

**GLYCEMIC INDEX OF KHANOM-TIAN (KT) IN TYPE 2
DIABETES AND IMPROVEMENT OF PLASMA GLUCOSE,
SERUM LIPIDS AND BLOOD VISCOSITY
AFTER MODIFICATION OF ITS COVERING FLOUR**

The seal of Mahidol University is a large, circular emblem in the background. It features a central blue circle with a golden Thai stupa (chedi) inside. The stupa has multiple tiers and a pointed top. Below the stupa are golden Thai script characters. The entire central emblem is surrounded by a wide golden ring containing Thai script. The outermost ring is a thin white border.

THANWARIN TANGSERMWONG

**A THESIS SUBMITTED IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR
THE DEGREE OF MASTER OF SCIENCE (NUTRITION)
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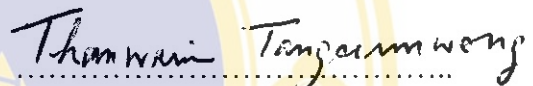
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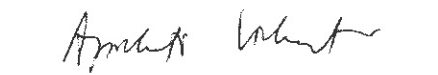
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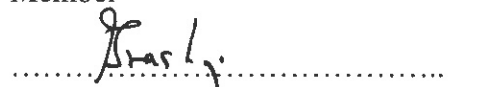

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GLYCEMIC INDEX OF KHANOM-TIAN (KT) IN TYPE 2 DIABETES AND IMPROVEMENT OF PLASMA GLUCOSE, SERUM LIPIDS AND BLOOD VISCOSITY AFTER MODIFICATION OF ITS COVERING FLOUR

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ABSTRACT

Desserts are almost always prohibited in diabetes. Too strict a limitation of food selection can lead to failure of patients' cooperation and diabetic control and may result in increased blood viscosity and compromised blood flow. This study wanted to develop new recipes of Khanom-Tian (KT), which produced lower plasma glucose and lipid responses, and had low glycemic indexes (GI), and observe the blood viscosity response to these KT recipes.

Study desserts consisted of a standard KT (stdKT) recipe and 4 modified KT recipes. All of the modified KT recipes contained the same amount of total carbohydrate but less sucrose content in the filling than the stdKT recipe. The 4 modified KT recipes consisted of modified stdKT (white glutinous rice flour cover), black glutinous rice flour KT, mungbean starch KT, and small sago (cassava-pearl) KT which contained the same amount of filling, and differed only in the covering flour. After a 10-h overnight fast, subjects ate one recipe of KT. Baseline and postprandial blood samples were collected to determine plasma glucose, serum lipids and blood viscosity. All subjects consumed 5 test desserts in a random order 4 weeks apart.

Area under plasma response curve over 5 h was largest after eating small sago KT recipe followed by stdKT recipe, black glutinous rice flour KT recipe, modified stdKT recipe and the smallest area was seen after mungbean starch KT recipe. Their glycemic indexes were 82, 72, 71, 61 and 57, respectively. The mean triglyceride levels at 5 h after ingesting all kinds of KT recipe were significantly increased from baseline. Although no significant rise of mean blood viscosity over the 5-h period was detected after eating each KT, whole blood viscosity of the whole group of subjects correlated well with serum triglyceride level ($r = 0.186$, $p < 0.05$) but not with plasma glucose level. The sensory evaluation test showed that modified stdKT had the highest score.

It was concluded that modification of KT covering flour could alter the glycemic response in type 2 diabetic. KT with mungbean starch cover gave the lowest GI whereas that with small sago (cassava pearl) gave the highest GI, proper selection of covering flour will be beneficial for health promotion, especially in diabetes.

KEY WORDS: GLYCEMIC INDEX / DESSERT / DIABETES / FLOUR

BLOOD VISCOSITY

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ดัชนีน้ำตาลของขนมเทียนในผู้ป่วยเบาหวานชนิดที่ 2 กับผลดีที่เกิดขึ้นต่อระดับน้ำตาล, ไขมัน และความหนืดของเลือด หลังจากดัดแปลงแป้งห่อด้วยแป้งชนิดต่างๆ (GLYCEMIC INDEX OF KHANOM-TIAN (KT) IN TYPE 2 DIABETES AND IMPROVEMENT OF PLASMA GLUCOSE, SERUM LIPIDS AND BLOOD VISCOSITY AFTER MODIFICATION OF ITS COVERING FLOUR)

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บทคัดย่อ

ผู้ป่วยเบาหวานมักจะถูกแพทย์ หรือนักกำหนดอาหารห้ามไม่ได้รับประทานขนมอยู่เสมอ การเพิ่มวจนเกินไป มักนำไปสู่ความล้มเหลวในการปฏิบัติตัว ซึ่งมีผลให้การควบคุมเบาหวาน คือ ระดับน้ำตาล ระดับไขมัน รวมไปถึงความหนืดของเลือดไม่ดีพอ การศึกษานี้ต้องการศึกษาถึงผลของการดัดแปลงสูตรของไส้ขนมเทียน และผลการดัดแปลงแป้งห่อด้วยแป้งชนิดต่างๆ ต่อการเปลี่ยนแปลงของระดับน้ำตาล ระดับไขมัน และความหนืดของเลือด ภายหลังจากรับประทานขนมเทียนดัดแปลงแต่ละชนิด รวมถึงการหาค่าดัชนีน้ำตาลของขนมเทียนเหล่านี้ ได้แก่ ขนมเทียนสูตรมาตรฐานที่มีอยู่ทั่วไป ขนมเทียนสูตรดัดแปลงไส้จากสูตรมาตรฐาน และสูตรดัดแปลงไส้และแป้งห่อโดยใช้แป้งชนิดอื่น จำนวน 5 สูตร แต่ละสูตรมีปริมาณคาร์โบไฮเดรต 50 กรัมเท่ากัน อาสาสมัครทุกคนรับประทานขนมเทียนทุกสูตรโดยวิธีสุ่ม และดื่มน้ำตาลกลูโคส 50 กรัม เพื่อเป็นตัวประเมินค่าดัชนีน้ำตาล โดยเริ่มจากการงดอาหาร 10 ชั่วโมง จากนั้นเจาะเลือด ที่เวลาเริ่มต้นก่อน และหลังจากรับประทานขนมเทียนแล้ว ที่เวลา 30, 60, 120, 180, 240 และ 300 นาที ผลการศึกษาพบว่าพื้นที่ใต้กราฟของการเปลี่ยนแปลงระดับน้ำตาลในเลือด ภายหลังรับประทานขนมเทียนสาकुเม็ดเล็กมีค่ามากที่สุด รองลงมาคือขนมเทียนสูตรมาตรฐาน ขนมเทียนสูตรแป้งข้าวเหนียวดำ ขนมเทียนสูตรดัดแปลงไส้จากสูตรมาตรฐาน และพื้นที่ใต้กราฟของการเปลี่ยนแปลงระดับน้ำตาลในเลือดน้อยที่สุดคือขนมเทียนสูตรแป้งถั่วเขียว โดยมีค่าดัชนีน้ำตาล คือ 82, 72, 71, 61 และ 57 ตามลำดับ ค่าเฉลี่ยของระดับไตรกลีเซอไรด์ในเลือดที่ 5 ชั่วโมง ภายหลังรับประทานขนมเทียนทุกสูตร เพิ่มขึ้นอย่างมีนัยสำคัญทางสถิติ ไม่พบความแตกต่างของการเปลี่ยนแปลงความหนืดของเลือดที่ 5 ชั่วโมง แต่พบว่าค่าระดับไตรกลีเซอไรด์ กับความหนืดของเลือดมีความสัมพันธ์เชิงบวก แต่ไม่สัมพันธ์กับระดับน้ำตาลในเลือด ผลการประเมินการยอมรับของขนมเทียน พบว่าขนมเทียนสูตรดัดแปลงไส้จากสูตรมาตรฐาน ได้รับการยอมรับสูงสุดในเรื่องลักษณะสัมผัสและความเหนียวของเปลือกแป้ง รสชาติของเปลือกและไส้ และภาพรวมทั้งหมด

CONTENTS

	PAGE
ACKNOWLEDGEMENT	iii
ABSTRACT	iv
LIST OF TABLES	ix
LIST OF FIGURES	x
LIST OF ABBREVIATIONS	xii
CHAPTER	
1 INTRODUCTION	
1.1 Background and Importance of the Problem	1
1.2 Research Hypothesis	3
1.3 Objective	3
2 LITERATURE REVIEWS	
2.1 Definition and Description of Diabetes Mellitus	4
2.2 Goals of Medical Nutrition Therapy for Diabetes	5
2.3 Recommended Glycemic Goals for Adults with Diabetes	5
2.4 Diabetic Complications	6
2.5 Carbohydrate and Diabetes	6
2.6 Glycemic Index (GI)	7
2.7 Starch	7
2.8 Gelatinization of Starch	8
2.9 Retrogradation of Starch	9
2.10 Starch and Glycemic Index	9
2.11 Metabolic Events Following Consumption of a High GI Meal	10
2.12 Glycemic Load and Type 2 Diabetes	11
2.13 Diabetes Mellitus and Dyslipidemia	12
2.14 Glycemic Index (GI) and Cardiovascular Disease (CVD)	13
2.15 Viscosity and Blood Viscosity	14
2.16 Blood Viscosity in Diabetes Mellitus and CVD	15

CONTENTS (cont)

	PAGE
3 MATERIALS AND METHODS	
3.1 Materials	16
3.1.1 Subjects	16
3.1.2 Dessert Preparation	16
3.1.3 Characteristics of Starch/Flour	17
3.2 Methods	22
3.2.1 Obtaining Approval	22
3.2.2 Experimental Design	22
3.3 The Sensory Evaluation Test	23
3.4 Anthropometric Measurement	23
3.4.1 Weight (Wt)	23
3.4.2 Height (Ht)	23
3.4.3 Body Mass Index (BMI)	23
3.4.4 Triceps Skinfold Thickness (TSF)	23
3.4.5 Mid Upper Arm Circumference (MUAC)	24
3.4.6 Upper Arm Muscle Circumference (UAMC)	24
3.4.7 Waist Circumference	24
3.4.8 Hip Circumference	25
3.4.9 Waist to Hip Circumference Ratio (WHR)	25
3.5 Biochemical Analysis	25
3.5.1 Determination of Glucose	25
3.5.2 Determination of Serum Triglyceride	26
3.5.3 Determination of Serum Total Cholesterol	27
3.5.4 Determination of High Density Lipoprotein Cholesterol (HDL-C)	28
3.5.5 Estimation of Low Density Lipoprotein Cholesterol (LDL-C)	28
3.6 Calculation of Glycemic Index (GI)	29
3.7 Measurement of Blood Viscosity	29
3.7.1 General Techniques in Viscometry	30

CONTENTS (cont)

	PAGE
3.8 Statistical Analysis	30
4 RESULTS	
4.1 Clinical Characteristics of Subjects	31
4.2 Plasma Glucose Levels	31
4.3 AUC and GI	39
4.4 Lipid Profile	39
4.4.1 Serum Triglyceride Levels	39
4.4.2 Serum Cholesterol Levels	39
4.4.3 Serum HDL-C Levels	39
4.4.4 Serum LDL-C Levels	40
4.5 Blood Viscosity	40
4.6 Blood Viscosity and Lipid Correlation	40
4.7 Sensory Evaluation Test	40
5 DISCUSSION	
5.1 Plasma Glucose Levels, AUC and GI	55
5.2 Different Amount of Sugar and Different Types of flour	56
5.3 Dessert and Sugar Content	58
5.4 The Benefit of Mungbean Starch	60
5.5 Lipid Profile	60
5.6 Blood Viscosity	61
6 CONCLUSION	
6.1 Outcome and Benefit	63
6.2 Recommendation for Future Research	64
REFERENCES	65
APPENDIX	73
BIOGRAPHY	105

LIST OF TABLES

		PAGE
TABLE		
MATERIALS AND METHODS		
3.1	Nutrient composition and energy distribution of each serving of KT	18
3.2	Time for blood withdrawal and testing	22
RESULTS		
4.1	Anthropometric measurements of the subjects at the first time	32
4.2	Clinical characteristics of the subjects before the study	34
4.3	Comparison of plasma glucose responses (Mean $\pm$ SD) between before and after ingestion of different recipes of KT	35
4.4	Comparison between percentage change of plasma glucose (Mean $\pm$ SD) after ingestion of different recipes of KT	37
4.5	Area under the glucose curve (AUC) (mean $\pm$ SD), glycemic index (GI) and glycemic rank after ingestion of different recipes of KT	41
4.6	Comparison of serum triglycerides responses before and after ingestion of different recipes of KT	43
4.7	Comparison of serum cholesterol responses before and after ingestion of different recipes of KT	45
4.8	Comparison of serum HDL-C responses before and after ingestion of different recipes of KT	47
4.9	Comparison of serum LDL-C responses before and after ingestion of different recipes of KT	49
4.10	Comparison of blood viscosity (measured at shear rate of 264 s^{-1} , 37°C) before and after ingestion of different recipes of KT	51
4.11	Taste preference of different recipes of KT by using the sensory evaluation scores (Mean $\pm$ SD)	54

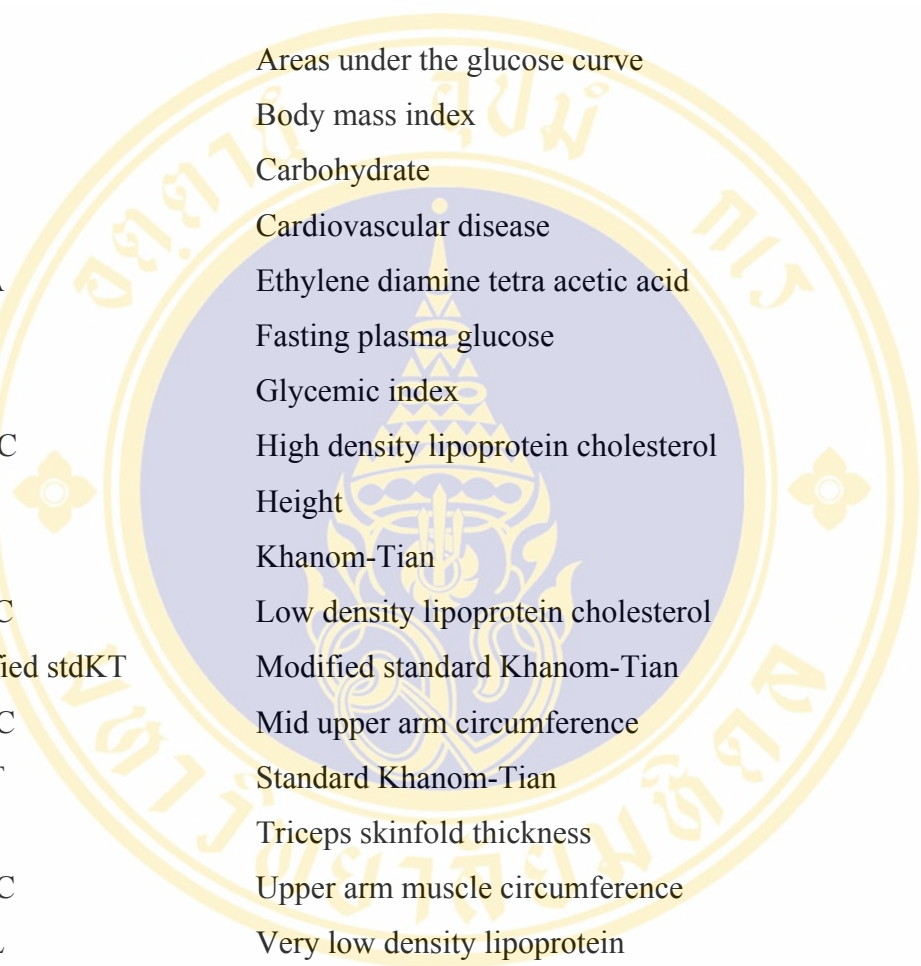
LIST OF FIGURES

FIGURE	PAGE
LITERATURE REVIEWS	
2.1 Amylose structure	8
2.2 Amylopectin structure	8
2.3 Potential mechanisms whereby high glycemic-load diets could increase risk of type 2 diabetes	11
MATERIALS AND METHODS	
3.1 Standard Khanom-Tian (stdKT)	19
3.2 Modified standard Khanom-Tian (modified stdKT)	19
3.3 Black glutinous rice flour Khanom-Tian	20
3.4 Mungbean starch Khanom-Tian	20
3.5 Small sago Khanom-Tian	21
3.6 Khanom-Tian	21
RESULTS	
4.1 Mean plasma glucose responses after ingestion of different recipes of KT	36
4.2 Percentage change of mean plasma glucose after ingestion of different recipes of KT	38
4.3 Mean areas under the glucose response curves after ingestion of different recipes of KT	42
4.4 Glycemic index of different recipes of KT	42
4.5 Mean triglyceride levels before and after ingestion of different recipes of KT	44
4.6 Mean percentage change of triglyceride after ingestion of different recipes of KT	44
4.7 Mean cholesterol levels before and after ingestion of different recipes of KT	46

LIST OF FIGURES (cont)

FIGURE	PAGE
4.8 Mean percentage change of cholesterol after ingestion of different recipes of KT	46
4.9 Mean HDL-C levels before and after ingestion of different recipes of KT	48
4.10 Mean percentage change of HDL-C after ingestion of different recipes of KT	48
4.11 Mean LDL-C levels before and after ingestion of different recipes of KT	50
4.12 Mean percentage change of LDL-C after ingestion of different recipes of KT	50
4.13 Mean blood viscosity (measured at shear rate of 264 s^{-1} , 37°C) before and after ingestion of different recipes of KT	52
4.14 Mean percentage change of blood viscosity (measured at shear rate of 264 s^{-1} , 37°C) at 60 and 300 min after ingestion of different recipes of KT	52
4.15 Correlation between serum triglyceride and blood viscosity in all subjects	53

LIST OF ABBREVIATIONS



AUC	Areas under the glucose curve
BMI	Body mass index
CHO	Carbohydrate
CVD	Cardiovascular disease
EDTA	Ethylene diamine tetra acetic acid
FPG	Fasting plasma glucose
GI	Glycemic index
HDL-C	High density lipoprotein cholesterol
Ht	Height
KT	Khanom-Tian
LDL-C	Low density lipoprotein cholesterol
Modified stdKT	Modified standard Khanom-Tian
MUAC	Mid upper arm circumference
StdKT	Standard Khanom-Tian
TSF	Triceps skinfold thickness
UAMC	Upper arm muscle circumference
VLDL	Very low density lipoprotein
WHR	Waist to hip circumference ratio
Wt	Weight

CHAPTER 1

INTRODUCTION

1.1 Background and Importance of the Problem

Diabetes is a global health problem. Although medical science is progressing and people are recognizing the danger of diabetes mellitus, incidence of diabetes is still rising. Diabetes is one such degenerative disease that has a profound influence on heart, renal, and neurological disease (1). Heart-disease death rates of people with diabetes are two to four times as high as those of adults without diabetes. Person with diabetes have five times the risk of having a major coronary event as do persons without diabetes. More than 40% of all persons undergoing dialysis for renal disease are people with diabetes mellitus.

A recent report from the International Collaborative Study of Cardiovascular Disease in Asia demonstrated the prevalence of diabetes in Thai adults age over 35 year old to be 9.6% (2.4 million people) which was formerly diagnosed 4.8% and have just diagnosed 4.8% (2). It is clear from numerous investigations that intensive lifestyle intervention through diet and exercise can improve insulin sensitivity. Diabetes Complications Control Trial (DCCT) research (3) confirmed that in addition to reduction of fasting glucose, the control of postprandial glucose can also reduce the risk of developing type 2 diabetes. Thus postprandial glucose control diet is necessary for these patients.

Fortunately, good care with diet, exercise, and medications to control blood sugar clearly prevents or delays the development of diabetes-related blindness, kidney failure, and nerve damage.

Traditional dietary advice given to diabetic patients includes eliminating simple sugars from the diet to avoid the excessive blood glucose level. Many people have difficulty following this recommendation. Because of their happiness of eating sweet or dessert, many patients tended to neglect the advice even though it is important to their health. Psychologically the patients prefer the word “allow” to “avoid” or “do

not”. It is hard to change patient’s food habit totally, hence modification of these desserts by reducing the sugar in the dessert can be acceptable by the patients.

In our pilot study by questionnaire, in type 2 diabetes of out patient department of Ramathibodi Hospital during October to November 2003. We found that no one denied of having eat some desserts during the week and 86.7% had inadequate knowledge of “Glycemic Index” which is important for slowing down the blood glucose rising.

In hospital, patients who are admitted with diabetic problems, the physician always orders the diabetic diet for treatment. Diabetic diet is one of the therapeutic diets, always restricted in the distribution of nutrients. In the serving dishes carbohydrate is always restricted, only rice, meat, vegetables, fat and fruits are allowed in the meal. There is no dessert, because most desserts are made from starch and sugars.

In the past, diabetic patients were prohibited from eating dietary sucrose because it was thought to raise blood glucose concentrations. Although neither the American Diabetes Association (ADA) nor the American Dietetic Association endorse the glycemic index as a tool for construction diet for individuals with diabetes (4), research published during the past decade makes an argument that the glycemic index of foods is one factor effecting the overall quality of the diet. At present, the ADA (5) recommended that carbohydrate and monounsaturated fat together should provide 60-70% of daily energy intake, with individual flexibility in the respective proportions, whereas intake of saturated fats is limited to less than 10% of energy intake. This new emphasis reflects greater awareness of the importance of responding to individual and cultural dietary preference and the need to address treatment of both hyperglycemia and dyslipidemia in diabetes mellitus.

Komindr et al (6, 7) proved that glutinous rice stimulated postprandial insulin and glucose response, in contrast mungbean noodle (vermicelli) has been designed as a low glycemic index diet slowed down their rising rate as well as their peaks. Several products on the market serve to aid diabetics in controlling their diets and blood glucose levels. For example: commercial formula, diet beverage, snack bar, health food and others. Most Thai desserts are made from several kinds of starch or flour

containing high sugar content. The idea that if we can modify the dessert by using good starch or flour and modified the sugars to achieve low glycemic responses, it should provide flexibility in diabetes self-management and therefore, a better diabetic control.

Thus, the modified dessert (Khanom-Tian) which has a low glycemic index may be a particularly important dessert for individuals with diabetes.

1.2 Research Hypothesis

Mungbean starch replacing glutinous rice flour in the making of Khanom-Tian cover could reduce blood sugar, lipid and viscosity.

