patients with hypercholesterolemia. Clin Nutr 1991; 4: 77-97. [in Thai]
6. Diabetes and Nutrition Study Group of the European Association for the Study of Diabetes. Recommendations for the nutritional management of patients with diabetes mellitus. Eur J Clin Nutr 2000; 54: 353-5.
7. American Diabetes Association. Nutrition Recommendations and interventions for diabetes: a position statement of the American Diabetes Association. Diabetes Care 2007; 30 (Suppl 1): S48-65.
8. Canadian Diabetes Association Clinical Practice Guidelines Expert Committee, Canadian Diabetes Association 2003 clinical practice guidelines for the prevention and management of diabetes in Canada. Can J Diabetes 2003; 27 (Suppl 2): S1-140.
9. Connor H, Annan F, Bunn E, Frost G, McGough N, Sarwar T, et al. The implementation of nutritional advice for people with diabetes. Diabet Med 2003; 20: 786-807.
10. Voss AC, Maki KC, Garvey WT, Hustead DS, Alish C, Fix B, et al. Effect of two carbohydrate-modified tube-feeding formulas on metabolic responses in patients with type 2 diabetes. Nutrition 2008; 24: 990-7.
11. American Diabetes Association. Executive summary: standards of medical care in diabetes-2014. Diabetes Care 2014; 37 (Suppl 1): S5-13.
12. Hofman Z, van Drunen JD, de Later C, Kuipers H. The effect of different nutritional feeds on the postprandial glucose response in healthy volunteers and patients with type II diabetes. Eur J Clin Nutr 2004; 58: 1553-6.
13. Ceriello A, Bortolotti N, Motz E, Pieri C, Marra M, Tonutti L, et al. Meal-induced oxidative stress and low-density lipoprotein oxidation in diabetes: the possible role of hyperglycemia. Metabolism 1999; 48: 1503-8.
14. Sanz-Paris A, Calvo L, Guallard A, Salazar I, Albero R. High-fat versus high-carbohydrate enteral formulae: effect on blood glucose, C-peptide, and ketones in patients with type 2 diabetes treated with insulin or sulfonylurea. Nutrition 1998; 14: 840-5.
15. Nuttall FQ, Gannon MC, Burmeister LA, Lane JT, Pyzdrowski KL. The metabolic response to various doses of fructose in type II diabetic subjects. Metabolism 1992; 41: 510-7.
16. Wolf BW, Humphrey PM, Hadley CW, Maharry KS, Garleb KA, Firkins JL. Supplemental fructose attenuates postprandial glycemia in Zucker fatty fa/fa rats. J Nutr 2002; 132: 1219-23.
17. Mann J. Dietary fibre and diabetes revisited. Eur J Clin Nutr 2001; 55: 919-21.
18. Cani PD, Neyrinck AM, Maton N, Delzenne NM. Oligofructose promotes satiety in rats fed a high-fat diet: involvement of glucagon-like Peptide-1. Obes Res 2005; 13: 1000-7.
19. Delzenne NM, Cani PD, Daubioul C, Neyrinck AM. Impact of inulin and oligofructose on gastrointestinal peptides. Br J Nutr 2005; 93 (Suppl 1): S157-61.
20. Bouma M, Dekker JH, de Sonnaville JJ, van der Does FE, de Vries H, Kriegsman DM, et al. How valid is fasting plasma glucose as a parameter of glycemic control in non-insulin-using patients with type 2 diabetes? Diabetes Care 1999; 22: 904-7.
21. Ceriello A. Cardiovascular effects of acute hyperglycaemia: pathophysiological underpinnings. Diab Vasc Dis Res 2008; 5: 260-8.
22. Monnier L, Mas E, Ginet C, Michel F, Villon L, Cristol JP, et al. Activation of oxidative stress by acute glucose fluctuations compared with sustained chronic hyperglycemia in patients with type 2 diabetes. JAMA 2006; 295: 1681-7.
23. Ceriello A, Quagliaro L, Piconi L, Assaloni R, Da Ros R, Maier A, et al. Effect of postprandial hypertriglyceridemia and hyperglycemia on circulating adhesion molecules and oxidative stress generation and the possible role of simvastatin treatment. Diabetes 2004; 53: 701-10.