1.3 Objective

1.3.1 General Objective

To develop new recipes of Khanom-Tian (KT) which had low glycemic index.

1.3.2 Specific Objectives

1. To compare the responses of standard Khanom-Tian recipe (stdKT) with the modified standard Khanom-Tian recipe (modified stdKT) on fasting and postprandial plasma glucose, lipid profile and blood viscosity.
2. To compare the responses of white glutinous rice flour, black glutinous rice flour, mungbean starch and small sago (cassava pearl) in several recipes of Khanom-Tian on fasting and postprandial plasma glucose, lipid profile and blood viscosity.
3. To find glycemic index of these modified Khanom-Tian recipes.
4. To evaluate the sensory acceptability of these Khanom-Tian recipes.
5. To determine the relation between plasma glucose, lipid profile and blood viscosity.

CHAPTER 2

LITERATURE REVIEWS

Type 2 diabetes develops (8) when the production of the insulin is insufficient to overcome the underlying abnormality of increased resistance to its action. The early stages of type 2 diabetes are characterized by overproduction of insulin. As the disease progress, process insulin levels may fall as a result of partial failure of the insulin producing β cells of the pancreas. Complications of type 2 diabetes include blindness, kidney failure, foot ulceration which may lead to gangrene and subsequent amputation and appreciably increased risk of infections, coronary heart disease and stroke.

The prevalence (9) of diabetes for all age-groups worldwide was estimated to be 2.8% in 2000 and to be rise to 4.4% by the year 2030. Hence, the total number of people with diabetes is projected to rise from 171 million in 2000 to 366 million in 2030. The urban population in developing countries is projected to double between 2000 and 2030.

2.1 Definition and Description of Diabetes Mellitus

Diabetes mellitus (10) is a group of metabolic diseases defined by elevated circulating glucose concentrations resulting from defects in insulin secretion, insulin action, or both. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels. The American Diabetes Association (11) set the criteria for the diagnosis of diabetes if fasting plasma glucose ≥ 126 mg/dL (7.0 mmol/L) or 2-h after meal plasma glucose ≥ 200 mg/dL (11.1 mmol/L).

Diabetes mellitus is a disease directly related to carbohydrate, lipid, and protein metabolism, nutrition has always an integral role in its management. The contemporary term used to describe the dietary prescriptions is medical nutrition therapy (MNT) (12).

2.2 Goals of Medical Nutrition Therapy for Diabetes (5)

Goals of medical nutrition therapy that apply to all persons with diabetes are as follows:

1. Attain and maintain optimal metabolic outcomes including
 - 1.1 Blood glucose levels in the normal range or as close to normal as is safely possible to prevent or reduce the risk for complications of diabetes.
 - 1.2 A lipid and lipoprotein profile that reduces the risk for macrovascular disease.
 - 1.3 Blood pressure levels that reduce the risk for vascular disease.
2. Prevent and treat the chronic complications of diabetes. Modify nutrient intake and lifestyle as appropriate for the prevention and treatment of obesity, dyslipidemia, cardiovascular disease, hypertension, and nephropathy.
3. Improve health through healthy food choices and physical activity.
4. Address individual nutritional needs taking into consideration personal and cultural preferences and lifestyle while respecting the individual's wishes and willingness to change.

2.3 Recommended Glycemic Goals for Adults with Diabetes (12)

Glycemic Control

A _{1C}	< 7.0	%	
Preprandial plasma glucose	90 – 130	mg/dL	(5.0 – 7.2 mmol/L)
Postprandial plasma glucose	180	mg/dL	(< 10.0 mmol/L)
Blood pressure	< 130/80	mmHg	

Lipids

LDL	< 100	mg/dL	(< 2.6 mmol/L)
Triglycerides	< 150	mg/dL	(< 1.7 mmol/L)
HDL	> 40	mg/dL	(> 1.1 mmol/L)

2.4 Diabetic Complications (10, 13)

Diabetes experts believe that the high levels of blood sugar above 240 mg/dL are unacceptable and dangerous. The optimal range should be between 80 to 120 mg/dL range. The length of time a person has diabetes also comes into play when looking at complications. For the most part, complications appear in people who have had diabetes for fifteen years or more, although certain short-term complications can appear (and disappear) at any time.

Long-term complications of diabetes include retinopathy; nephropathy leading to renal failure; peripheral neuropathy with risk of foot ulcers, amputations; and autonomic neuropathy causing gastrointestinal, genitourinary, cardiovascular symptoms and sexual dysfunction. Patients with diabetes have an increased incidence of atherosclerotic cardiovascular, peripheral arterial, cerebrovascular disease, hypertension and abnormalities of lipoprotein metabolism.

Diabetes is a chronic illness that requires continuing medical care and patient self-management education to prevent the complications. Glycemic control is fundamental to the management of diabetes. Prospective randomized clinical trials such as the Diabetes Control and Complications Trial (DCCT) (14) and the U.K. Prospective Diabetes Study (UKPDS) (15, 16) have shown the improved glycemic control is associated with sustained decreased rate of retinopathy, nephropathy, and neuropathy (17).

2.5 Carbohydrate and Diabetes

There was general agreement that (18) consumption of simple sugars should be limited and that diabetic patients should avoid table sugar and glucose in their diets. These were assumed to be more rapidly absorbed, thus leading to more hyperglycemia than complex carbohydrates.

Fructose is a naturally occurring carbohydrate found in fruit and honey and is also being increasingly used as a sweetener in processed foods and beverages (19). The glycemic and insulinemic response to fructose has been reported to be lower than that of glucose and sucrose. Also, fructose incorporated as a sweetener in complex foods has been reported to be associated with significantly lower serum glucose and insulin responses as compared to sucrose sweetened foods (20). Additionally, studies

conducted in healthy subjects noted that different amounts of fructose intake did not change HbA_{1C} levels (21), yet they significantly elevated plasma triglycerides (22).

Nutritional recommendations (23) for diabetes mellitus treatment were revised recently. The new guidelines do not specifically restrict intake of sugars, although general recommendations are made for including fiber, whole grains, vegetables, and fruits within dietary selections containing starches. For carbohydrates, the principle focus is on overall caloric amounts. In type 2 diabetes, in addition to a focus on caloric content of carbohydrate, consideration continues to be given to the role of the glycemic index as a determinant of postprandial hyperglycemia and overall metabolic control.

2.6 Glycemic Index (GI)

The GI (18) is defined as the blood glucose response to the consumption of a foodstuff relative to consumption of an equivalent amount of glucose or white bread. In general, carbohydrate foodstuffs can be classified as falling into low-, medium-, or high-glycemic index categories, (0-54, 55-69, 70 or higher), but considerable variability in response can result for many reasons.

Factors influence glycemic responses (5, 24) to foods, including the amount of carbohydrate, type of sugar (glucose, fructose, sucrose, lactose), nature of the starch (amylose, amylopectin, resistant starch), cooking and food processing (degree of starch gelatinization, particle size, cellular form), and food form, as well as other food components (fat and natural substances that slow digestion: lectins, phytates, tannins, and starch-protein and starch-lipid combinations).

2.7 Starch

Starches are (25, 26) polysaccharides that consist of repeating glucose units. Starch molecules have one of two molecular structures: a linear structure, known as amylose (α 1-4 linked); and a branched structure, known as amylopectin (α 1-6 branching links). Amylose and amylopectin associate through hydrogen bonding and arrange themselves radially in layers to form granules. All starches are made up of varying proportions of amylose and amylopectin. This ratio varies not only among the different types of starch, but among the many plant varieties within a type. The ratio of

amylose to amylopectin has the greatest influence on the physicochemical properties. Normal native starches consist of a mixture of 15-30% amylose and 70-85% amylopectin.

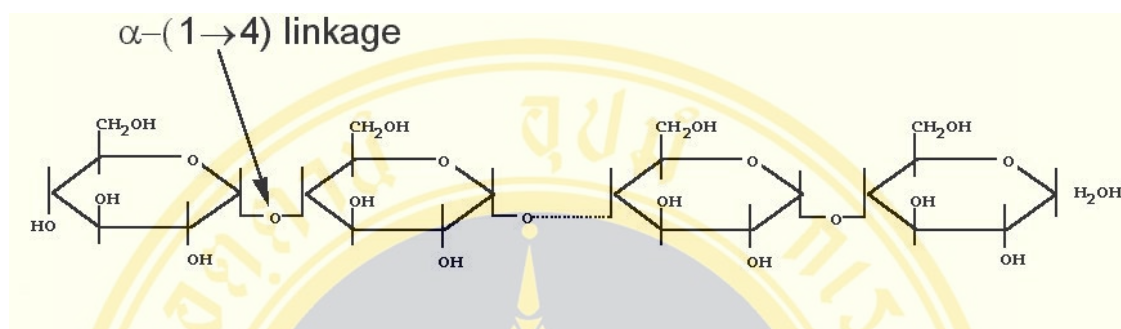


Figure 2.1 Amylose structure

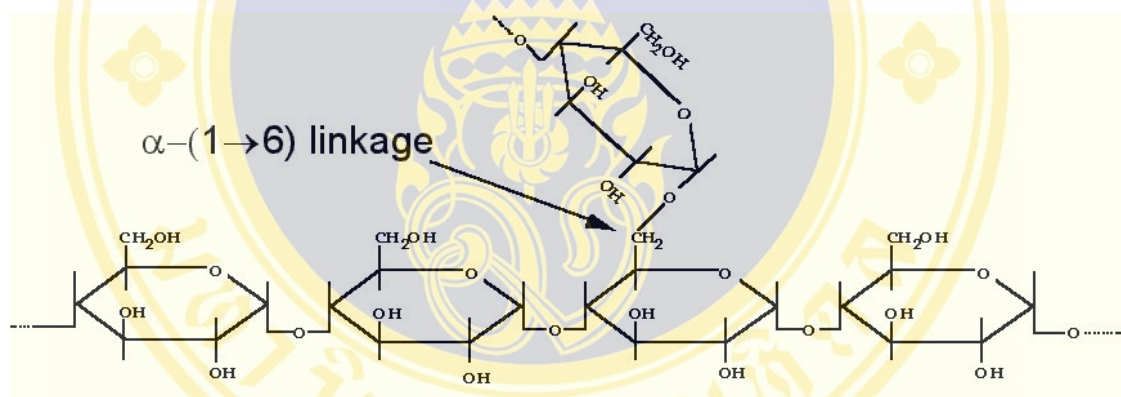


Figure 2.2 Amylopectin structure

2.8 Gelatinization of Starch (27)

When starch dispersed into water, the water penetrates into the starch granule from the outside inward until the granule is fully hydrated. With higher temperatures, molecule breaks down and loses its crystallinity. The granule is totally disrupted and at this stage the starch is readily attacked by enzymes. The whole process of disruption and swelling is known as gelatinization. Starch begins to gelatinize between 60 and 70°C, the exact temperature dependent on the specific starch. The largest granules, which are usually less compact, begin to swell first.

2.9 Retrogradation of Starch

When starch granules are fully gelatinized and dispersed, the starch becomes easily digestible. However, as the gel cools and ages, the polymers once more form a partially crystalline structure. Recrystallization or retrogradation depends on the formation of interchain hydrogen bonds and occurs most rapidly for the linear amylose. Retrogradation of amylopectin is limited by its branched structure and the polymers of retrograded amylopectin are less firmly bound than those of retrograded amylose.

2.10 Starch and Glycemic Index (28, 29)

Differences in the amylose content could explain much of the variation in the GI values because amylose is digested more slowly than is amylopectin starch. Amylose is a linear molecule with D-glucose units linked in an (α 1-4) fashion. Amylopectin has both (α 1-4) and (α 1-6) linkages and is thereby a branched structure. The higher the proportion of amylopectin, the higher the GI, because amylopectin, which is made up of branched-starch molecules, is more easily hydrolyzed in the gut than is the single-strand amylose.

A clinical study (30) with high-amylose corn starch demonstrating benefits to blood glucose and/or insulin levels. The amylose enhances the opportunity for retrogradation or crystallization of the glucose polymer, which limits digestibility, reducing the contribution to blood glucose levels.

Different sources of carbohydrate (31) and different types of starch affect the postprandial glucose and insulin responses. High amylose starches (32) are resistant to intestinal enzymes. Increase amylose consumption has been shown to decrease the concentration of glucose or insulin appearing in the blood after a meal. Many studies (33, 34) now have shown that food sources of carbohydrate vary greatly in their rate of absorption and effects on blood and insulin concentrations.

It was believed that (35) all starches were digested similarly and more slowly than simple carbohydrates. As result of the slower digestion or decreased stimulation of intestinal hormone release, glucose from complex carbohydrates was thought to reach the blood stream more slowly, resulting in smaller changes in blood glucose.

2.11 Metabolic Events Following Consumption of a High GI Meal (36)

The body has an obligatory requirement for glucose, approaching 200 g/d, determined largely by the metabolic demands of the brain. Should blood glucose concentration fall below 40 mg/dL (2.2 mmol/L), coma, seizure, or death may ensue. Conversely, blood glucose levels exceeding about 180 mg/dL (10.0 mmol/L), are associated with immediate (glycosuria and calorie loss) and long-term (renal failure, retinopathy, atherosclerosis) consequences. For these reasons, blood glucose concentration is tightly regulated by homeostatic regulatory systems. Hyperglycemia stimulates insulin secretion, promoting uptake of glucose by muscle and adipose tissue. Hypoglycemia elicits secretion of glucagon, epinephrine, cortisol, and growth hormone, counterregulatory hormones that antagonize insulin action and restore normoglycemia.

The rapid absorption of glucose following consumption of a high GI meal challenges these homeostatic mechanisms, complicating in effect the transition from the postprandial to the postabsorptive state. Within the first 2-h after a high GI meal, integrated incremental blood glucose concentration can be at least twice that after a low GI meal containing identical nutrients and energy. This relative hyperglycemia, acting in concert with elevated concentrations of the gut hormones glucagon-like peptide-1 and glucose dependent insulinotropic polypeptide, potently stimulates insulin release from pancreatic β cells and inhibits glucagon release from α cells. The resultant high insulin-to-glucagon ratio would tend to exaggerate the normal anabolic responses to eating, including uptake of nutrients by insulin-responsive tissue, stimulation of glycogenesis and lipogenesis, and suppression of gluconeogenesis and lipolysis. Between 2 and 4 hours after a high GI meal, nutrient absorption from the gastrointestinal tract declines, but the biological effects of the high insulin and low glucagon levels persist. Consequently, blood glucose concentration falls rapidly, often into the hypoglycemia range. The physiological significance of this hypoglycemia is demonstrated by a greater fall in glucose oxidation rate after consumption of a high compared with a low GI carbohydrate during this interval.

Free fatty acid the other major metabolic fuel, is more suppressed after a high glycemic index meal. Approximately 4 to 6 hours after a high GI meal, the low circulating concentrations of metabolic fuels trigger a counterregulatory hormone

response that restores euglycemia by stimulating glycogenolytic and gluconeogenic pathways and elevates free fatty acid concentration to levels well above those observed after a low GI meal. This combination of elevated counterregulatory hormone and free fatty acid levels resembles a state of fasting normally reached only after many hours without food. After a low GI meal, by contrast, hypoglycemia and its hormonal sequelae do not occur during the postprandial period owing to continued absorption of nutrients from the gastrointestinal tract and rising hepatic glucose output. Thus, consumption of meals containing identical energy and nutrients can produce markedly different physiological responses through out a 6-h period.

2.12 Glycemic Load and Type 2 Diabetes

Chronic state of hyperglycemia leads to loss of pancreatic β cell function that can result in glucose intolerance and ultimately an irreversible state of diabetes (37) (Figure 2.3).

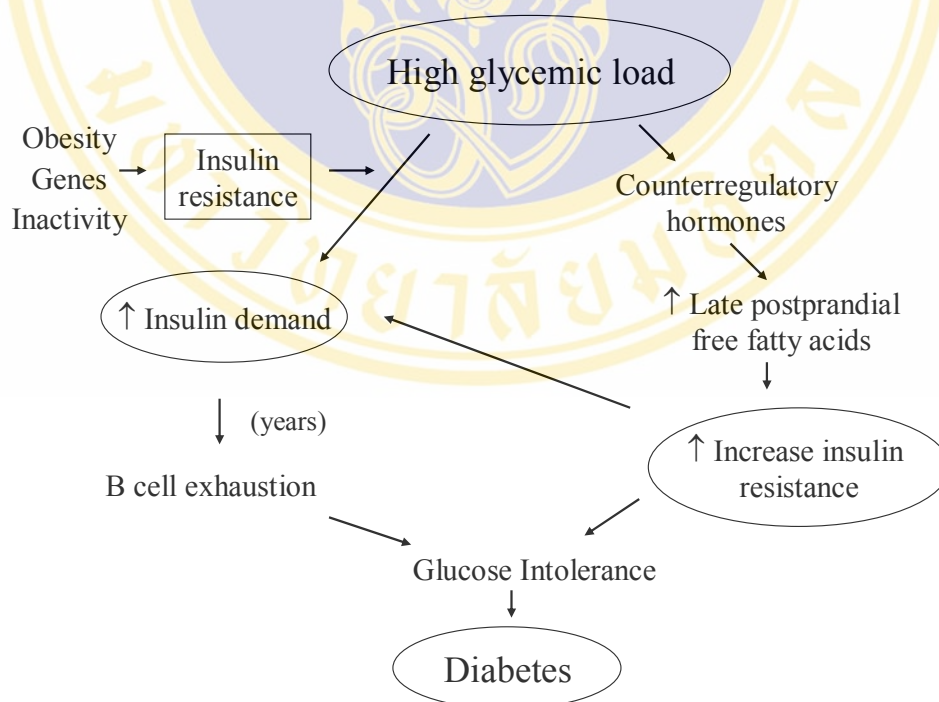


Figure 2.3 Potential mechanisms whereby high glycemic-load diets could increase risk of type 2 diabetes

This loss of pancreatic function results primarily from excessive secretion of insulin or toxicity to β cells because of hyperglycemia. Nevertheless, either mechanism would predict that a diet that produces higher blood glucose concentrations and greater demand for insulin would increase the risk of type 2 diabetes. By definition, high GI forms of carbohydrate are foods that produce high concentrations of blood glucose and increase insulin demand and that, therefore, could contribute to higher risk of type 2 diabetes.

The beneficial effects of low GI foods on postprandial metabolism might be useful not only for the treatment but also for the prevention of the metabolic syndrome diseases.

2.13 Diabetes Mellitus and Dyslipidemia

It has long been recognized that (38) the extent of atherosclerosis is markedly increased in diabetes mellitus. Serum lipid profile in poorly controlled type 2 diabetes patients consist of markedly increased triglyceride, increased total-cholesterol, increased low density lipoprotein cholesterol (LDL-C), and reduced high density lipoprotein cholesterol levels (HDL-C). The lipoprotein fractions are considered to represent different risk factors, in general terms it can be said that very low density lipoprotein (VLDL) is essentially neutral, high LDL is a positive risk factor and high HDL is a negative risk factor (protective against atherogenesis). Diets high in carbohydrates and low in fat have been reported to cause a reduction of HDL-C and LDL-C and to raise serum triglyceride levels (39-41) These increased triglyceride levels may result from an enhanced hepatic synthesis of VLDL triglycerides.

It has previously been hypothesized that the selection of foods that minimize the glucose and insulin responses may reduce the stimulus for hepatic triglyceride synthesis, i.e., hyperinsulinemia can promote hypertriglyceridemia. Indeed, it was shown in a study of Jenkins et al (42) that a reduction in the mean GI of the diet in 12 hyperlipidemic patients for a 1 month period led to a reduction in serum triglycerides and total serum and LDL-C.

2.14 Glycemic Index (GI) and Cardiovascular Disease (CVD)

The higher postprandial blood glucose and insulin levels found in a high GI diet may affect risk for CVD. High intake of starches (43) with high GI is associated with hyper-triglyceridemia. The epidemiologic data (44) suggest that a high dietary glycemic load from refined carbohydrates increases the risk of coronary heart disease (CHD), independent of known coronary disease risk factors. Hyperglycemia is associated with increased blood pressure, accelerated blood clot formation, and reduced endothelium-dependent blood flow (42, 45). In short-term intervention studies (46) of overweight and hyperlipidemic patients, low GI diets lead to improvements in CVD risk factors, including reduced LDL-C and improved insulin sensitivity.

Over the past decade, dietary advice to reduce the risk of CVD and obesity has recommended carbohydrates in place of saturated fatty acids, with little emphasis on the nature of the carbohydrate. There is concern, however, that some high-carbohydrate diets may increase the risk of CVD, type 2 diabetes and obesity, especially those that exaggerate postprandial glycemic and insulin responses and dyslipidemia (47, 48). Moreover, poor control of hyperglycemia plays a significant role in progression of CVD in people with diabetes. While large, prospective clinical trials have shown a direct relationship between the degree of glycemia and diabetic microvascular complications. In recent years, controlling postprandial glycemia has become the focus of new therapeutic approaches to reduce the burden of CVD complications in people with diabetes.

A lower GI suggests a slower rate of digestion and absorption of the sugars and starches in foods but may also indicate greater hepatic and peripheral extraction of the products of carbohydrate digestion (49). There is agreement that moderate to high carbohydrate based on low GI foods provide a benefit to glycemic control (50).

Many modern starchy foods produce higher levels of glycemia than sugar-containing foods (31). Dairy products, fruits and chocolate confectionery in particular may contain high levels of simple sugars, yet have low GI values (<50). At the other extreme, many low-fat, high-starch foods have exceptionally high GI values (>80), although pasta and legumes are important exceptions. Most wheatmeal breads and high-fiber breakfast cereals have high GI values that are similar to their refined

counterparts. For this reason, using the GI is a more precise instrument for teasing out the effects of postprandial glycemia on disease risk.

2.15 Viscosity and Blood Viscosity

The viscosity (51) of a fluid is due to the internal friction between the adjacent layers. In streamlined flow, where adjacent layers move parallel to each other, the velocity difference is a measure of the shearing within the flowing fluid and this velocity gradient is termed the shear rate. The velocity of flow and the shearing within the fluid is produced by a force which is called the shear stress. The viscosity of the fluid is defined as the ratio of the shear stress to the shear rate it produces.

Shear Rate	=	Velocity Gradient	s^{-1}
Shear Stress	=	Force / Area	Pa
Viscosity	=	Shear stress / Shear rate	Pa·s or cP

The concentration of red blood cells, or hematocrit, is the most important determinant of blood viscosity. Simple fluids (52) such as plasma and most oils, show a linear relationship between shear stress and shear rate (Newtonian behaviour) so that the viscosity remains constant. Whole blood behaves as a non-Newtonian fluid in that viscosity increases exponentially at the low shear rates (below 50 s^{-1}) that characterize venous flow. This increase is due to the larger molecular weight plasma proteins (fibrinogen and globulins) which overcome the zetapotential between erythrocytes and form rouleaux: these large cellular aggregates cause a disproportionate increase in viscosity. At shear rates below 1 s^{-1} , alterations in plasma fibrinogen around the upper limit of the physiological range have a pronounced effect on whole blood viscosity. At the high shear rates characteristic of arterial flow (above 100 s^{-1}) rouleaux are dispersed, and individual erythrocytes are deformed into ellipsoids with their long axes aligned in the direction of flow, thus viscosity is relatively low and virtually constant at high shear rates and is independent of the plasma fibrinogen.

The appropriate shear-rate range corresponding to blood flow in capillaries is difficult to determine since flow may be intermittent and, once blood has become stationary, a relatively large force (the yield stress) is required to restart movement. Thus there is uncertainty over the shear rates that characterize capillary blood flow and of the importance of measuring yield stress. About 10% of the total hemodynamic

resistance in the human circulation results from venous flow, arterioles and capillaries are responsible for approximately 70%, and the large and medium-size arteries account for only about 20%. Thus, the arteries most commonly affected by atherosclerosis normally offer relatively little resistance to arterial flow.

Relative viscosity (53) is a convenient way of expressing viscous properties by comparing the viscosity of a fluid to that of water. The relative viscosity of whole blood is in the range of 3 to 4: the relative viscosity of plasma is approximately 1.8.

2.16 Blood Viscosity in Diabetes Mellitus and CVD

It is well established that circulatory insufficiency plays a major pathological role in the long-term complication of diabetes. Blood viscosity is one of the determinants of flow abnormalities of vessel cause of circulatory insufficiency.

Physical properties of blood directly affect blood flow, and sufficient blood flow is essential for the health of all organs. Since blood supplies oxygen and nutrients needed for living cells and removes the cells waste products, when blood flow is impeded in any way, medical problems arise. Diabetes is associated with a greatly increased risk of further cardiovascular event and death. Chemical composition of blood, such as cholesterol and triglyceride levels, has been focused exclusively and related to various CVD.

Banerjee et al (54) observed that an increased whole blood viscosity >6 cP was associated with an increased risk of stroke. Barnes (55) found that whole blood viscosity at the shear rates of 0.77 s^{-1} and 2.62 s^{-1} was significantly higher in the diabetics than in the controls. Higher blood viscosity indicate the impaired function of endothelial cell, high viscosity in type 2 diabetes patient blood because of hypertriglyceridemia. Hoieggen (56) et al examined with the hyperinsulinemic isoglycemic glucose clamp technique demonstrated that at shear rate 0.5 s^{-1} whole blood viscosity tended to be correlated negatively to HDL-C ($r = -0.44$, $p = 0.1$) and was correlated positively and significantly to serum triglyceride ($r = 0.51$, $p = 0.04$) and total serum cholesterol ($r = 0.67$, $p = 0.006$) levels. At shear rate 201 s^{-1} whole blood viscosity correlated negatively to HDL-C ($r = -0.53$, $p = 0.04$) and positively to serum triglyceride ($r = 0.54$, $p = 0.04$) and total serum cholesterol ($r = 0.52$, $p = 0.05$) levels.

CHAPTER 3

MATERIALS AND METHODS

3.1 Materials

3.1.1 Subjects

The study was carried out in hyperlipidemic type 2 diabetic subjects from out patient department of Ramathibodi Hospital. Inclusion criteria: fasting plasma glucose less than 300 mg/dL, total cholesterol and triglyceride levels more than 200 and 150 mg/dL, respectively. Subjects had been treated with diet alone or diet and oral hypoglycemic agents, did not have acute or chronic complications of diabetes mellitus. All subjects continued and kept constant all therapies throughout the whole study.

3.1.2 Dessert Preparation

1. First, surveying the popular dessert consumed by type 2 diabetes, by using questionnaire at the out-patient department of Ramathibodi Hospital. The results confirmed that most diabetes patients consumed desserts, although these desserts were prohibited (questionnaire and results are shown in Appendix).
2. The reason for selecting Khanom-Tian (KT) as the test model because it can be made from several kinds of flour, easy to make, can be prepared in every house.
3. Establishment of the standard recipe from generally sold Thai dessert recipe to make Khanom-Tian call “Standard Khanom-Tian” (StdKT).
4. Adjustment of the StdKT to a new recipe call “Modified standard KT (Modified stdKT) which contained less sugar content than StdKT but contained the same amount of total carbohydrate.
5. Modification of the Modified stdKT by changing the covering with other kinds of flour according to the concept of GI testing (50 g CHO) and the acceptability.

The final study desserts were 5 types of KT recipe and 50 g glucose drink (used as a reference standard for determination of the GI). Study desserts consisted of:

- a) Standard KT (StdKT) recipe composing of sucrose with other constituents as the filling, wrapped with white glutinous rice flour as the cover (Figure 3.1).
- b) Modified standard KT* (Modified stdKT) is the recipe modified from StdKT which had less sugar content than StdKT but had the same 50 g CHO, being wrapped with the same white glutinous rice flour (Figure 3.2).
- c) Black glutinous rice flour KT* (Figure 3.3)
- d) Mungbean starch KT* (Figure 3.4)
- e) Small sago (cassava-pearl) KT* (Figure 3.5)

* b-e contained the same amount of the filling and sucrose content, differed only the covering starch or flour.

Nutrient composition and energy distribution of each serving of Khanom-Tian were calculated based on data from food composition table of Division of Health, Ministry of Public Health, 1987 described in Table 3.1 and the detail is shown in Appendix.

3.1.3 Characteristics of Starch/Flour

- a) **Glutinous rice flour (57, 58)** (Pang Kow Neow) Glutinous rice flour is also known as sweet rice flour or sweet rice powder, this flour is made from short-grain rice that becomes moist, firm and sticky when cooked, a result of its higher proportion of waxy starch molecules. Because of its chewy texture, glutinous rice flour is a favorite base for dumplings, buns and pastries. Sweets made with glutinous rice flour are popular and widely available throughout Southeast Asia. Glutinous rice flour has ~3-4% amylose.
- b) **Small sago (cassava pearl) (26, 58)** (sa khu met lek) are the tiny balls (about 2 mm in diameter) made from tapioca, some used in sweets. They should be mixed with hot, but not scalding, water and kneaded, and then allowed to stand for a time covered with a damp cloth to permit the water to penetrate to the core. Small sago granules are smooth, irregular spheres with sizes ranging from 5 to 25 microns. Small sago has ~21% amylose.

Table 3.1 Nutrient composition and energy distribution of each serving of KT

	StdKT	Modified stdKT	Black glutinous rice flour KT	Mungbean starch KT	Small sago (Cassava pearl) KT
CHO, g (%)	50.1 (74)	50.9 (80)	50.2 (79)	50.7 (83)	50.5 (83)
- sugar, g (%)	22.0 (33)	12.5 (20)	12.5 (20)	12.5 (20)	12.5 (20)
- flour, g (%)	19.0 (28)	26.5 (41)	25.7 (40)	26.2 (43)	26.0 (43)
- mungbean dehulled, g (%)	8.9 (13)	11.9 (19)	11.9 (19)	11.9 (20)	11.9 (20)
Protein, g (%)	5.4 (8)	7.3 (11.5)	7.4 (12)	5.2 (8.5)	5.2 (8.5)
Fat, g (%)	5.3 (18)	2.4 (8.5)	2.6 (9)	2.3 (8.5)	2.3 (8.5)
Energy (Kcal)	270	254	254	244	244
Pieces	3	4	4	8	4

- c) **Mungbean starch (58, 59)** made from 100% mungbean (Tau keaw). The starch is available in its natural (white) state or tinted pink or green, the color only showing when water is added. This starch is used to make transparent bean thread vermicelli, variously called cellophane noodles, jelly noodles, glass noodles and, even more poetically, spring rain noodles. Mungbean starch has ~30% amylose and advantageous properties of short texture, high gel strength and easy peeling. It makes an excellent ingredient for many kinds of food such as soup, dessert, bean sheet and vermicelli.

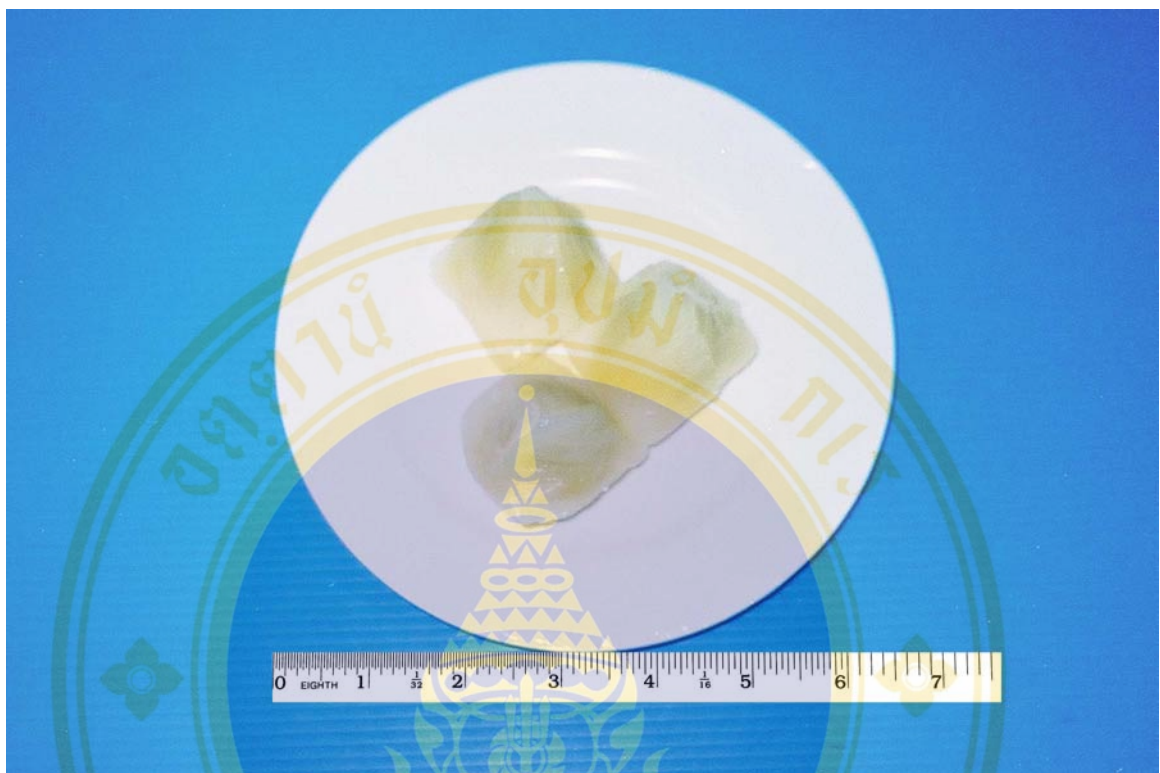


Figure 3.1 Standard Khanom-Tian (stdKT)



Figure 3.2 Modified standard Khanom-Tian (modified stdKT)



Figure 3.3 Black glutinous rice flour Khanom-Tian



Figure 3.4 Mungbean starch Khanom-Tian

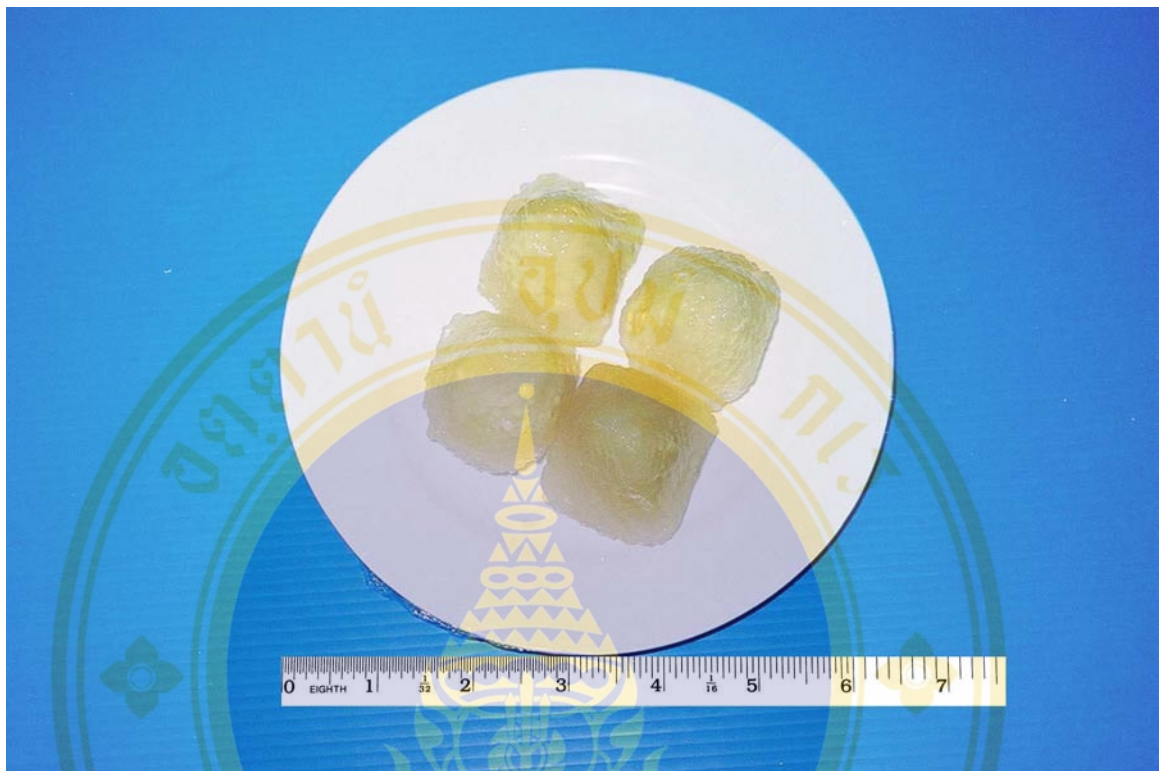


Figure 3.5 Small sago Khanom-Tian



Figure 3.6 Khanom-Tian

3.2 Methods

3.2.1 Obtaining Approval

The objectives and details of the study were explained, and a written informed consent was obtained from all subjects. The study was approved by the ethics committees of Ramathibodi Hospital, and performed in accordance with the principles of the declaration of Helsinki.

3.2.2 Experimental Design

This study was a randomized control-trial design. All subjects consumed 5 study KT_s and 50 g glucose drink (dissolved in 150 ml water, was used as the reference food) at 4 weeks apart.

In the morning, after 10-h overnight fast, subjects had omitted their ordinary medication. Blood samples were drawn through an indwelling catheter from an antecubital vein in the fasting state to determine the baseline of plasma glucose, lipid profile (triglyceride, total-cholesterol, LDL-C, HDL-C) and blood viscosity. After that, subjects ate one recipe of KT within 15 min, no other food or beverage was permitted, only 150 ml of water (Each KT was prepared before the evaluation and stored at room temperature). Postprandial blood samples were collected to determine the biochemical parameter at the time described in Table 3.2. Sensory testing was evaluated during the last time of the study.

Table 3.2 Time for blood withdrawal and testing

Time (min)	0	30	60	120	180	240	300
Plasma glucose	X	X	X	X	X	X	X
Lipid profile	X	-	-	-	-	-	X
Blood viscosity	X	-	X	-	-	-	X

3.3 The Sensory Evaluation Test

For sensory evaluation test, each KT was prepared in the plate and labeled with a number 1-5. The sensory evaluation questionnaire was used to evaluate after subject tasted. The acceptance of each KT was scored as follow: 1 = very unpleasant, 2 = unpleasant, 3 = acceptable, 4 = good, 5 = very good. The aims of evaluation were texture, and taste of covering flour, filling, size, overall and the appropriate piece/serving. The questionnaire was attached in the Appendix.

3.4 Anthropometric Measurement

Anthropometric measurements were performed before the study for assessment of nutrition status. Body weight was measured by using Tanita weighting scale (Tokyo Japan), waist, hip and mid upper arm circumference using non-stretching tape, triceps skin-fold thickness using Harpenden skin-fold calipers (British Indicators, LTD, London). Body mass index, waist to hip circumference ratio and upper arm muscle circumference was calculated from the formula mentioned in the following.

3.4.1 Weight (Wt) (60)

Subjects should be weighted wearing light clothing and standing without shoes or socks in a position using the accurate weighing scale.

3.4.2 Height (Ht) (60)

Subjects should stand erect with eyes facing forward and with feet, knees, buttocks and shoulder blades making contact with the vertical surface.

3.4.3 Body Mass Index (BMI) (61)

BMI is the preferred weight-for-height standard and is used as a determinant of health risk and a predictor of mortality.

$$\text{BMI} = \text{weight (kg)} / \text{height}^2 \text{ (m)}$$

3.4.4 Triceps Skinfold Thickness (TSF) (62, 63)

TSF is the site most frequently used to obtain a single indirect measure of body fat. The measurement of the TSF is performed at the midpoint between the tip of

the acromial process of the scapula and the olecranon process of the ulna. The fold run parallel to the long axis of the arm. Care should be taken to ensure that the measurement is made in the midline posteriorly and that the arm hangs relaxed and vertical. Lift the skin and fat layer from the underlying tissue by grasping the tissue with the thumb and forefinger. Apply calipers about 1 cm distal from the thumb and forefinger, midway between the apex and base of the skinfold. Continue to support the skinfold with the thumb and forefinger for the duration of the measurement. After 2 to 3 sec of caliper application, read skinfold to the nearest 0.5 mm.

3.4.5 Mid Upper Arm Circumference (MUAC)

The arm contains subcutaneous fat and muscle, a decrease in MUAC may therefore reflect either a reduction in muscle mass, a reduction in subcutaneous tissue, or both. This circumference is taken at the midpoint between the acromial and olecranon processes of the scapula and the ulna, respectively. The arm should hang relaxed at the patient's side. The non-stretching tape should be maintained in a horizontal position touching the skin and following the contours of the limb, but not compressing underlying tissue.

3.4.6 Upper Arm Muscle Circumference (UAMC)

UAMC represents the circumference of the inner circle of muscle mass surrounding a small central core of bone, can be used to assess total body muscle mass.

$$\text{UAMC} = \text{MUAC} - 3.1416 \text{TSF}$$

3.4.7 Waist Circumference (61, 62)

Waist circumference is an indicator of upper abdominal fat (stomach area) accumulation and is used to assess central or upper-body obesity. To perform the waist measurement, non-stretching tape is applied horizontally midway between the lowest rib and the iliac crest, and tied firmly so that it stays in position around the abdomen about the level of the umbilicus.

3.4.8 Hip Circumference (61)

For the hip circumference measurement, be taken at the point yielding the maximum circumference over the buttocks with the non-stretching tape held in a horizontal plane.

3.4.9 Waist to Hip Circumference Ratio (WHR) (64)

WHR is a method used to assess abdominal fat distribution by measuring both waist and hip circumference and calculating ratio. A WHR of 1.0 or greater in men and 0.8 in women is indicative of android obesity and an increased risk of obesity-related disease.

$$\text{WHR} = \text{waist circumference} / \text{hip circumference}$$

3.5 Biochemical Analysis

Blood samples were collected in sodium fluoride tubes for determining plasma glucose and clotted blood for lipid profile. All analyses were performed on the Dimension© Clinical Chemistry System (Dade Behring Company, Newyork, NJ)

Plasma glucose levels were measured using the hexokinase-glucose-6-phosphate-dehydrogenase method (65). Total cholesterol (66), HDL-C (67) and triglycerides were determined by enzymatic colorimetric method (68) and LDL-C was calculated according to Friedewald equation (69).

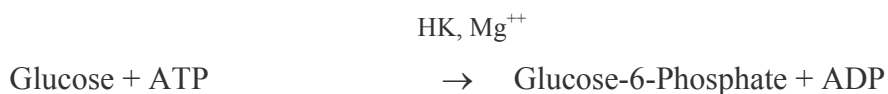
3.5.1 Determination of Glucose

Dade Behring Company

Machine: Dimension© Clinical Chemistry System

The glucose method is an adaptation of the hexokinase-glucose-6-phosphate-dehydrogenase method, presented as a general clinical laboratory method.

Principle



Glucose-6-PDH



NADPH (and glucose concentration) absorbance is determined using a biochromatic (340 and 383 nm) endpoint technique.

Glucose concentration was calculated according to the following equation:

$$\text{Glucose concentration} = (A \times 100) / A_s$$

3.5.2 Determination of Serum Triglyceride

Dade Behring Company

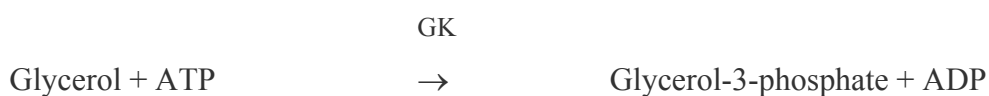
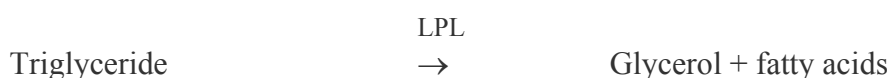
Machine: Dimension® Clinical Chemistry System

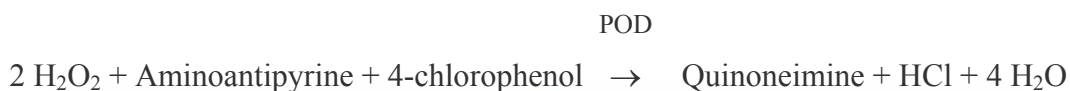
Reagent: Flex® reagent cartridge

Principle

Triglyceride method is based on an enzymatic procedure in which a combination of enzyme are employed for the measurement of serum or plasma triglyceride.

The sample is incubated with lipoprotein lipase (LPL) enzyme reagent that converts triglyceride into free glycerol and free fatty acids. Glycerol kinase (GK) catalyze the phosphorylation of glycerol by adenosine-5-triphosphate (ATP) to glycerol-3-phosphate. Glycerol-3-phosphate oxidase (GPO) oxidize glycerol-3-phosphate to dihydroxyacetone phosphate and hydrogen peroxide. The catalytic reaction of peroxidase (POD) produces quinoneimine chromophore.