24. Hanefeld M, Koehler C, Henkel E, Fuecker K, Schaper F, Temelkova-Kurktschiev T. Post-challenge hyperglycaemia relates more strongly than fasting hyperglycaemia with carotid intima-media thickness: the RIAD Study. Risk factors in impaired glucose tolerance for atherosclerosis and diabetes. Diabet Med 2000; 17: 835-40.
25. Ning F, Tuomilehto J, Pyorala K, Onat A, Soderberg S, Qiao Q. Cardiovascular disease mortality in Europeans in relation to fasting and 2-h plasma glucose levels within a normoglycemic range. Diabetes Care 2010; 33: 2211-6.
26. Utarwuthipong T, Komindr S, Pakpeankitvatana V, Songchitsomboon S, Thongmuang N. Small dense low-density lipoprotein concentration and oxidative susceptibility changes after consumption of soybean oil, rice bran oil, palm oil and mixed rice bran/palm oil in hypercholesterolaemic women. J Int Med Res 2009; 37: 96-104.

การศึกษาเปรียบเทียบระดับน้ำตาลในเลือดหลังบริโภคอาหารเสริมทางการแพทย์สูตรเบาหวาน และอาหารเสริมทางการแพทย์สูตรมาตรฐานในผู้ป่วยเบาหวานชนิดที่ 2

ศุภวรรณ บูรณพิร, สงวนศักดิ์ เสียงเรืองแสง, วิภาวรรณ ฉันทพานิชย์, ณัทวุฒิ เฮงจีระจรัส

วัตถุประสงค์: *เพื่อศึกษาและเปรียบเทียบระดับน้ำตาลหลังการบริโภคอาหารเสริมทางการแพทย์สำหรับผู้ป่วยเบาหวานกับอาหารเสริมสูตรมาตรฐานในผู้ป่วยเบาหวานชนิดที่ 2*

วัสดุและวิธีการ: *เป็นการศึกษาแบบสุ่ม แบบปกปิดทั้ง 2 ฝ่าย โดยให้ผู้ป่วยเบาหวานชนิดที่ 2 จำนวน 30 ราย ดื่มอาหารเสริมทางการแพทย์สูตรเบาหวานหรืออาหารเสริมทางการแพทย์สูตรมาตรฐานอย่างใดอย่างหนึ่ง 400 มิลลิลิตร ให้พลังงาน 400 กิโลแคลอรี่ แล้วดื่มอาหารอีกสูตรหนึ่งในอีก 7 วันต่อมา ในปริมาณพลังงานที่เท่ากัน และวัดระดับน้ำตาลในเลือดก่อนดื่มและหลังดื่มอาหารทั้ง 2 ชนิด ที่ 30, 60, 90, 120 และ 180 นาทีต่อมา โดยให้อาสาสมัครกินยาเบาหวานหรือฉีดยาตามเดิม*

ผลการศึกษา: *ระดับน้ำตาลหลังอาหารที่ 2 ชั่วโมง หลังการดื่มอาหารเสริมสูตรเบาหวานต่ำกว่าระดับน้ำตาลหลังอาหารที่ 2 ชั่วโมง หลังการดื่มอาหารเสริมสูตรมาตรฐานอย่างมีนัยสำคัญ ($p<0.001$) และระดับน้ำตาลที่ขึ้นตลอด 3 ชั่วโมง หลังการดื่มอาหารเสริมสูตรเบาหวานต่ำกว่าระดับน้ำตาลตลอด 3 ชั่วโมง หลังการดื่มอาหารเสริมสูตรมาตรฐาน 33% ($p<0.001$)*

สรุป: *การบริโภคอาหารเสริมทางการแพทย์สูตรเบาหวานจะมีผลเพิ่มระดับน้ำตาลหลังอาหารน้อยกว่าการบริโภคอาหารเสริมสูตรมาตรฐานอย่างมีนัยสำคัญ และควรเลือกอาหารเสริมทางการแพทย์สูตรเบาหวาน ในผู้ป่วยเบาหวานหรือผู้ที่มีระดับน้ำตาลในเลือดสูงในคนที่ต้องการอาหารเสริม*