Wavelengths biochromic end point: 510 and 700 nm

3.5.3 Determination of Serum Total Cholesterol

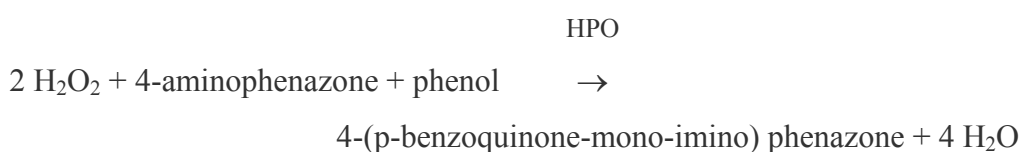
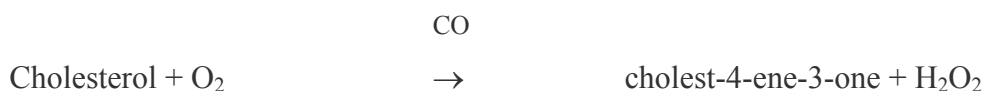
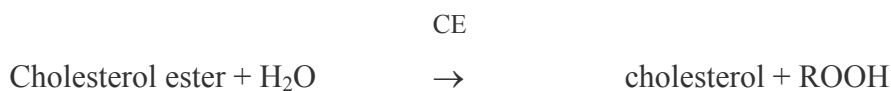
Dade Behring Company

Machine: Dimension® Clinical Chemistry System

Reagent: Flex™ reagent cartridge

Principle

Cholesterol esterase (CE) catalyzes the hydrolysis of cholesterol esters to produce free cholesterol which, along with preexisting free cholesterol, is oxidized in a reaction catalyzed by cholesterol oxidase (CO) to form cholest-4-ene-3-one and hydrogen peroxide. In the presence of horseradish peroxidase (HPO), the hydrogen peroxide thus formed is used to oxidize N, N-diethylaniline-HCl/4-aminoantipyrine (DEA-HCl/AAP) to produce a chromophore that absorbs at 540 nm. The absorbance due to oxidized DEA-HCl/AAP is directly proportional to the total cholesterol concentration and is measured using a trichromatic (540, 452, 700 nm) endpoint technique.



Calculation

$$\text{Cholesterol} = \frac{5.17 \times \Delta A (\text{sample})}{\Delta A (\text{standard})} \quad (\text{mmol/L})$$

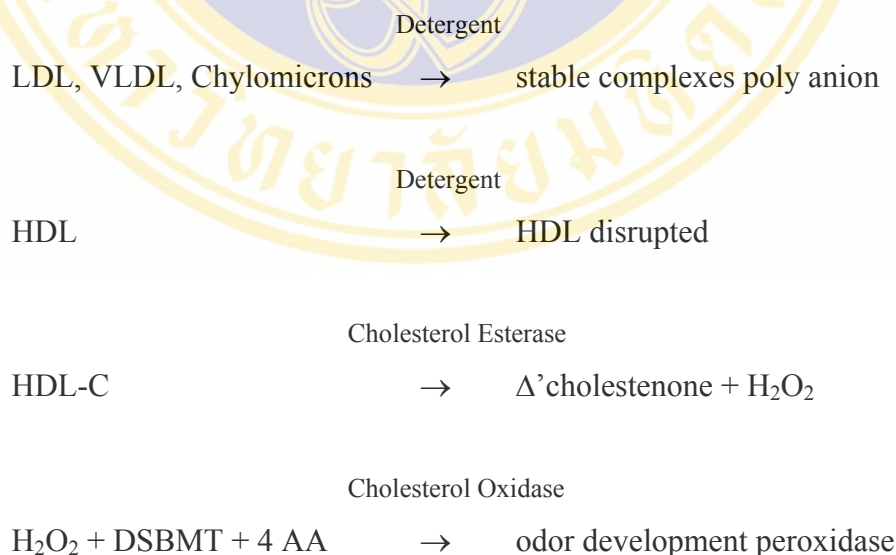
3.5.4 Determination of High Density Lipoprotein Cholesterol (HDL-C)

Dimension® Clinical Chemistry System

Automated HDL-C Flex™ reagent cartridge

Principle

Primary reagent solubilized only HDL lipoprotein particle, thus releasing HDL-C to react with cholesterol esterase to produce color. In addition to selectively disrupting the HDL lipoprotein particles, this detergent also inhibits the reaction of the cholesterol enzyme with LDL, VLDL and chylomicron lipoproteins by adsorbing to their surface. A poly anion is contained in the first reagent to assist with complex LDL, VLDL and chylomicron lipoproteins, further enhancing the selectivity of the detergent and enzymes for HDL-C



3.5.5 Estimation of Low Density Lipoprotein Cholesterol (LDL-C)

LDL-C level was calculated from Friedewald's formula as follow:

$$\text{LDL-C} = \text{Total-Cholesterol} - (\text{TG}/5) - \text{HDL-C}$$

3.6 Calculation of Glycemic Index

The GI (70) is the incremental area under the curve (AUC) of blood glucose produced by a standard amount of carbohydrate (CHO) in food, usually 50 g, relative to the incremental area produced by the same amount of carbohydrate from a standard food, usually white bread or glucose, taken by the same subject.

The AUC value in this study was calculated over 5 h for 7 times point used the program AUC by applied Wolever rule (71) using glucose as the reference food, only the area above the fasting level is included.

$$GI = \frac{\text{AUC after eating food containing 50 g CHO} \times 100}{\text{AUC after drinking 50 g glucose}}$$

3.7 Measurement of Blood Viscosity

Eight ml of venous whole blood was drawn into separate tubes from each subject. The tube contained ethylene diamine tetra acetic acid (EDTA) (1 mg/ml) as anticoagulant. Brookfield viscometer Model LVDV-II+ (Brookfield Engineering Laboratories, Inc., Middleboro, MA, USA) coupled with a small sample adapter was used to measure the viscosity of blood at 37°C using spindle NO SC4-18 at a shear rate of 264 s⁻¹. The measurements were performed as early as possible to avoid the problem of time.

Principle (72)

The principal of operation of Brookfield viscometer Model LVDV-II+ is to drive a spindle in the test fluid through a calibrated spring. The viscous drag of the fluid against the spindle is measured by the spring deflection. Spring deflection is measured with a rotary transducer. Viscosity appears in units of centipoise (shown as cP) is determined by the rotational speed of the spindle, the size and shape of the spindle, the container the spindle is rotating in, and the full scale torque of the calibrated spring.

3.7.1 General Techniques in Viscometry (51)

Venous blood is used routinely, and it is important to avoid stasis. The specimen is placed in an anti-coagulant container and gently mixed. The most commonly used anti-coagulants in the literature are solid lithium heparin and EDTA.

One of the biggest problems in viscosity measurement is the effect of storage on the physical properties of whole blood. The results of measurements of whole-blood viscosity begin to change within a few hours of withdrawal of the blood. If the sample has been kept at room temperature all measurements should be carried out within 4-8 h. At 4°C the sample may maintain its essential physical characteristics for approximately 12 h. These changes will be greater if measurements are made at low rather than high shear rates. Measurements of viscosity are usually carried out at 37°C and all viscometers should be equipped with systems for thermostatically controlling the temperature of the sample.

3.8 Statistical Analysis

Statistical analysis was performed using the computerized statistical program (SPSS 11.5 for Windows Inc., 2002 Chicago, Illinois, USA). The results were reported as mean $\pm$ SD. Percentage changes between baseline and each time periods were calculated for plasma glucose, blood viscosity and lipid profile. Comparisons of plasma glucose and percentage changes of plasma glucose responses between time periods and blood viscosity before 60 and 300 min by Friedman's Test and multiple comparisons by Bonferroni adjusted Mann-Whitney test. Wilcoxon Signed Ranks test was used to compare the difference between serum lipids before and after ingestion the test dessert. Comparisons of AUC, GI, actual plasma glucose, actual serum lipid, actual blood viscosity, percentage change of serum lipid and sensory evaluation by Kruskal-Wallis H Test and Multiple comparisons by Bonferroni adjusted Mann-Whitney test. Spearman correlation was used to determine the relationship of plasma glucose response and lipid profile with blood viscosity. P-value of less than 0.05 was considered significant.

CHAPTER 4

RESULTS

4.1 Clinical Characteristics of Subjects

Table 4.1 shows the anthropometric measurements before the study of the 17 hyperlipidemic type 2 diabetic subjects, 3 men and 14 women. The biochemical profiles before the study are shown in Table 4.2. All the subjects completed the study.

4.2 Plasma Glucose Levels

Table 4.3 compares the mean $\pm$ SD of plasma glucose levels between before and after ingestion of all recipes of KT. The glucose levels rose significantly from baseline at 30, 60 and 120 min in all recipes of KT. The postprandial glucose curves after the 5 different KTs are shown clearly in Figure 4.1. In all cases the highest mean in the postprandial curves were observed 60 min after the start of the meal. The mean plasma glucose levels, only mungbean starch KT recipe and modified stdKT recipe were lower than baseline at 240 min, whereas the others remained higher, finally dropped below the baseline at 300 min in all recipes of KT. Mungbean starch KT gave the lowest peak, whereas glucose gave the highest peak.

Table 4.4 and Figure 4.2 show the percentage change of plasma glucose levels between before and after ingestion of all recipes of KT. There were no significant differences in the mean plasma glucose levels and the mean percentage change of plasma glucose levels at fasting and during postprandial period between all recipes of KT.

Table 4.1 Anthropometric measurements of the subjects at the first time

Subject	Sex	Age yrs	Wt kg	Ht m	BMI kg/m ²	% Body Fat	Waist cm	Hip cm	WHR	MUAC cm	TSF mm	UAMC cm
1	M	61	81.4	1.65	29.9	34.0	109.0	107.0	1.02	34.5	21.0	27.91
2	F	53	74.2	1.51	32.5	37.0	98.0	102.0	0.96	38.0	31.0	28.27
3	F	61	50.4	1.45	24.0	30.0	83.0	89.0	0.93	24.5	19.0	18.53
4	F	61	52.2	1.53	22.3	28.5	81.0	95.0	0.85	24.5	24.5	16.81
5	F	54	69.2	1.59	27.4	28.0	88.0	96.0	0.92	30.0	31.0	20.27
6	F	66	70.6	1.50	31.4	43.5	90.0	103.5	0.87	35.0	38.0	23.07
7	F	73	54.2	1.60	21.2	31.5	76.5	91.5	0.84	28.0	23.0	20.78
8	M	57	60.2	1.65	22.1	17.5	86.0	94.0	0.91	28.0	23.0	20.78
9	F	57	56.0	1.55	23.3	29.5	81.5	86.0	0.95	27.0	18.0	21.35
10	F	65	56.8	1.55	23.6	28.0	80.5	96.0	0.84	26.5	15.0	21.79
11	F	65	53.2	1.51	23.3	30.5	77.5	93.5	0.83	28.0	25.0	20.15
12	F	45	126.2	1.60	49.3	56.0	129.5	144.0	0.90	45.0	46.0	30.56

Table 4.1 (cont) Anthropometric measurements of the subjects at the first time

Subject	Sex	Age yrs	Wt kg	Ht m	BMI kg/m ²	% Body Fat	Waist cm	Hip cm	WHR	MUAC cm	TSF mm	UAMC cm
13	M	50	81.0	1.78	25.6	22.5	94.0	100.0	0.94	31.0	16.0	25.98
14	F	76	62.2	1.48	28.4	37.0	89.0	103.5	0.86	27.0	30.0	17.58
15	F	51	90.6	1.55	37.7	47.5	107.0	125.0	0.86	37.0	36.5	25.54
16	F	70	65.4	1.57	26.5	46.0	93.5	101.0	0.93	29.0	21.0	22.41
17	F	67	53.6	1.43	26.2	32.5	77.0	94.0	0.82	31.0	26.0	22.84
Mean	F + M	61	68.1	1.56	27.9	34.1	90.7	101.2	0.90	30.8	26.1	22.6
SD	F + M	9	19.1	0.08	7.0	9.6	14.0	14.1	0.06	5.5	8.4	3.9
Mean	F	62	66.8	1.53	28.4	36.1	89.4	101.4	0.88	30.8	27.4	22.1
SD	F	9	20.4	0.05	7.6	8.8	14.5	15.4	0.05	5.9	8.6	3.9
Mean	M	56	74.2	1.67	25.9	24.7	96.3	100.3	0.96	31.2	20.0	24.9
SD	M	6	12.1	0.03	3.9	8.5	11.7	6.5	0.06	3.3	3.6	3.7

Table 4.2 Clinical characteristics of the subjects before the study

Subject	FPG	Triglyceride	Cholesterol	HDL-C	LDL-C
	mg/dL	mg/dL	mg/dL	mg/dL	mg/dL
1	133	266	307	76	178
2	146	168	269	56	180
3	207	313	263	36	164
4	139	181	278	56	186
5	211	266	325	42	231
6	141	392	207	34	95
7	160	189	205	35	132
8	117	550	238	43	76
9	103	188	280	22	222
10	225	166	262	48	181
11	166	173	252	65	153
12	162	191	225	46	142
13	124	171	227	42	151
14	169	157	208	55	122
15	198	157	207	26	150
16	139	165	204	23	148
17	141	150	201	58	113
Mean	158	226	245	45	154
SD	35	106	39	15	41

Table 4.3 Comparison of plasma glucose responses (Mean $\pm$ SD) between before and after ingestion of different recipes of KT

Type of KT	Time (min)						
	0	30	60	120	180	240	300
	<-----mg/dL----->						
Standard	144 ± 40	207 ± 50 ^a	251 ± 64 ^{a,b}	219 ± 78 ^a	176 ± 68 ^{c,d}	147 ± 61 ^{b,c,d,e}	125 ± 50 ^{b,c,d,e,f}
Modified std	147 ± 38	197 ± 45 ^a	250 ± 55 ^{a,b}	204 ± 70 ^{a,c}	175 ± 63 ^{c,d}	143 ± 55 ^{b,c,d,e}	130 ± 48 ^{b,c,d,e}
Black glutinous rice flour	134 ± 41	182 ± 49 ^a	242 ± 63 ^{a,b}	207 ± 79 ^{a,c}	168 ± 79 ^{c,d}	137 ± 66 ^{b,c,d,e}	122 ± 54 ^{b,c,d,e}
Mungbean starch	146 ± 43	197 ± 44 ^a	234 ± 57 ^{a,b}	208 ± 70 ^a	164 ± 63 ^{c,d}	135 ± 53 ^{c,d,e}	121 ± 42 ^{a,b,c,d,e,f}
Small sago	134 ± 36	196 ± 36 ^a	239 ± 61 ^{a,b}	210 ± 72 ^a	179 ± 67 ^{c,d}	152 ± 57 ^{b,c,d,e}	130 ± 43 ^{b,c,d,e,f}
Glucose drink	138 ± 25	228 ± 39 ^a	296 ± 58 ^{a,b}	245 ± 72 ^{a,c}	169 ± 68 ^{b,c,d}	126 ± 49 ^{b,c,d,e}	108 ± 28 ^{a,b,c,d,e}

There was no significant difference between different recipes of KT

^a Significantly different from baseline

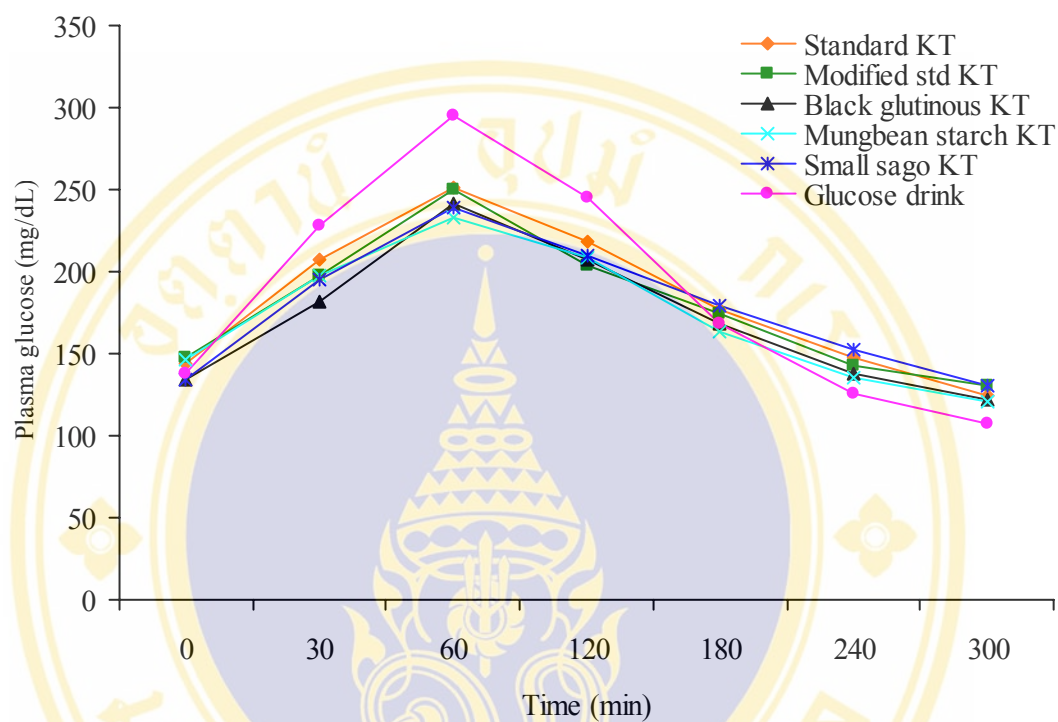
^b Significantly different from 30 min

^c Significantly different from 60 min

^d Significantly different from 120 min

^e Significantly different from 180 min

^f Significantly different from 240 min



There was no significant difference between different recipes of KT

Figure 4.1 Mean plasma glucose responses after ingestion of different recipes of KT

Table 4.4 Comparison between percentage change of plasma glucose (Mean $\pm$ SD) after ingestion of different recipes of KT

Type of KT	Time (min)						
	0	0-30	0-60	0-120	0-180	0-240	0-300
		<-----mg/dL----->					
Standard	-	46.7 ± 32.7 ^a	77.8 ± 40.3 ^{a,b}	52.7 ± 43.4 ^{a,c}	22.4 ± 37.8 ^{c,d}	1.6 ± 30.9 ^{b,c,d,e}	- 13.6 ± 25.4 ^{b,c,d,e,f}
Modified Standard	-	35.8 ± 21.5 ^a	72.8 ± 29.1 ^{a,b}	38.4 ± 35.4 ^{a,c}	17.7 ± 31.3 ^{c,d}	- 4.3 ± 27.8 ^{b,c,d,e}	- 12.0 ± 24.6 ^{b,c,d,e}
Black glutinous rice flour	-	38.3 ± 25.5 ^a	83.3 ± 25.6 ^{a,b}	53.7 ± 32.9 ^{a,c}	21.7 ± 33.8 ^{c,d}	- 0.7 ± 25.4 ^{b,c,d,e}	- 10.8 ± 17.3 ^{b,c,d,e}
Mungbean starch	-	39.3 ± 25.2 ^a	64.5 ± 30.4 ^{a,b}	44.2 ± 30.4 ^{a,c}	12 ± 25.9 ^{b,c,d}	- 8.1 ± 20.5 ^{b,c,d,e}	- 17.3 ± 12.6 ^{a,b,c,d,e,f}
Small sago	-	51.3 ± 28.9 ^a	81.9 ± 36.0 ^a	59.3 ± 45.1 ^a	35.5 ± 45.1 ^{c,d}	15.0 ± 36.7 ^{b,c,d,e}	- 0.2 ± 30.1 ^{c,d,e,f}
Glucose drink	-	67.6 ± 23.3 ^a	116.8 ± 34.2 ^{a,b}	77.6 ± 38.4 ^{a,c}	19.7 ± 37.6 ^{b,c,d}	- 10.8 ± 27.1 ^{b,c,d,e}	- 22.1 ± 14.5 ^{a,b,c,d,e}

There was no significant difference between different recipes of KT

^a Significantly different from baseline

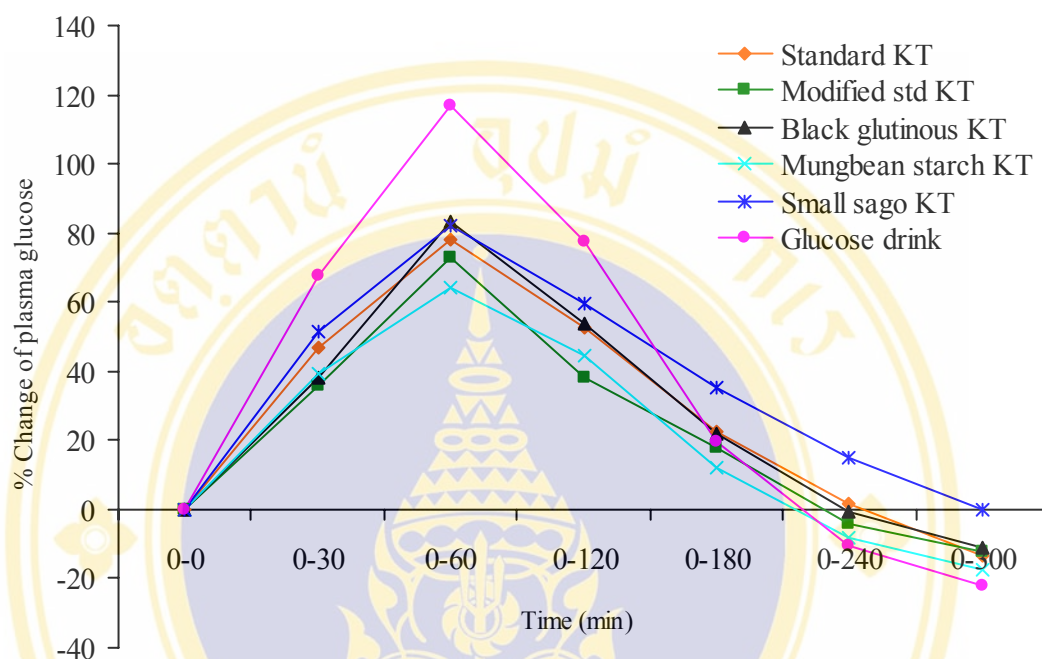
^b Significantly different from 0-30 min

^c Significantly different from 0-60 min

^d Significantly different from 0-120 min

^e Significantly different from 0-180 min

^f Significantly different from 0-240 min



There was no significant difference between different recipes of KT

Figure 4.2 Percentage change of mean plasma glucose after ingestion of different recipes of KT

4.3 AUC and GI

Table 4.5 and Figures 4.3-4.4 show the AUC and GI of all recipes of KT. The AUC over 5 h was largest after eating small sago KT recipe followed by stdKT recipe, black glutinous rice flour KT recipe, modified stdKT recipe and the smallest area was mungbean starch KT recipe. Their glycemic indexes were 82, 72, 71, 61 and 57, respectively. There was no significant difference in AUC between different recipes of KT. But GI of all recipes of KT were significantly different from glucose drink, except small sago KT.

4.4 Lipid Profile

4.4.1 Serum Triglyceride Levels

Table 4.6 and Figures 4.5-4.6 show the actual levels of serum triglycerides and the percentage changes of serum triglyceride level after ingestion of all recipes of KT and glucose drink. Except for the glucose drink, the mean postprandial triglyceride levels at 300 min after ingesting all recipes of KT were significantly increased from baseline. There were no significant differences in the mean and percentage change of triglyceride levels between all recipes of KT at 0 and 300 min.

4.4.2 Serum Cholesterol Levels

Table 4.7 and Figures 4.7-4.8 show the actual levels of serum cholesterol and the percentage changes of serum cholesterol level after ingestion of all recipes of KT and glucose drink. There were significant decreases in the means of cholesterol levels after ingestion of small sago KT and glucose drink at 300 min from baseline. Between all recipes of KT at 0 and 300 min, there was no significant difference in the mean and mean percentage change of cholesterol.

4.4.3 Serum HDL-C Levels

Table 4.8 and Figures 4.9-4.10 show the actual levels of serum HDL-C and the percentage changes of serum HDL-C level after ingestion of all recipes of KT and glucose drink. Except for the glucose drink there was no significant difference in the mean of HDL-C levels at 300 min after ingestion of all KT. There were no significant differences in the mean and mean percentage change of HDL-C between all of them.

4.4.4 Serum LDL-C Levels

Table 4.9 and Figures 4.11-4.12 show the actual levels of serum LDL-C and the percentage changes of serum LDL-C level after ingestion of all recipes of KT and glucose drink. The mean of LDL-C after eating modified stdKT was significantly decreased from baseline. There were no significant differences in the mean and mean percentage change of LDL-C between all of them.

4.5 Blood Viscosity

Table 4.10 and Figures 4.13-4.14 show the actual levels of blood viscosity and the percentage changes of blood viscosity after ingestion of all recipes of KT and glucose drink. The mean of blood viscosity was significant decreases at 300 min from baseline after ingestion of small sago KT. At 60 min from baseline there were significant decreased of mean blood viscosity in all recipes of KT and glucose drink. There were no significant differences in the mean and mean percentage change of blood viscosity between all recipes of KT.

4.6 Blood Viscosity and Lipid Correlation

Although, there was no significant differences happen in whole blood viscosity after ingestion of the KTs. We found a significant positive correlation among triglycerides levels and blood viscosity of the whole group of subjects ($r = 0.186$, $p < 0.05$) as shown in Figure 4.15.

4.7 Sensory Evaluation Test

Table 4.11 shows the sensory evaluation scores after ingestion of all recipes of KT by 17 subjects during the last visit by using 5 point score scale (1 = very unpleasant, 2 = unpleasant, 3 = acceptable, 4 = good, 5 = very good). Modified stdKT was accepted with the highest score in the covering: texture, stickiness and its taste, also taste for filling and overall. In contrast, mungbean starch KT received the lowest score in all of characteristics. Three pieces per serving is the number of subjects accepted.

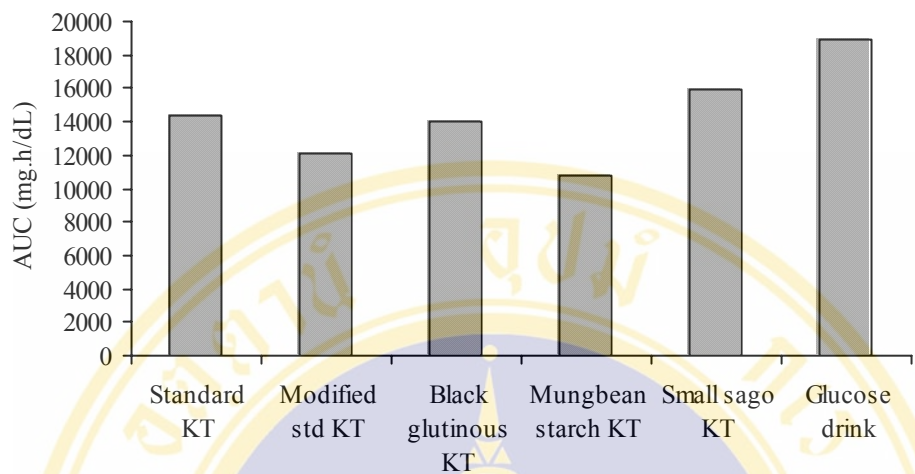
Table 4.5 Area under the glucose curve (AUC) (mean $\pm$ SD), glycemic index (GI) and glycemic rank after ingestion of different recipes of KT

Type of KT	AUC (mg.h/dL)	GI*	Glycemic rank
Standard KT	14394 $\pm$ 9673	72 ^a	2
Modified standard KT	12120 $\pm$ 8245	61 ^a	4
Black glutinous rice flour KT	14031 $\pm$ 8217	71 ^a	3
Mungbean starch KT	10778 $\pm$ 5674	57 ^a	5
Small sago KT	15976 $\pm$ 10494	82	1
Glucose drink	18945 $\pm$ 8793	100	

* Glycemic indexes were determined with 50 g of carbohydrate ingestion

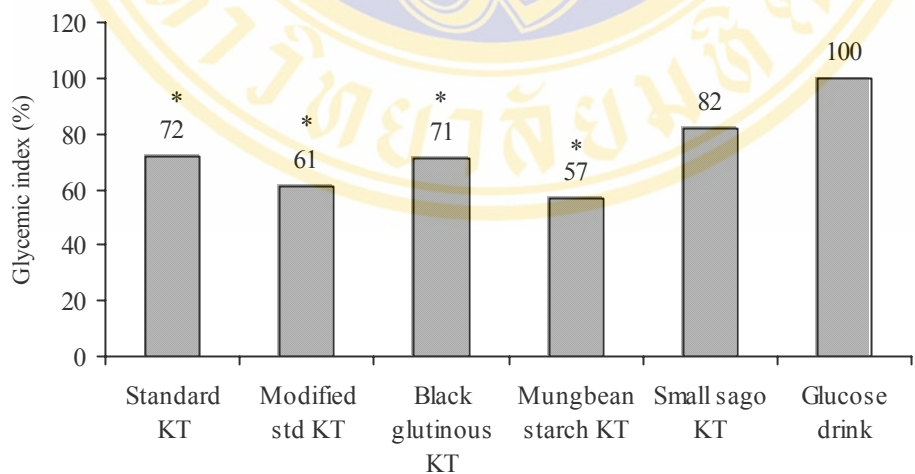
There was no significant difference in AUC between different recipes of KT

^a Significantly different from glucose drink



There was no significant difference between different recipes of KT

Figure 4.3 Mean areas under the glucose response curves after ingestion of different recipes of KT



* Significantly different from glucose drink

Figure 4.4 Glycemic indexes of different recipes of KT

Table 4.6 Comparison of serum triglycerides responses before and after ingestion of different recipes of KT

Type of KT	Time (min)		
	0	300	0-300
	<----- mg/dL ----->		% Change
Standard KT	198 ± 180	203 ± 120 [*]	15.1 ± 24.5
Modified standard KT	179 ± 138	222 ± 104 [*]	36.9 ± 26.3
Black glutinous rice flour KT	164 ± 121	167 ± 79 [*]	12.3 ± 24.7
Mungbean starch KT	171 ± 147	181 ± 74 [*]	25.0 ± 35.3
Small sago KT	195 ± 163	202 ± 99 [*]	20.0 ± 24.6
Glucose drink	189 ± 122	196 ± 79	16.7 ± 31.3

* Significantly different from baseline

There was no significant difference between different recipes of KT

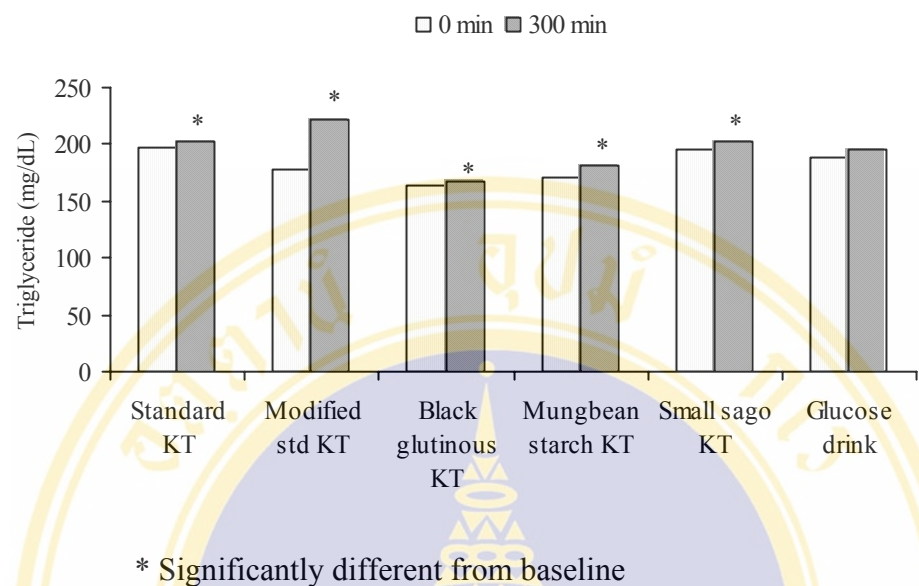
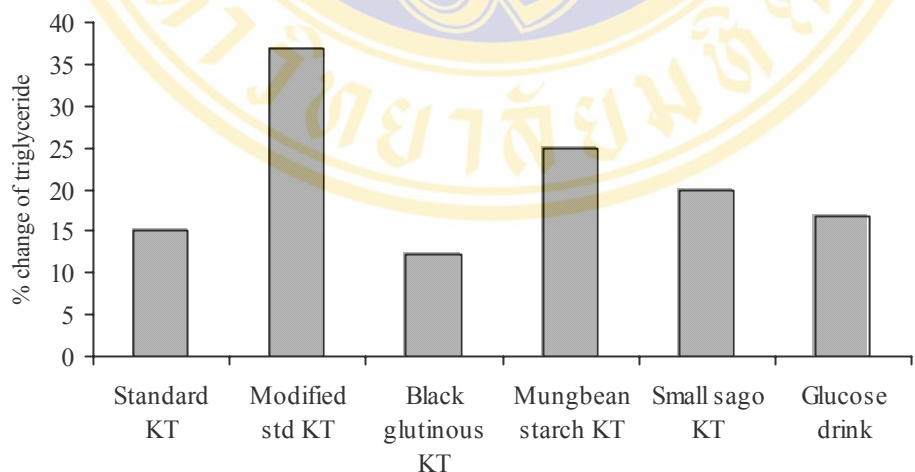


Figure 4.5 Mean triglyceride levels before and after ingestion of different recipes of KT



There was no significant difference between different recipes of KT

Figure 4.6 Mean percentage change of triglyceride after ingestion of different recipes of KT

Table 4.7 Comparison of serum cholesterol responses before and after ingestion of different recipes of KT

Type of KT	Time (min)		
	0	300	0-300
	<----- mg/dL ----->		% Change
Standard KT	205 ± 34	204 ± 32	- 0.2 ± 4.6
Modified standard KT	202 ± 30	204 ± 27	1.5 ± 6.2
Black glutinous rice flour KT	195 ± 30	193 ± 24	- 0.2 ± 7.4
Mungbean starch KT	205 ± 32	203 ± 32	- 0.7 ± 6.7
Small sago KT	211 ± 37	205 ± 37*	- 2.6 ± 5.3
Glucose drink	197 ± 23	192 ± 25*	- 2.5 ± 5.0

* Significantly different from baseline

There was no significant difference between different recipes of KT

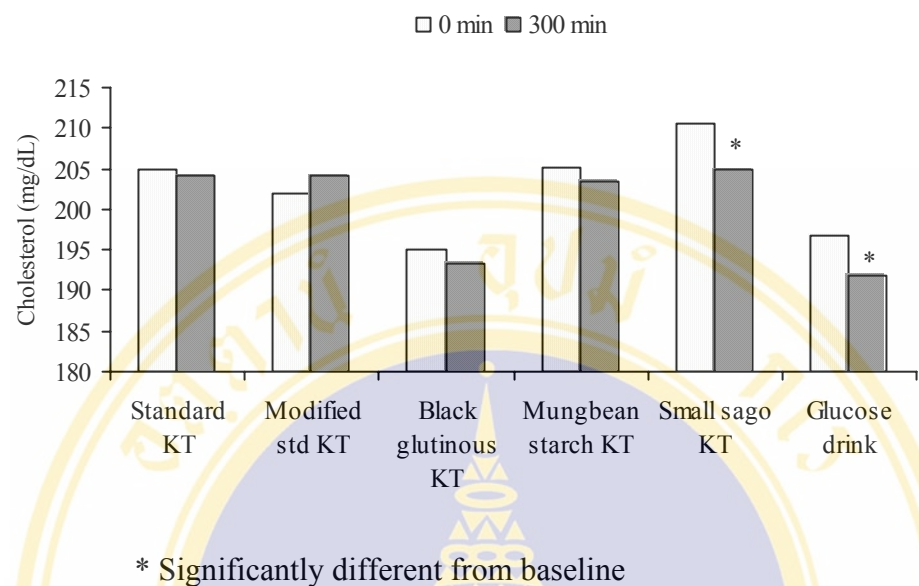
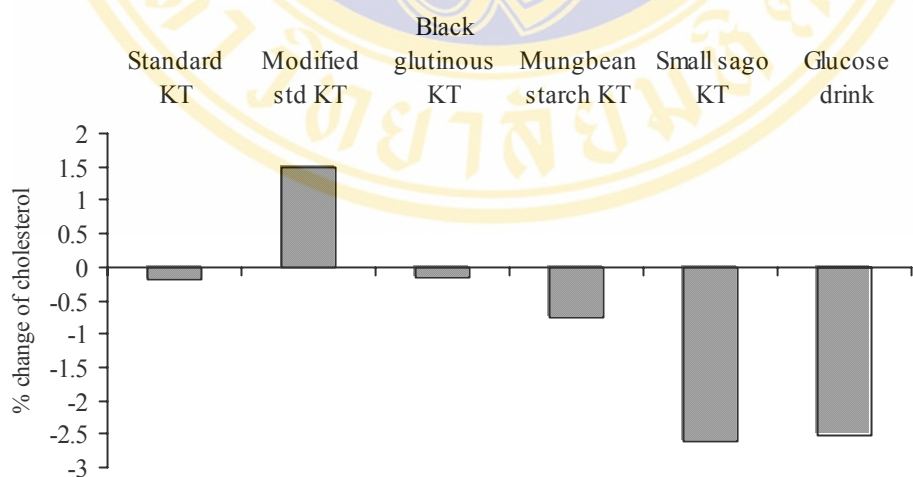


Figure 4.7 Mean cholesterol levels before and after ingestion of different recipes of KT



There was no significant difference between different recipes of KT

Figure 4.8 Mean percentage change of cholesterol after ingestion of different recipes of KT

Table 4.8 Comparison of serum HDL-C responses before and after ingestion of different recipes of KT

Type of KT	Time (min)		
	0	300	0-300
	<----- mg/dL ----->		% Change
Standard KT	43 ± 12	43 ± 11	0.4 ± 6.5
Modified standard KT	42 ± 12	42 ± 12	0.2 ± 8.6
Black glutinous rice flour KT	47 ± 14	46 ± 12	- 0.8 ± 9.2
Mungbean starch KT	41 ± 10	42 ± 10	3.5 ± 8.9
Small sago KT	43 ± 15	41 ± 12	- 2.2 ± 14.2
Glucose drink	43 ± 13	41 ± 13*	- 5.1 ± 9.1

* Significantly different from baseline

There was no significant difference between different recipes of KT

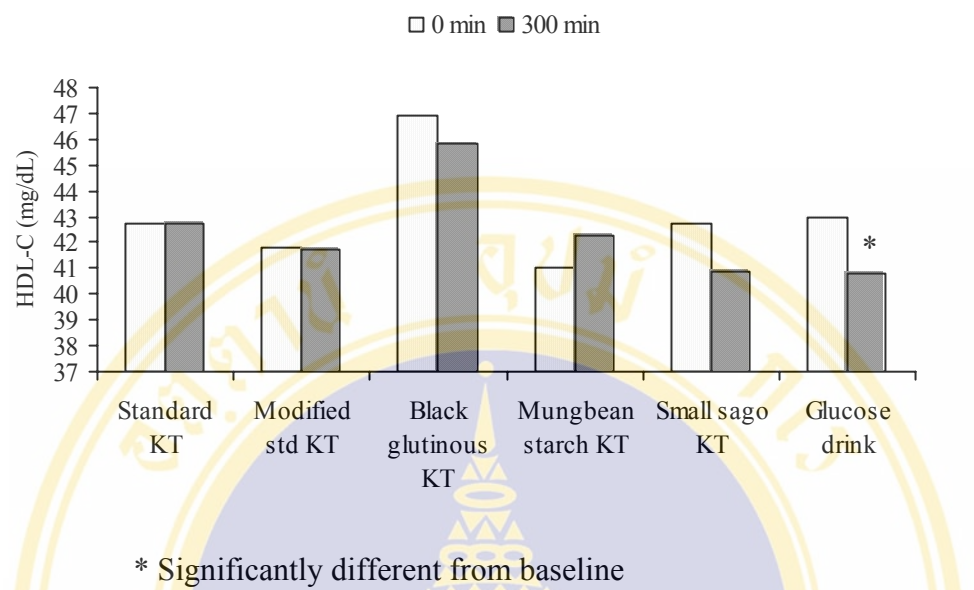
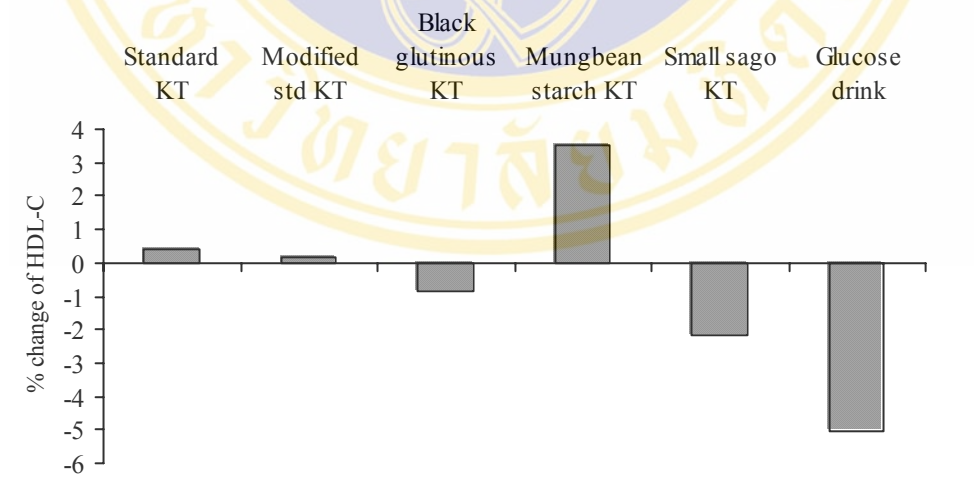


Figure 4.9 Mean HDL-C levels before and after ingestion of different recipes of KT



There was no significant difference between different recipes of KT

Figure 4.10 Mean percentage change of HDL-C after ingestion of different recipes of KT

Table 4.9 Comparison of serum LDL-C responses before and after ingestion of different recipes of KT

Type of KT	Time (min)		
	0	300	0-300
	<----- mg/dL ----->		% Change
Standard KT	126 ± 38	121 ± 35	- 3.8 ± 6.6
Modified standard KT	125 ± 38	118 ± 32*	- 3.7 ± 9.5
Black glutinous rice flour KT	117 ± 32	114 ± 29	- 0.3 ± 14.6
Mungbean starch KT	130 ± 30	125 ± 32	- 3.1 ± 17.6
Small sago KT	127 ± 32	125 ± 32	- 1.5 ± 10.6
Glucose drink	118 ± 30	114 ± 29	- 3.4 ± 9.9

* Significantly different from baseline

There was no significant difference between different recipes of KT

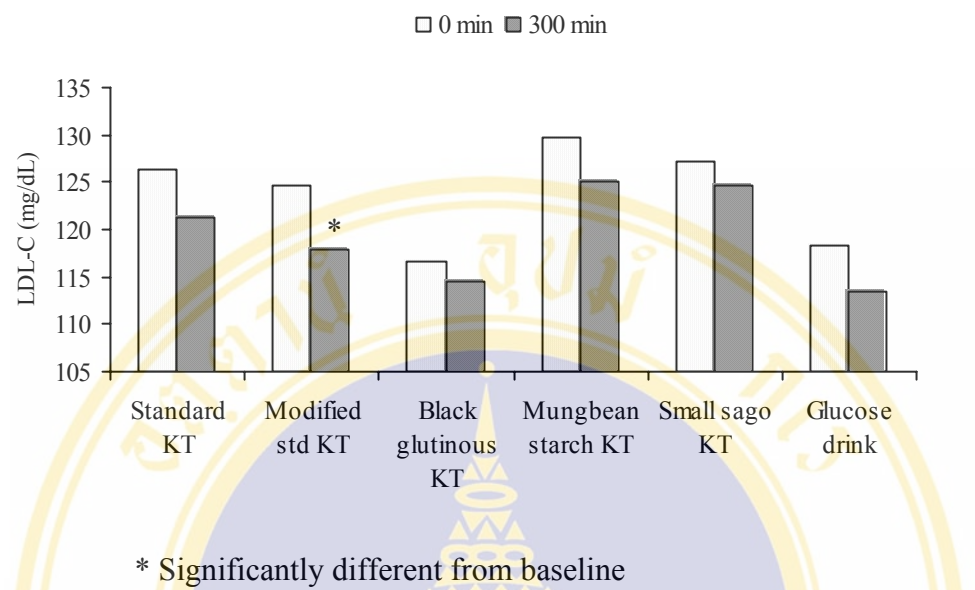
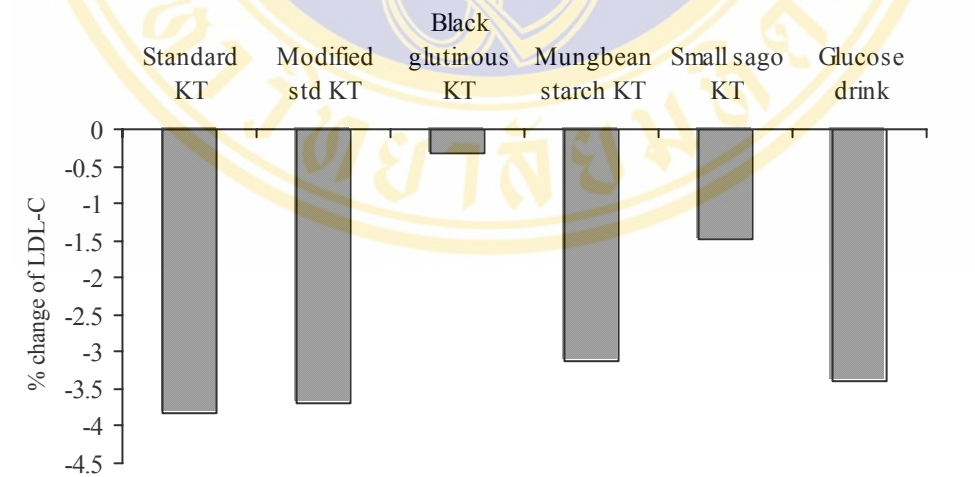


Figure 4.11 Mean LDL-C levels before and after ingestion of different recipes of KT



There was no significant difference between different recipes of KT

Figure 4.12 Mean percentage change of LDL-C after ingestion of different recipes of KT

Table 4.10 Comparison of blood viscosity (measured at shear rate of 264 s^{-1} , 37°C) before and after ingestion of different recipes of KT

Type of KT	Time (min)		
	0	60	300
	←----- cP ----->		
Standard KT	3.85 ± 0.37	3.65 ± 0.36*	3.88 ± 0.43
Modified standard KT	3.87 ± 0.48	3.66 ± 0.42*	3.77 ± 0.51
Black glutinous rice flour KT	3.80 ± 0.44	3.55 ± 0.42*	3.70 ± 0.39
Mungbean starch KT	3.80 ± 0.35	3.56 ± 0.34*	3.77 ± 0.31
Small sago KT	3.74 ± 0.48	3.57 ± 0.55*	3.64 ± 0.45*
Glucose drink	3.89 ± 0.48	3.63 ± 0.41*	3.79 ± 0.48
	←----- % Change ----->		
Standard KT	-	- 5.20 ± 3.15	0.75 ± 5.53
Modified standard KT	-	- 5.14 ± 3.56	- 2.34 ± 7.18
Black glutinous rice flour KT	-	- 6.45 ± 4.15	- 2.39 ± 5.62
Mungbean starch KT	-	- 6.14 ± 3.83	- 0.46 ± 5.24
Small sago KT	-	- 4.63 ± 4.86	- 2.48 ± 3.53
Glucose drink	-	- 6.43 ± 4.96	- 2.38 ± 4.85

* Significantly different from baseline

There was no significant difference between different recipes of KT

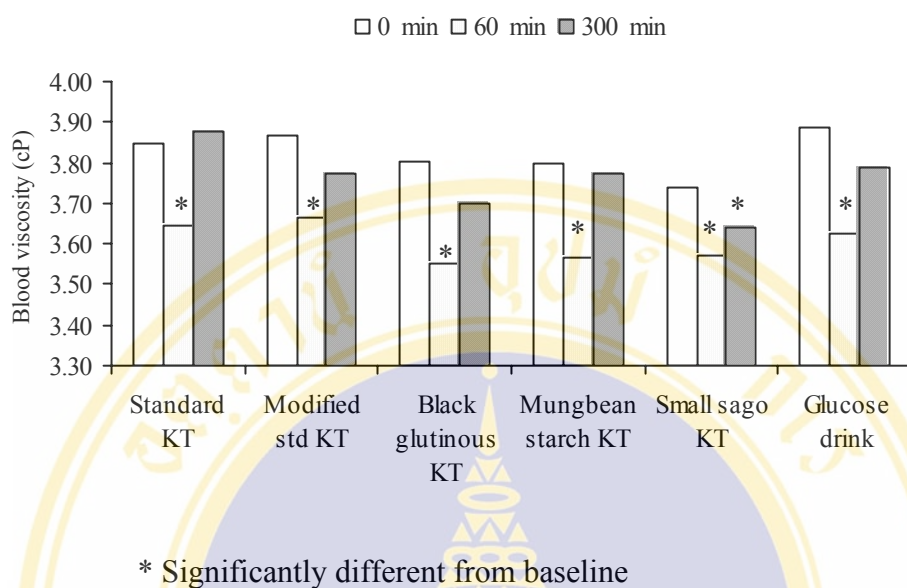
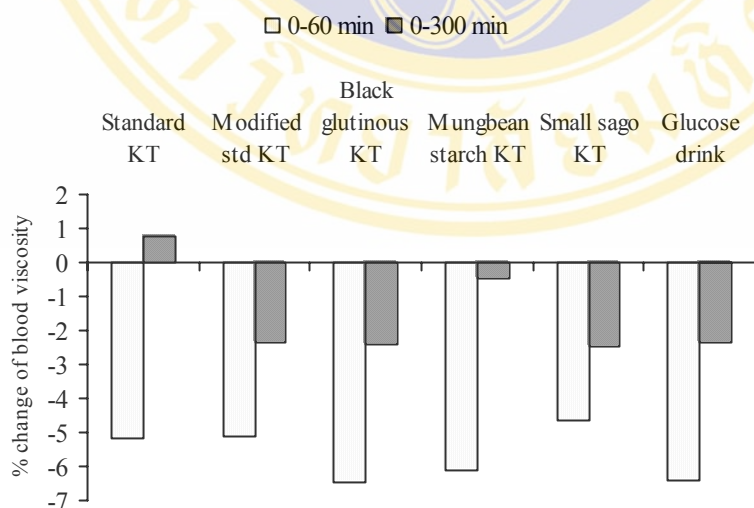


Figure 4.13 Mean blood viscosity (measured at shear rate of 264 s^{-1} , 37°C) before and after ingestion of different recipes of KT



There was no significant difference between different recipes of KT

Figure 4.14 Mean percentage change of blood viscosity (measured at shear rate of 264 s^{-1} , 37°C) at 60 and 300 min after ingestion of different recipes of KT

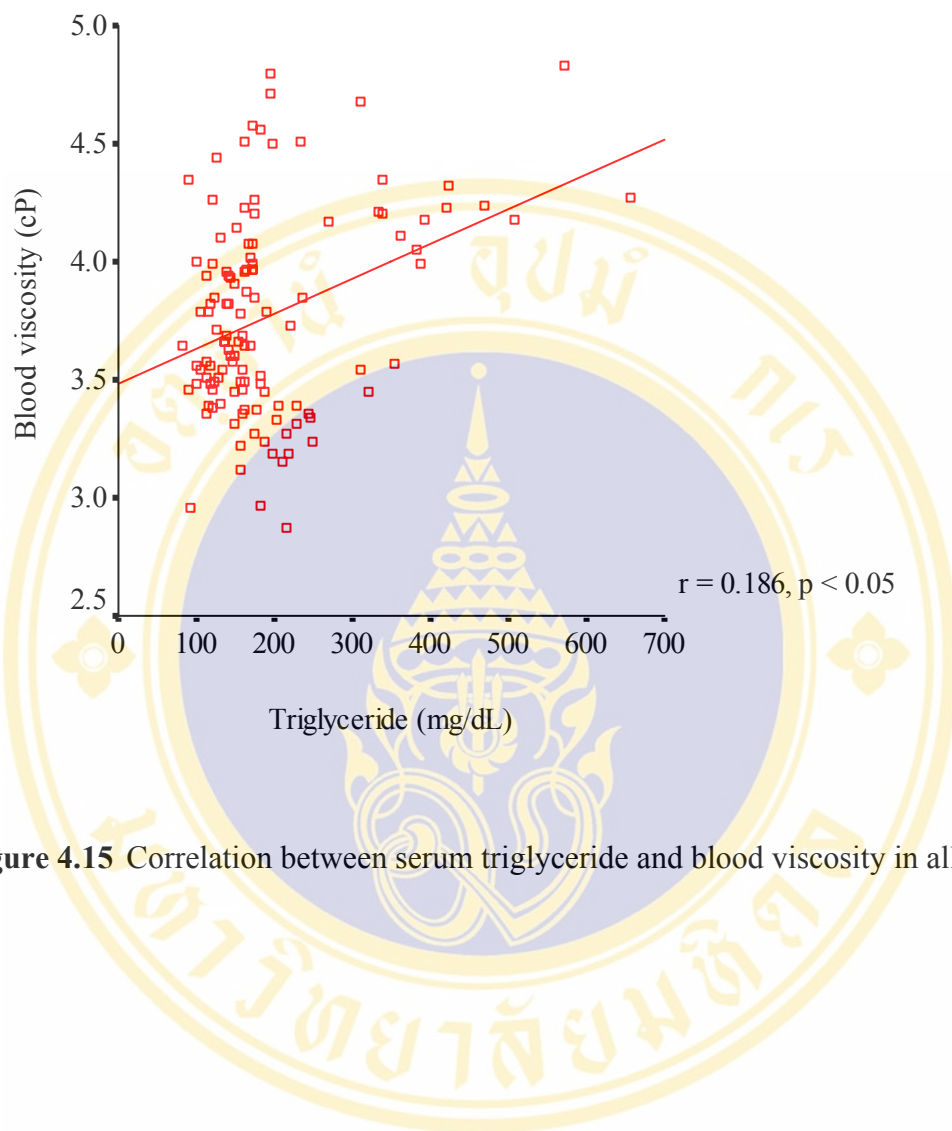


Figure 4.15 Correlation between serum triglyceride and blood viscosity in all subjects

Table 4.11 Taste preference of different recipes of KT by using the sensory evaluation scores (Mean $\pm$ SD)

Type of KT	Covering			Filling		Overall	Piece/serving
	Texture	Stickiness	Taste	Taste	Size		
Standard	3.9 $\pm$ 0.8	3.9 $\pm$ 0.9	3.9 $\pm$ 0.9	4.1 $\pm$ 0.9	4.0 $\pm$ 0.6	4.1 $\pm$ 0.9	2.7 $\pm$ 1.1
Modified standard	4.1 $\pm$ 0.7	4.3 $\pm$ 0.5	3.9 $\pm$ 0.8	4.1 $\pm$ 0.5	3.9 $\pm$ 0.6	4.2 $\pm$ 0.6	3.1 $\pm$ 1.1
Black glutinous rice flour	3.5 $\pm$ 0.7	3.8 $\pm$ 1.0	3.8 $\pm$ 0.8	4.1 $\pm$ 0.7	3.9 $\pm$ 0.4	4.0 $\pm$ 0.7	2.8 $\pm$ 1.0
Mungbean starch	2.9 $\pm$ 1.1	2.8 $\pm$ 1.3	3.0 $\pm$ 1.2	3.7 $\pm$ 0.6	3.5 $\pm$ 0.9	3.3 $\pm$ 1.2	2.4 $\pm$ 1.3
Small sago	3.7 $\pm$ 1.2	3.4 $\pm$ 1.2	3.7 $\pm$ 0.9	3.9 $\pm$ 0.7	3.8 $\pm$ 0.5	4.0 $\pm$ 0.9	2.7 $\pm$ 1.2

Use 5 point score scale 1 = very unpleasant, 2 = unpleasant, 3 = acceptable, 4 = good, 5 = very good

CHAPTER 5

DISCUSSION

In this study we wanted to choose the starch or flour which has a low GI to make a popular dessert for the diabetic. Khanom-Tian is the dessert which serves this purpose since it can be applied by using several types of starch or flour. In addition, its filling can also be modified by using less amount of sugar. Each KT had the same constituents.

Seventeen subjects took part in the study, 3 men and 14 women, mean ages 61 ± 9 and age ranging from 45 to 76 yrs. The mean body weight of subjects at the start of the study was 68.1 ± 19.1 kg, mean BMI was 27.9 ± 7 kg/m² ranging from 21.2 to 49.3 kg/m². Only 7 subjects were normal (BMI = 18.5-24.9), 6 subjects were overweight (BMI = 25-29.9) and 4 subjects were obese (BMI >30). The mean % body fat of subjects were 24.7 ± 8.5 for men and 36.1 ± 8.8 for women. Zhu et al (73) had demonstrated the mean % body fat cutoffs at BMI of 25 to be 20% for men and 31% for women. Therefore, our subjects both men and women had fat accumulation far beyond the healthy cutoffs (1/3 for men and 8/14 for women).

Furthermore, individual who have android obesity (64) with a waist to hip ratio 1.0 or greater in men and 0.8 in women have an increased risk of obesity-related disease. Our 16/17 subjects had android obesity.

Before entering the study, mean fasting plasma glucose (FPG) and lipid profile of subject were moderately high, (Table 4.2) only 3 subjects had FPG more than 200 mg/dL, 1 subject had triglyceride more than 500 mg/dL even though most of these subjects had been on medication to control their diseases.

5.1 Plasma Glucose Levels, AUC and GI

Basal fasting plasma glucose concentrations were not different between all subjects. After ingestion of all recipes of KT, plasma glucose response rose rapidly and reached the peaks at 60 min, the same as glucose drink. Peak glucose

concentrations after ingestion of all recipes of KT were significantly higher than baseline. Small sago KT elicited the largest glucose responses (largest AUC) among the 5 test KT (Table 4.5).

From Somnuk's study (74) postprandial plasma glucose response reached their peaks at various time periods, at about 30 min in most fruits –mango, pineapple, longan, guava (GI = 51, 45, 43 and 17) and at 60 min in durian and dragon fruit (GI = 39 and 37). However, after eating all KT the glucose peaks were present at 60 min. This might be due to the same rate of digestion. Since sucrose was the only source of sweetener used in these KTs. Different fruits were composed of different types of sugar which affected the rate of digestion and absorption causing the difference in the glucose peak time. Crapo et al (75) founded that different kinds of sugar created different plasma glucose responses both when given alone or as a constituent in a test meal.

5.2 Different Amount of Sugar and Different Types of flour

Modified stdKT had lower glycemic and low GI response than stdKT (Table 4.5). This was due to the lower sucrose content in the modified stdKT. Since modified stdKT had less sugar content than stdKT, while both of them contained 50 g CHO, and being wrapped with the same white glutinous rice flour. The content of sugar in modified stdKT was reduced from 22 g in stdKT to 12.5 g in modified stdKT and resulted in the reduction of GI from 72 to 61, respectively. This different result certainly caused by the reduction of sugar in spite of the presence of more CHO in covering flour (19 g VS 26.5 g). Lowering sugar can lower the GI.

Comparing the 4 modified KT recipes which had the same filling but had different kinds of covering flour would reflect the glucogenesis quality of such flours or starches. The highest GI of the modified KT was small sago followed by black glutinous rice flour KT, modified stdKT and the lowest GI was mungbean starch KT. Their GI were 82, 71, 61 and 57, respectively. These differences were mainly caused by the amylose content found in the starch or flour, mungbean starch had high amylose content (30%), small sago (cassava pearl) had medium amylose content (21%), and the glutinous rice flour a low amylose content (3-4%).

Behall et al (76) did a study in bread containing 30-70% amylose, the results indicated that a low GI (<55) can be obtained with about 30% amylose content. However the amylose content of the starch needed to be greater than 50% to significantly reduce plasma glucose and insulin in the subjects. The GI of the 30, 40, 50, 60 and 70% amylose breads were 46.7, 48.3, 43.3, 33.6, and 29.1%, respectively.

The mungbean starch KT had highest amylose content and thus it could be predicted to have the lowest GI. At first, we predicted that the small sago KT may have lower GI than glutinous rice flour KT, because it contained higher amylose content, but the result was in contrast, small sago KT gave the highest GI. KT from black and white glutinous rice flour gave lower GI than small sago. Thus, it could be said that, amylose content was not the only factor determining the GI.

GI values in our test KT ranged between 57-82%. The GI of 3 recipes of KT were classified as high GI foods (70 or higher) –small sago KT, stdKT and black glutinous rice flour KT which had GI of 82, 72 and 71, respectively. Two KTs were classified as medium GI foods (55-69) –modified stdKT (GI = 61) and mungbean starch KT (GI = 57). Modification of KTs by adjusting the sugar content and changing the flour or starch will be valuable ways to gain better glycemic control.

Another factor which may affect the glycemic response; e.g., Panlasigui et al (77) founded that rice varieties with similar amylose content and cooked under the same conditions differed in glycemic responses. Wolever et al (78) found that both the amount and source of carbohydrate affected the GI response in normal subjects. Physical form of carbohydrate and cooking method are other causes affecting GI as shown by Collier (79) and Fernandes (34), respectively.

The risks and benefits of sugar ingestion in metabolic studies of diabetes as well as the data on primary prevention, on long-term effects of sugar intake on risk of becoming type 2 diabetes have been controversial. There is no specific diet (80) for diabetics, but emphasis is placed on normalizing blood glucose levels. The American Diabetes Association (ADA) in its most recent dietary recommendations for people with diabetes stated that “In some individuals, modest amounts of sucrose and other refined sugars may be acceptable, contingent on metabolic control and body weight”.

5.3 Dessert and Sugar Content

In general, it has been thought that, the delicious dessert must be sweet and has high amount of sugar. In this study we have shown that modified KT which contained lower amount of sugar than the original KT (sugar from 22 g to 12.5 g) established the highest score in the sensory test (Table 4.11), modified stdKT also had lower GI than stdKT (61 VS 72) though they contained the same covering flour. So it was confirmed that amount of sugar affected the GI as shown by Wolever et al (78).

Comparison of the GI results of our dessert (KT) with other studies. In the study by Visavajarn (81), Tako dessert made from mungbean flour and rice flour were found to have a GI of 73.4 and 74.2, respectively. All of them were in the high GI rank foods, higher than GI of most of our KTs. The reason may come from the effect of the amount of sucrose. Mungbean Tako and rice Tako were made from 25 g of sucrose while our KT used only 12.5 g of sucrose. It had only a half amount of sugar compared with Tako dessert. In addition, the percentage of refined sugar in the whole serving of modified KT was only less than 20% as compared to Tako which occupied about 25%. Moreover, our KT contained only 250 Kcal per 1 serving of GI, while Tako had 385 Kcal. The lower energy and lower sugar content are better for diabetic patients.

This agrees with the results of Reiser et al (82) demonstrating that increasing levels of dietary sucrose 33% was significantly greater on glucose response than 5% and 18% in carbohydrate sensitive subjects. Other studies about sucrose, Wise et al and Peters et al (83, 84) concluded that when sucrose was fed to diabetic subjects as part of a snack it did not cause a greater rise in blood glucose than isocaloric amounts of starch-containing foods. Although the American Diabetes Association (ADA) recommended on CHO that the amount of CHO consumed is more important than its source, also concerning in both amount and type ought to be better than only one.

Our KTs used sucrose as the main source of sweetener. Sucrose is a disaccharide, which is broken down into glucose and fructose before absorption. Crapo et al (75) showed that fructose ingestion resulted in significantly lower serum glucose and insulin responses than did other sugar. Wolf et al (85) demonstrated in rats that the addition of a small amount of oral fructose or sucrose can reduce

postprandial glycemia. Therefore, if an appropriate amount of sugar (sucrose) is used it may lower the postprandial blood glucose response.

Fructose is often recommended for, and consumed by, people with diabetes mellitus or hypoglycemia, because it has a very low GI (GI = 32). The low GI is due to the unique and lengthy metabolic pathway of fructose, which involves phosphorylation and a multi-step enzymatic process in the liver. In humans, fructose (86) is absorbed largely intact into the portal blood and is almost totally cleared in a single passage through the liver. Thus, there is essentially no appreciable fructose in the blood. After a large oral dose of 1 g of fructose/kg body weight, the blood level will increase to 0.5 mmol/L in 30 min and then slowly decrease during the next 90 min. In the liver, fructose is phosphorylated by the abundant enzyme fructokinase into fructose-1-phosphate, which is cleaved by hepatic aldolase into glyceraldehydes and dihydroxyacetone phosphate (DHA phosphate). DHA phosphate is an intermediary metabolite in both the glycolytic and gluconeogenesis pathways. Glyceraldehyde, while not intermediary in either pathway, can be converted by various liver enzymes into glycolytic intermediary metabolites available to be metabolized ultimately to produce glycogen. This glycogen can then be broken down into glucose by glycogenolysis in a later state. Hence, ingestion of a small amount of fructose or sucrose should be beneficial to the glycemic response.

During cooking (34) starch granules take up water and swell (gelatinization), which irreversibly disrupts the crystalline structure of the starch, making it able to be readily hydrolyzed by amylase. When starch is cooled, it retrogrades, a process in which the starch molecules begin to reassociate with each other by hydrogen bonding forming an irregular structure that is resistant to digestion. A study done by Komindr et al (6) in several forms of CHO diet, they found that mungbean noodle (mungbean vermicelli) had the lowest GI (45%), compared with other CHO, e.g., glutinous rice, rice, wheat noodles, rice noodles, fine rice noodles (rice vermicelli) which had GI = 75, 71, 57, 54, and 53%, respectively. Retrogradation is the important process in mungbean noodle manufacturing (keep in freezing room), and affects the properties of mungbean noodle and resulted in lowering GI. Frei et al (87) recently demonstrated that the adaptation of processing by storing rice in the refrigerator for 24 h at a temperature of 4°C in order to induce a retrogradation effect led to a reduction of GI.

In case of mungbean starch used in our dessert which has high amylose and amylopectin ratio, it was kept only in the room temperature after cooking, but it also produced the lowest GI. This data indicate that mungbean starch may also undergo partial retrogradation or have other special properties which need further studies.

5.4 The Benefit of Mungbean Starch

The number of pieces of each KT being consumed to attain 50 g CHO in the GI measuring were not equal, 3 pieces for stdKT, 4 pieces for modified stdKT, black glutinous rice flour KT and small sago KT. However, 8 pieces of mungbean KT were required for GI determination. The sensory evaluation tests, revealed that in most KT only 3 pieces per serving was the most appropriate amount to be served. From these reasons the amount of CHO in mungbean starch KT to be consumed in general must be even lower; since 3 pieces of mungbean starch KT are equal to 1/3 of the amount served in the GI test (CHO ~ 16 g, 64 Kcal). This portion is close to 1 exchange of fruit (CHO = 15 g, 60 Kcal) and should be safe for postprandial glucose response in the diabetics.

5.5 Lipid Profile

Carbohydrate-containing foods are digested into glucose, which is then absorbed into the blood, and to the liver and other tissues. Liver and adipose tissue make triacylglycerol from glucose under conditions of abundant carbohydrate intake.

The effects of KT on mean serum lipids are summarized in Table 4.6-4.9. The starting values of lipids before ingestion of each KT were not significantly different in all subjects. Ingestion of all KT resulted in significant changes only triglyceride levels, but did not affect other lipid levels in this short-term study; whereas no change in triglyceride was seen after glucose drink.

Since sucrose is broken down to glucose and fructose, the difference in serum triglyceride level between KT and glucose drink should be due to fructose released after sucrose being digested. Fructose (88) by-passes the phosphofructokinase regulatory step of glycolysis, in which glucose can be converted to glycogen rather than entering the glycolytic pathway. As a result, fructose increases hepatic pyruvate and lactic acid production, activates pyruvate dehydrogenase, and shifts the balance

from oxidation to esterification of fatty acids, which can increase very low density lipoprotein synthesis.

Presumably, the amount of sugars and CHO in our KT were not high enough and the test time was too short to significantly affect cholesterol, HDL-C and LDL-C levels. It took Reiser et al (89), 5 wk in the hyperinsulinemic subjects to show that 20% of fructose diet compared with a high-amylose cornstarch diet resulted in significantly increased total triglycerides, total and very-low-density lipoprotein cholesterol.

Bantle et al (90) found that dietary fructose was associated with increased fasting and postprandial plasma triglyceride concentrations in healthy men, and had no effect on fasting plasma cholesterol, HDL-C and LDL-C in healthy subjects for 6 wk study. With these reasons if a portion of dessert or food being consumed is not overloading, it should not cause significant health problem.

5.6 Blood Viscosity

Our diabetic subjects did not have high blood viscosity, ranging from 3.74 to 3.89 cP (measured at shear rate of 264 s^{-1} , 37°C) at baseline (Table 4.10). Five hour after consuming all KT there was no increase in whole blood viscosity, whereas the viscosity was decreased at 60 min where the plasma glucose reached its peak. The reason was not clear. However, only stdKT showed increase in viscosity but it was not statistically significant. The effect of diet on the whole blood viscosity is unclear. The findings in other Thai dessert (Tako) and many fruits from studies of Visavajarn (81) and Somnuk (74) concurred with this study. Fifty grams of CHO in both desserts and fruits did not significantly affect blood viscosity after 5 h of ingestion (measured at shear rates of 225 s^{-1} and 450 s^{-1} , 37°C for Tako dessert and 132 s^{-1} , 37°C for fruits).

The peak plasma glucose of all recipes of KT occurred at 60 min after ingestion while, blood viscosity at 60 min after all KTs were significantly decreased from baseline. These show that blood glucose per se does not adversely affect blood viscosity.

There was a positive correlation between serum triglyceride and blood viscosity (Figure 4.15) which agrees with previous studies, Hoiegggen et al (56) demonstrated that whole blood viscosity was correlated positively to serum

triglyceride ($r = 0.51$, $p = 0.04$, measured at shear rate of 0.5 s^{-1} and $r = 0.54$, $p = 0.04$, measured at shear rate of 201 s^{-1} , carried out at 37°C). In our study, postprandial triglyceride at 5 h was significantly increased from fasting levels whereas blood viscosity was not statistically elevated. However, the actual level of triglyceride was only about 10 mg difference. It is likely that postprandial hypertriglyceridemia from our KT was not high enough to remarkably affect blood viscosity. This might be due to the small adjustment of sucrose content in these KT.



CHAPTER 6

CONCLUSION

Dietary management for diabetics should aim to normalize their blood glucose levels, lipid profile level. To achieve these goals, quantity and quality of diets must be considered according to individual clinical condition.

The results demonstrated that many factors were responsible, such as the type of flour, amylose content, the type and amount of sugars, the cooking process and other unknown reasons.

Modification of KT either sugar content or covering flour could alter the glycemic response in type 2 diabetics. KT with mungbean starch cover gave the lowest GI whereas that with small sago cover (cassava-pearl) gave the highest GI. Postprandial triglyceride levels were increased by all recipes of KT. There was a correlation between triglyceride and blood viscosity but after a single ingestion none of KT produced significant elevation of blood viscosity.

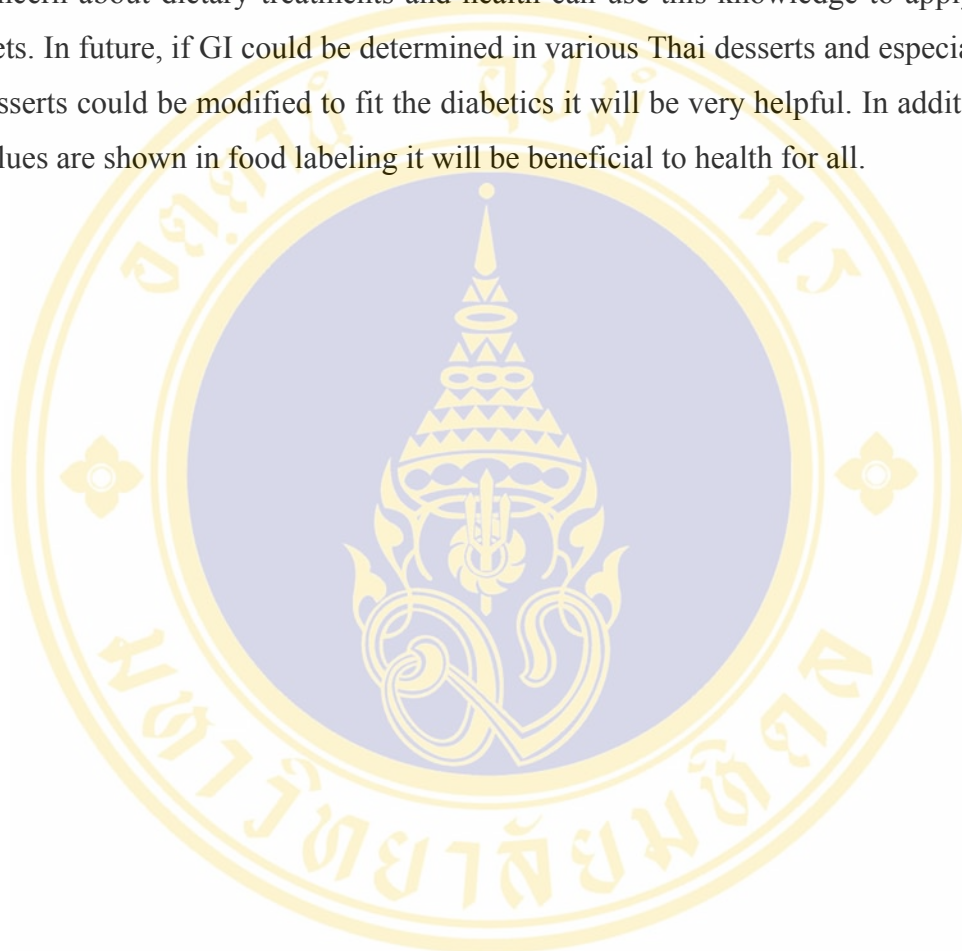
However, if diabetes patients are allowed to consume desserts or other sugar-containing diets, it should be substituted for other carbohydrate foods in the diet. To do such substitutions (a dessert exchange), information about the nutrient content is needed. The GI of each food is an important tool for making dietary recommendations. Thus, putting GI values on food labels is required as a tool for helping consumers in making proper foods selection.

6.1 Outcome and Benefit

The results from this study showed the expected and unexpected data on the type of flour or starch in our KT dessert. Knowledge from this study will help the health care personnels and diabetic patients making the proper dietary planning.

6.2 Recommendation for Future Research

In this study only four types of flour were used. There are many types of flour being used as diet constituents either as main ingredients or as supplements. If GI could be determined in all types of flour or starch, the dietitians and patients who are concern about dietary treatments and health can use this knowledge to apply in their diets. In future, if GI could be determined in various Thai desserts and especially if the desserts could be modified to fit the diabetics it will be very helpful. In addition, if GI values are shown in food labeling it will be beneficial to health for all.



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แบบสอบถามการรับประทานขนม ในผู้ที่มีปัญหาระดับน้ำตาลในเลือดสูง

- ❁ ข้อมูลนี้นำไปเพื่อเป็นแนวทางในการประเมินว่า ผู้ป่วยที่เป็นเบาหวาน มีความรู้เกี่ยวกับการรับประทานขนมอย่างไร
- ❁ ข้อมูลนี้จะเป็นความลับ ไม่นำไปเปิดเผยให้แพทย์และผู้อื่นทราบ และไม่มีผลต่อการรักษา

ขณะนี้ท่าน ☐ รับประทานยาเบาหวาน ☐ ฉีด insulin
☐ รับประทานยาเบาหวาน และฉีด insulin
☐ ไม่ได้รับประทานยาเบาหวาน และไม่ได้ฉีด insulin

ท่านทราบว่าเบาหวานมา ปี

- หากท่านมีปัญหาในเรื่องการควบคุมระดับน้ำตาลในเลือดและท่านได้รับคำแนะนำว่าไม่ควรรับประทานขนม ท่านปฏิบัติตัวอย่างไร
 - () ปฏิบัติตาม คือ ไม่รับประทานขนมเลย
 - () ไม่สามารถปฏิบัติตามได้ ยังคงรับประทานขนม
- หากท่านรับประทานขนม ส่วนใหญ่ท่านรับประทานขนมกี่วันใน 1 สัปดาห์
 - () ไม่รับประทานขนมเลย
 - () 1-2 วัน ต่อสัปดาห์
 - () 3-4 วัน ต่อสัปดาห์
 - () 4-5 วัน ต่อสัปดาห์
 - () รับประทานเป็นประจำทุกวัน
- หากท่านรับประทานขนม ส่วนใหญ่ท่านรับประทานขนมกี่ครั้งใน 1 วัน
 - () ไม่รับประทานขนมเลย
 - () 1 ครั้งใน 1 วัน
 - () 2 ครั้งใน 1 วัน
 - () 3 ครั้งใน 1 วัน
 - () มากกว่า 3 ครั้งใน 1 วัน

4. หากอนุญาตให้ท่านรับประทานขนมได้ ขนมชนิดใดที่ท่านชอบมากที่สุด

- () ขนมประเภทดัดน้ำตาล เช่น ถั่วเขียวต้มน้ำตาล, มันต้มน้ำตาล, เผือกต้มน้ำตาล
- () ขนมแงบวดจากพืช ผัก ผลไม้ต่างๆ เช่น กล้วยบวชชี, ถั่วดำแงบวด, ฟักทองแงบวด, เต้าส่วน
- () ขนมที่ทำจากแป้งต่างๆ เช่น ข้าวเกรียบปากหม้อ, ขนมกล้วย, บัวลอย, ขนมถั่วแปบ, ตะโก้, ขนมลิ่มกลิ้ง ขนมเทียน
- () ขนมอบที่ทำจากแป้งต่างๆ เช่น ขนมปัง ขนมเค้ก
- () อื่นๆ

5. ขอให้ท่านบอกชื่อของขนมที่ท่านชอบมากที่สุดและรองลงมาเป็นลำดับ

1.
2.
3.
4.

6. ถ้ามีทางเลือกให้ท่านรับประทานขนมได้ โดยท่านต้องทำขนมรับประทานเอง ท่านคิดว่าจะทำหรือไม่

- () ไม่ทำ เพราะไม่ชอบทำ
- () ไม่ทำ เพราะ สามารถดัดขนมได้เพื่อรักษาสุขภาพ
- () ทำ ถึงแม้จะยุ่งยาก
- () ทำ แต่ต้องเป็นขนมที่สามารถทำได้ง่าย
- () อื่นๆ

7. ถ้ามีขนมสำเร็จรูปสำหรับผู้ที่เป็นเบาหวานจำหน่าย ท่านมีความเห็นว่อย่างไร

- () ดี จะทดลองรับประทาน
- () ดี จะทดลองรับประทาน แต่จะต้องมีรายละเอียดอธิบายไว้อย่างชัดเจน ถูกต้อง เชื่อถือได้
- () ไม่ดี เพราะเชื่อว่าผู้ที่เป็นเบาหวาน ไม่ควรรับประทานขนม
- () อื่นๆ

8. ท่านคิดว่าท่านมีความรู้ในเรื่องเหล่านี้บ้างหรือไม่

8.1 น้ำตาลเทียม

() ไม่รู้จัก () รู้จักและเคยใช้ () รู้จักแต่ไม่เคยใช้

8.2 น้ำตาลฟรุกโตส

() ไม่รู้จัก () รู้จักและเคยใช้ () รู้จักแต่ไม่เคยใช้

8.3 ดัชนีน้ำตาล (glycemic index)

() ไม่รู้จัก () รู้จักและนำมาพิจารณาใช้ในการเลือกรับประทานอาหาร

() รู้จักแต่ไม่ได้นำมาพิจารณาใช้ในการเลือกรับประทานอาหาร

8.4 ใยอาหาร

() ไม่รู้จัก () รู้จักและนำมาพิจารณาใช้ในการเลือกรับประทานอาหาร

() รู้จักแต่ไม่ได้นำมาพิจารณาใช้ในการเลือกรับประทานอาหาร

ขอขอบคุณในความร่วมมือ

ผลการสำรวจการรับประทานขนม ในผู้ที่มีปัญหาระดับน้ำตาลในเลือดสูง (n = 30)

ข้อมูลของผู้ตอบแบบสอบถาม เป็นผู้ป่วยที่มารับการตรวจที่ คลินิกผู้ป่วยนอกแผนกอายุรกรรม และ เป็นโรคเบาหวาน ประเภทที่ 2

ข้อมูลของผู้ตอบแบบสอบถาม	ร้อยละ (n = 30)
รับประทานยาเบาหวาน	79.9 (24)
ฉีด insulin	6.7 (2)
รับประทานยาเบาหวาน และฉีด insulin	6.7 (2)
ไม่ได้รับประทานยาเบาหวาน และไม่ได้ฉีด insulin	6.7 (2)
หากท่านมีปัญหาในเรื่องการควบคุมระดับน้ำตาลในเลือดและท่านได้รับคำแนะนำว่าไม่ควรรับประทานขนม ท่านปฏิบัติตัวอย่างไร	
การปฏิบัติตามคำแนะนำของแพทย์	ร้อยละ (n = 30)
ปฏิบัติตาม คือไม่รับประทานขนมเลย	3.3 (1)
ไม่สามารถปฏิบัติตามได้ ยังคงรับประทานขนม	96.7 (29)
หากท่านรับประทานขนม ส่วนใหญ่ท่านรับประทานขนมกี่วันใน 1 สัปดาห์	
จำนวนวันที่รับประทานขนมใน 1 สัปดาห์	ร้อยละ (n = 30)
ไม่รับประทานขนมเลย	3.3 (1)
1-2 วัน ต่อสัปดาห์	56.7 (17)
3-4 วัน ต่อสัปดาห์	13.3 (4)
4-5 วัน ต่อสัปดาห์	6.7 (2)
รับประทานเป็นประจำทุกวัน	20 (6)
หากท่านรับประทานขนม ส่วนใหญ่ท่านรับประทานขนมกี่ครั้งใน 1 วัน	
จำนวนครั้งที่รับประทานขนมใน 1 วัน	ร้อยละ (n = 30)
ไม่รับประทานขนมเลย	3.3 (1)
1 ครั้ง	73.3 (22)
2 ครั้ง	20 (6)
3 ครั้ง	3.3 (1)
มากกว่า 3 ครั้ง	0 (0)

หากอนุญาตให้ท่านรับประทานขนมได้ ขนมชนิดใดที่ท่านชอบมากที่สุด

ชนิดของขนม	ร้อยละ (n = 30)
ขนมประเภทตม้น้ำตาล เช่น ถั่วเขียวตม้น้ำตาล, มันทม้น้ำตาล เผือกตม้น้ำตาล	16.7 (5)
ขนมแงบวดจากพืช ผัก ผลไม้ต่างๆ เช่น กล้วยบวชชี ถั่วดำแงบวด, ฟักทองแงบวด, เต้าส่วน	30 (9)
ขนมที่ทำจากแป้งต่างๆ เช่น ข้าวเกรียบปากหม้อ, ขนมกล้วย บัวลอย, ขนมถั่วแปบ, ตะโก้, ขนมลิ่มกลิ้ง ขนมเทียน	13.3 (4)
ขนมอบที่ทำจากแป้งต่างๆ เช่น ขนมปัง ขนมเค้ก อื่นๆ	23.3 (7) 16.7 (5)

อื่นๆ ได้แก่ ขนมใส่น้ำแข็ง, ไม่ชอบขนม, อะไรก็ได้, ไม่ชอบอะไรเป็นพิเศษ

ขอให้ท่านบอกชื่อของขนมที่ท่านชอบมากที่สุดและรองลงมาเป็นลำดับ

ลำดับที่ 1

ขนมกล้วย, ไอศกรีม, ขนมปัง, สาเกเปียก, ทองหยอด, ฟอยทอง, ถั่วเขียว, ถั่วดำ, ถั่วแดง, เค้ก, ขนม
เปียะ, บัวลอยไข่หวาน, สาเกไส้หมู, ข้าวเกรียบปากหม้อ, ขนมตาล, ถั่วดำน้ำกะทิ, ลูกก๊ี้, ฟักทอง
แงบวด, ข้าวเหนียวทุเรียน, หวานเย็น

ลำดับที่ 2

แดงไทยน้ำกะทิ, ข้าวตม้น้ำตาล, เค้ก, ขนมกล้วย, ขนมตาล, ฟักทองแงบวด, ข้าวเหนียวหน้าต่างๆ, วุ้น,
กล้วยบวชชี, ข้าวเหนียวมะม่วง, กล้วยทอด, เต้าส่วน, ขนมตม้น้ำตาล, เบเกอรี่, ขนมไส้ไก่, ถั่วเขียว
ตม้น้ำตาล

ลำดับที่ 3

ข้าวตม้น้ำตาล, เต้าส่วน, ถั่วเขียวตม้น้ำตาล, เผือก, มันทม้น้ำตาล, มันทเทศตม้น้ำตาล, ไอศกรีม, ถั่วกวน, สาเก
ไส้หมู, ขนมแงบวด, ซ่าหริ่ม, โคนัท, ขนมกล้วย

ลำดับที่ 4

ถั่วดำแงบวด, กล้วยเชื่อม, เบเกอรี่, โรตีสี, ลอยแก้ว, ไอศกรีม, กล้วยบวชชี

ถ้ามีทางเลือกให้ท่านรับประทานขนมได้ โดยต้องทำขนมรับประทานเอง ท่านคิดว่าจะทำหรือไม่

การทำขนมรับประทานเอง	ร้อยละ (n = 30)
ไม่ทำ เพราะไม่ชอบทำ	43.3 (13)
ไม่ทำ เพราะ สามารถดขนมได้เพื่อรักษาสุขภาพ	20 (6)
ทำ ถึงแม้จะยุ่งยาก	3.3 (1)
ทำ แต่ต้องเป็นขนมที่สามารถทำได้ง่าย	33.3 (10)
ถ้ามีขนมสำเร็จรูปสำหรับผู้ที่เป็นเบาหวานจำหน่าย ท่านมีความเห็นอย่างไร	
ขนมสำเร็จรูปจำหน่าย	ร้อยละ (n = 30)
ดี จะทดลองรับประทาน	30 (9)
ดี จะทดลองรับประทาน แต่จะต้องมีรายละเอียดอธิบายไว้	60 (18)
อย่างชัดเจน ถูกต้อง เชื่อถือได้	
ไม่ดี เพราะเชื่อว่าผู้ที่เป็นเบาหวาน ไม่ควรรับประทานขนม	10 (3)
ท่านคิดว่าท่านมีความรู้ในเรื่องเหล่านี้บ้างหรือไม่	
น้ำตาลเทียม	ร้อยละ (n = 30)
ไม่รู้จัก	16.7 (5)
รู้จักและเคยใช้	50 (15)
รู้จักแต่ไม่เคยใช้	33.3 (10)
น้ำตาลฟรุคโตส	ร้อยละ (n = 30)
ไม่รู้จัก	66.7 (20)
รู้จักและเคยใช้	10 (3)
รู้จักแต่ไม่เคยใช้	23.3 (7)
ดัชนีน้ำตาล (glycemic index)	ร้อยละ (n = 30)
ไม่รู้จัก	86.7 (26)
รู้จักและนำมาพิจารณาใช้ในการเลือกรับประทานอาหาร	3.3 (1)
รู้จักแต่ไม่ได้นำมาพิจารณาใช้ในการเลือกรับประทานอาหาร	10 (3)
ใยอาหาร	ร้อยละ (n = 30)
ไม่รู้จัก	23.3 (7)
รู้จักและนำมาพิจารณาใช้ในการเลือกรับประทานอาหาร	43.3 (13)
รู้จักแต่ไม่ได้นำมาพิจารณาใช้ในการเลือกรับประทานอาหาร	33.3 (10)

Table A 1 Nutrient composition of standard Khanom-Tian (stdKT) recipe

	Weight	Prot	Fat	CHO	Energy
	g	g	g	g	Kcal
Covering ingredients					
White glutinous rice flour	23	1.52	0.09	19.02	85.56
Sugar	12	-	-	12	48
Vegetable oil	1	-	1	-	9
Water	17	-	-	-	-
Vegetable oil for covering	1.5	-	1.5	-	13.5
Salt	0.2	-	-	-	-
Filling ingredients					
Mungbean dehulled	15	3.84	0.17	8.91	52.5
Sugar	10	-	-	10	40
Vegetable oil	2.5	-	2.5	-	22.5
White pepper	0.2	0.02	0.01	0.14	0.76
Salt	0.4	-	-	-	-
Total		5.4	5.3	50.1	270

Table A 2 Nutrient composition of modified stdKT recipe

	Weight	Prot	Fat	CHO	Energy
	g	g	g	g	Kcal
Covering ingredients					
White glutinous rice flour	32	2.11	0.13	26.46	119.04
Sugar	2.5	-	-	2.5	10
Vegetable oil	-	-	-	-	-
Water	30	-	-	-	-
Vegetable oil for covering	2	-	2	-	18
Salt	0.3	-	-	-	-
Filling ingredients					
Mungbean dehulled	20	5.12	0.22	11.88	70
Sugar	10	-	-	10	40
Vegetable oil	-	-	-	-	-
White pepper	0.2	0.02	0.01	0.14	0.76
Salt	0.6	-	-	-	-
Total		7.3	2.4	50.9	254

Table A 3 Nutrient composition of black glutinous rice flour KT recipe

	Weight	Prot	Fat	CHO	Energy
	g	g	g	g	Kcal
Covering ingredients					
Black glutinous rice flour	32	2.27	0.32	25.70	114.75
Sugar	2.5	-	-	2.5	10
Vegetable oil	-	-	-	-	-
Water	25	-	-	-	-
Vegetable oil for covering	2	-	2	-	18
Salt	0.3	-	-	-	-
Filling ingredients					
Mungbean dehulled	20	5.12	0.22	11.88	70
Sugar	10	-	-	10	40
Vegetable oil	-	-	-	-	-
White pepper	0.2	0.02	0.01	0.14	0.76
Salt	0.6	-	-	-	-
Total		7.4	2.6	50.2	254

Table A 4 Nutrient composition of mungbean starch KT recipe

	Weight	Prot	Fat	CHO	Energy
	g	g	g	g	Kcal
Covering ingredients					
Mungbean starch	29	0.03	0.03	26.19	105.27
Sugar	2.5	-	-	2.5	10
Vegetable oil	-	-	-	-	-
Water	260	-	-	-	-
Vegetable oil for covering	-	-	-	-	-
Salt	0.3	-	-	-	-
Filling ingredients					
Mungbean dehulled	20	5.12	0.22	11.88	70
Sugar	10	-	-	10	40
Vegetable oil	2	-	2	-	18
White pepper	0.2	0.02	0.01	0.14	0.76
Salt	0.6	-	-	-	-
Total		5.2	2.3	50.7	244

Table A 5 Nutrient composition of small sago KT recipe

	Weight	Prot	Fat	CHO	Energy
	g	g	g	g	Kcal
Covering ingredients					
Small sago	30	0.06	0.03	25.98	104.4
Sugar	2.5	-	-	2.5	10
Vegetable oil	-	-	-	-	-
Water	35	-	-	-	-
Vegetable oil for covering	2	-	2	-	18
Salt	0.3	-	-	-	-
Filling ingredients					
Mungbean dehulled	20	5.12	0.22	11.88	70
Sugar	10	-	-	10	40
Vegetable oil	-	-	-	-	-
White pepper	0.2	0.02	0.01	0.14	0.76
Salt	0.6	-	-	-	-
Total		5.2	2.3	50.5	244

Table A 6 Nutrient composition and energy distribution of each serving of KT

	StdKT	Modified stdKT	Black glutinous rice flour KT	Mungbean starch KT	Small sago (Cassava pearl) KT
CHO, g (%)	50.1 (74)	50.9 (80)	50.2 (79)	50.7 (83)	50.5 (83)
- sugar, g (%)	22.0 (33)	12.5 (20)	12.5 (20)	12.5 (20)	12.5 (20)
- flour, g (%)	19.0 (28)	26.5 (41)	25.7 (40)	26.2 (43)	26.0 (43)
- mungbean dehulled, g (%)	8.9 (13)	11.9 (19)	11.9 (19)	11.9 (20)	11.9 (20)
Protein, g (%)	5.4 (8)	7.3 (11.5)	7.4 (12)	5.2 (8.5)	5.2 (8.5)
Fat, g (%)	5.3 (18)	2.4 (8.5)	2.6 (9)	2.3 (8.5)	2.3 (8.5)
Energy (Kcal)	270	254	254	244	244
Pieces	3	4	4	8	4

Table A 7 Plasma glucose responses before and after ingestion of stdKT

Subject	Time (min)						
	0	30	60	120	180	240	300
	← mg/dL →						
1	128	191	225	164	115	92	86
2	119	139	213	160	121	90	70
3	169	234	281	321	292	279	240
4	115	213	222	121	96	90	86
5	141	178	267	218	168	124	92
6	150	204	193	175	191	169	128
7	241	288	389	414	323	252	187
8	133	175	182	139	114	108	110
9	141	187	254	213	153	110	81
10	94	232	290	263	218	162	137
11	160	252	294	220	164	117	106
12	126	148	180	139	123	108	105
13	232	299	350	292	258	241	223
14	150	265	297	285	195	171	139
15	128	164	214	211	178	132	106
16	94	130	144	137	85	77	77
17	132	218	274	245	205	182	144

Table A 8 Plasma glucose responses before and after ingestion of modified stdKT

Subject	Time (min)						
	0	30	60	120	180	240	300
	← mg/dL →						
1	135	205	214	142	114	96	90
2	126	178	209	142	126	112	86
3	121	180	267	272	240	213	200
4	133	220	254	159	115	88	92
5	187	225	290	243	204	164	137
6	166	225	229	187	218	166	137
7	177	222	323	362	254	180	141
8	117	157	187	133	110	103	101
9	88	141	189	117	76	58	72
10	128	229	296	236	211	159	124
11	142	157	261	189	177	133	108
12	96	99	135	94	99	81	85
13	200	241	287	258	223	202	175
14	162	223	281	245	209	150	173
15	234	256	323	270	261	245	232
16	126	146	195	171	105	72	72
17	166	249	308	249	231	205	184

Table A 9 Plasma glucose responses before and after ingestion of black glutinous rice flour KT

Subject	Time (min)						
	0	30	60	120	180	240	300
	← mg/dL →						
1	112	180	193	124	92	83	85
2	86	157	177	115	90	70	72
3	252	301	371	393	364	321	283
4	126	193	238	144	99	83	90
5	150	202	234	220	177	137	114
6	151	187	238	198	180	169	162
7	159	205	319	315	265	189	151
8	124	178	207	148	106	94	92
9	77	103	148	137	79	56	67
10	115	216	278	247	207	151	114
11	166	220	299	241	182	132	103
12	85	92	135	105	88	85	85
13	157	193	283	240	211	196	182
14	157	155	285	274	238	180	151
15	123	160	220	202	177	146	108
16	114	133	220	155	83	70	70
17	124	216	287	265	213	171	137

Table A 10 Plasma glucose responses before and after ingestion of mungbean starch KT

Subject	Time (min)						
	0	30	60	120	180	240	300
	← mg/dL →						
1	133	171	216	166	119	99	96
2	135	191	222	166	108	88	88
3	211	270	330	344	285	252	207
4	124	243	229	141	99	81	92
5	186	191	231	232	173	139	110
6	150	227	245	198	173	150	128
7	218	247	351	368	281	214	175
8	115	150	155	135	108	110	106
9	90	157	151	139	101	74	70
10	77	144	184	180	142	110	83
11	124	173	274	196	133	108	96
12	115	144	162	130	115	97	96
13	180	240	259	241	218	191	171
14	168	218	285	259	205	153	137
15	207	252	249	243	231	198	193
16	108	132	186	164	99	76	77
17	142	202	241	238	195	157	130

Table A 11 Plasma glucose responses before and after ingestion of small sago KT

Subject	Time (min)						
	0	30	60	120	180	240	300
	← mg/dL →						
1	124	160	198	162	119	88	90
2	103	168	211	141	112	105	86
3	99	151	252	247	232	171	159
4	121	189	218	144	105	79	81
5	177	204	279	270	222	168	132
6	160	227	256	209	193	180	155
7	166	267	369	387	328	252	184
8	97	146	159	144	124	110	103
9	144	196	245	225	180	141	101
10	88	198	229	209	193	169	144
11	150	214	263	139	130	105	97
12	96	135	142	108	96	99	92
13	196	250	301	283	241	241	213
14	169	214	296	247	223	198	162
15	173	218	252	256	231	207	168
16	85	177	128	146	90	72	76
17	126	213	265	250	227	207	175

Table A 12 Plasma glucose responses before and after drinking glucose

Subject	Time (min)						
	0	30	60	120	180	240	300
	← mg/dL →						
1	117	195	270	153	88	77	83
2	88	169	218	193	97	52	47
3	135	229	326	324	238	173	115
4	121	202	238	141	83	77	90
5	159	216	267	254	182	135	99
6	157	250	324	270	214	159	119
7	187	285	369	398	278	195	141
8	139	180	254	202	92	70	83
9	99	196	236	178	74	65	76
10	126	249	350	303	222	178	141
11	159	274	333	274	220	150	124
12	121	189	209	160	135	108	115
13	155	229	324	249	191	151	139
14	148	296	377	296	216	162	135
15	164	229	292	292	216	171	132
16	124	205	250	178	90	59	76
17	142	285	387	306	234	157	117

Table A 13 Lipid profile before and after ingestion stdKT

Subject	Triglyceride		Cholesterol		HDL-C		LDL-C	
	<————— mg/dL —————>							
	0	300	0	300	0	300	0	300
1	187	197	223	213	61	66	125	108
2	78	106	171	170	55	52	101	97
3	111	120	188	198	33	37	133	138
4	80	141	220	211	58	53	146	131
5	230	309	294	280	39	40	210	178
6	362	381	169	170	36	37	62	58
7	178	174	200	199	33	33	132	131
8	836	573	213	222	35	38	71	69
9	221	205	214	204	44	41	126	123
10	106	100	219	220	40	43	157	157
11	104	141	257	271	54	56	181	187
12	172	181	186	186	42	42	110	108
13	228	234	201	205	39	40	117	119
14	130	139	202	183	53	49	124	107
15	130	160	148	162	22	22	100	108
16	89	112	204	202	26	24	161	156
17	125	173	175	173	56	53	93	86

Table A 14 Lipid profile before and after ingestion modified stdKT

Subject	Triglyceride		Cholesterol		HDL-C		LDL-C	
	<————— mg/dL —————>							
	0	300	0	300	0	300	0	300
1	123	175	207	193	55	51	127	107
2	223	354	233	230	45	41	144	119
3	114	130	199	192	29	27	147	140
4	108	156	210	218	52	50	137	137
5	146	216	263	264	43	41	191	180
6	375	470	169	188	32	34	63	72
7	146	167	178	187	31	30	118	124
8	642	422	209	195	30	34	51	56
9	164	211	214	211	38	39	143	130
10	160	237	232	247	42	43	158	157
11	91	169	222	218	48	47	156	137
12	131	150	149	163	42	47	80	87
13	199	309	235	226	46	42	149	122
14	112	172	169	193	51	62	96	97
15	96	150	185	191	32	32	134	129
16	112	161	188	189	24	23	142	134
17	94	121	171	165	70	66	82	75

Table A 15 Lipid profile before and after ingestion black glutinous rice flour KT

Subject	Triglyceride		Cholesterol		HDL-C		LDL-C	
	<————— mg/dL —————>							
	0	300	0	300	0	300	0	300
1	188	89	181	173	78	62	66	93
2	69	90	140	169	43	45	83	107
3	133	163	217	192	41	36	151	123
4	120	162	209	201	51	44	134	125
5	128	157	165	167	48	48	92	88
6	316	338	185	178	38	38	85	73
7	137	173	181	184	43	45	111	105
8	583	388	219	194	49	53	69	64
9	157	181	233	236	39	40	163	160
10	104	156	194	202	46	50	128	121
11	98	123	245	233	66	62	160	147
12	169	183	174	175	41	46	100	93
13	124	120	239	244	57	59	157	160
14	127	135	214	206	56	51	132	128
15	118	127	167	178	21	23	123	130
16	108	123	181	189	21	21	139	144
17	107	137	170	167	60	56	88	84

Table A 16 Lipid profile before and after ingestion mungbean starch KT

Subject	Triglyceride		Cholesterol		HDL-C		LDL-C	
	<————— mg/dL —————>							
	0	300	0	300	0	300	0	300
1	161	169	239	226	64	60	143	132
2	112	221	202	192	47	45	132	103
3	104	134	194	200	31	31	142	142
4	58	99	206	193	54	49	140	124
5	211	320	264	264	36	35	187	166
6	272	361	171	165	31	33	86	60
7	143	143	199	193	36	34	135	131
8	709	270	270	229	36	41	86	134
9	189	216	170	180	34	39	98	98
10	100	138	208	217	42	43	146	146
11	97	144	244	263	50	58	175	177
12	144	113	163	143	42	37	92	83
13	108	125	215	227	42	47	153	156
14	130	150	180	176	43	46	111	101
15	111	146	193	207	30	33	141	144
16	131	172	194	203	25	27	143	142
17	127	161	176	179	55	60	96	87

Table A 17 Lipid profile before and after ingestion small sago KT

Subject	Triglyceride		Cholesterol		HDL-C		LDL-C	
	<————— mg/dL —————>							
	0	300	0	300	0	300	0	300
1	146	165	296	292	80	71	187	188
2	140	219	186	177	57	43	101	90
3	158	202	246	243	52	41	163	161
4	70	92	169	159	58	46	97	95
5	183	248	242	259	47	41	159	168
6	519	507	207	213	32	36	92	88
7	150	159	176	175	27	26	119	117
8	688	334	233	207	36	40	78	101
9	189	228	181	175	31	34	112	97
10	119	159	253	253	44	48	185	173
11	82	117	216	215	47	51	153	142
12	254	246	195	193	36	41	108	103
13	170	196	256	213	31	34	120	141
14	139	157	183	179	48	51	107	98
15	93	113	171	170	22	24	131	124
16	109	153	182	180	23	18	137	132
17	102	142	187	180	56	50	112	102

Table A 18 Lipid profile before and after drinking glucose

Subject	Triglyceride		Cholesterol		HDL-C		LDL-C	
	<————— mg/dL —————>							
	0	300	0	300	0	300	0	300
1	169	181	208	203	72	70	103	97
2	135	197	200	180	53	46	120	95
3	81	104	189	182	34	31	140	130
4	55	116	196	162	62	53	123	85
5	166	174	228	222	45	43	150	145
6	508	420	181	172	33	38	70	73
7	142	162	195	193	38	36	129	125
8	474	339	188	178	43	49	61	62
9	206	228	199	195	37	36	120	114
10	123	158	217	216	34	34	159	151
11	132	191	254	256	56	53	172	165
12	248	244	166	171	33	30	84	93
13	195	196	216	223	40	39	137	145
14	139	151	188	186	51	43	110	113
15	172	161	178	179	26	24	118	123
16	138	174	167	170	21	17	118	118
17	128	138	176	174	53	51	98	96

Table A 19 Blood viscosity (measured at shear rate of 264 s^{-1} , 37°C) before after ingestion stdKT

Subject	Time (min)		
	0	60	300
	← cP →		
1	4.50	4.32	4.50
2	3.55	3.49	3.54
3	3.70	3.51	3.46
4	4.02	3.57	3.63
5	3.57	3.52	3.54
6	3.99	3.81	4.05
7	4.12	3.75	4.20
8	4.23	4.17	4.83
9	3.39	3.22	3.39
10	3.72	3.34	4.00
11	3.69	3.34	3.82
12	3.49	3.43	3.52
13	4.42	4.27	4.51
14	4.29	3.99	3.96
15	3.31	3.16	3.46
16	3.48	3.36	3.51
17	3.94	3.73	3.97

Table A 20 Blood viscosity (measured at shear rate of 264 s^{-1} , 37°C) before and after ingestion modified stdKT

Subject	Time (min)		
	0	60	300
	<----- cP ----->		
1	4.75	4.41	4.26
2	3.70	3.39	3.57
3	3.96	3.54	3.40
4	3.55	3.39	3.12
5	3.46	3.28	2.87
6	4.14	4.10	4.24
7	4.15	3.79	4.08
8	4.26	4.29	4.32
9	2.98	2.94	3.15
10	3.72	3.39	3.85
11	3.33	3.33	3.64
12	3.42	3.39	3.31
13	4.72	4.33	4.68
14	3.93	3.72	3.97
15	3.45	3.31	3.45
16	4.15	3.81	4.23
17	4.08	3.85	3.99

Table A 21 Blood viscosity (measured at shear rate of 264 s^{-1} , 37°C) before and after ingestion black glutinous rice flour KT

Subject	Time (min)		
	0	60	300
	← cP →		
1	4.68	4.21	4.35
2	3.75	3.33	3.46
3	4.12	3.70	3.87
4	3.61	3.49	3.37
5	3.12	3.13	3.22
6	4.27	4.08	4.20
7	3.99	3.66	4.08
8	4.18	3.99	3.99
9	3.16	2.94	2.97
10	3.55	3.18	3.49
11	3.67	3.34	3.49
12	3.72	3.42	3.48
13	4.38	4.45	4.26
14	4.00	3.69	3.66
15	3.15	3.12	3.51
16	3.58	3.43	3.85
17	3.70	3.25	3.66

Table A 22 Blood viscosity (measured at shear rate of 264 s^{-1} , 37°C) before and after ingestion mungbean starch KT

Subject	Time (min)		
	0	60	300
	← cP →		
1	4.30	3.96	4.02
2	3.91	3.72	3.73
3	3.78	3.64	3.54
4	3.9	3.39	3.48
5	3.46	3.4	3.45
6	4.17	3.91	4.11
7	4.00	3.82	3.93
8	4.29	4.00	4.17
9	3.18	3.12	3.27
10	3.46	3.22	3.69
11	3.42	3.27	3.60
12	3.36	3.12	3.58
13	4.21	4.15	4.44
14	3.85	3.28	3.60
15	3.51	3.13	3.58
16	3.87	3.76	3.99
17	3.91	3.70	3.96

Table A 23 Blood viscosity (measured at shear rate of 264 s^{-1} , 37°C) before and after ingestion small sago KT

Subject	Time (min)		
	0	60	300
	← cP →		
1	4.19	3.74	3.97
2	3.49	3.21	3.19
3	3.45	3.19	3.33
4	3.23	2.94	2.96
5	3.24	3.03	3.24
6	4.36	4.03	4.18
7	3.51	3.67	3.69
8	4.38	4.29	4.21
9	3.32	3.22	3.31
10	3.58	3.45	3.54
11	3.46	3.24	3.48
12	3.30	3.15	3.34
13	4.83	5.16	4.71
14	3.96	3.67	3.78
15	3.36	3.39	3.36
16	3.71	3.51	3.66
17	4.17	3.78	3.94

Table A 24 Blood viscosity (measured at shear rate of 264 s^{-1} , 37°C) before and after drinking glucose

Subject	Time (min)		
	0	60	300
	← cP →		
1	4.63	4.35	4.56
2	3.88	3.42	3.19
3	3.91	3.79	3.79
4	3.51	3.36	3.39
5	3.37	3.18	3.27
6	4.12	4.14	4.23
7	3.70	3.48	3.64
8	4.29	4.21	4.35
9	3.39	3.36	3.39
10	3.31	3.00	3.36
11	3.78	3.31	3.79
12	3.43	3.43	3.36
13	4.90	4.30	4.80
14	4.59	3.78	4.14
15	3.48	3.31	3.49
16	3.94	3.66	3.85
17	3.82	3.57	3.82

แบบประเมินการยอมรับขนมเทียน

ผู้ตอบแบบสอบถาม เพศ () ชาย () หญิง อายุ ปี

ให้ใส่เครื่องหมาย ✓ ในช่องที่ต้องการ

ขนมเทียนทำจาก () แป้งข้าวเหนียวขาว () แป้งข้าวเหนียวดำ () แป้งถั่วเขียว
() สาเกเม็ดเล็ก () สูตรทั่วไป

	ดีมาก (5)	ดี (4)	พอใช้ (3)	ไม่ดี (2)	ไม่ดีเลย (1)
ลักษณะเนื้อแป้ง					
ความเหนียวของแป้ง					
รสชาติของแป้ง					
รสชาติของไส้					
ขนาดแต่ละลูก					
ในภาพรวมทั้งหมด					

ถ้าให้ท่านรับประทานเป็นอาหารว่าง 1 มื้อ ท่านคิดว่าท่านจะรับประทาน ลูก จึงจะเหมาะสม

ข้อเสนอแนะ

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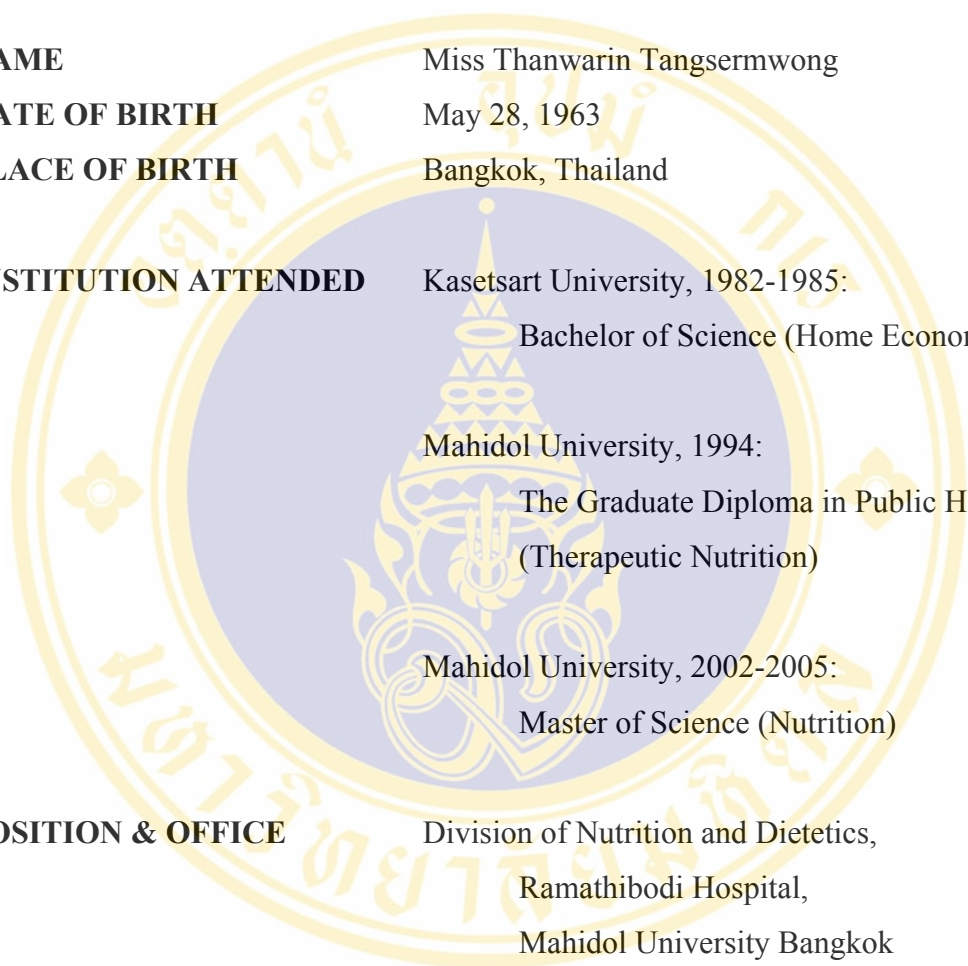
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ขอขอบคุณในความร่วมมือ

BIOGRAPHY



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