GLYCEMIC INDEXES OF TANGERINE, BANANA, PAPAYA, RAMBUTAN, POMELO, AND ROSE APPLE AND ACUTE RESPONSES OF SERUM LIPID AND BLOOD VISCOSITY IN DM TYPE 2 PATIENTS

PASINEE CHARTCHUATHAICHAROEN

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Pasinee Chartchuathaicharoen
....
Miss Pasinee Chartchuathaicharoen
Candidate

....
Prof. Surat Komindr, M.D., F.R.C.P. (T)
Major-Advisor

Manop Suphantharika
....
Assoc. Prof. Manop Suphantharika, Ph.D.
(Biochemical Engineering)
Co-Advisor

Salee Kiewkarnka
....
Asst. Prof. Salee Kiewkarnka, Ph.D
Acting Dean
Faculty of Graduate Studies

Emorn Wasantwisut
....
Assoc. Prof. Emorn Wasantwisut, Ph.D.
Chair
Master of Science Programme in Nutrition
Faculty of Medicine, Ramathibodi Hospital and Institute of Nutrition

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on
May 31, 2006

Pasinee Chartchuathaicharoen
....
Miss Pasinee Chartchuathaicharoen
Candidate

Surat Komindr, MD.
....
Prof. Surat Komindr, M.D., F.R.C.P. (T)
Chair

Manop Suphantharika
....
Assoc. Prof. Manop Suphantharika,Ph.D.
(Biochemical Engineering)
Member

Pongamorn Bunnag
....
Assist. Prof. Pongamorn Bunnag,
M.D., F.R.C.P. (T)
Member

Emorn Wasantwisut
....
Assoc. Prof. Emorn Wasantwisut, Ph.D.
Director
Institute of Nutrition
Mahidol University

Salee Kiewkarnka
....
Asst. Prof. Salee Kiewkarnka, Ph.D
Acting Dean
Faculty of Graduate Studies
Mahidol University

....
Prof. Rajata Rajatanavin, M.D., F.A.C.E.
Dean
Faculty of Medicine,
Ramathibodi Hospital
Mahidol University

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Pasinee Chartchuathaicharoen

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PASINEE CHARTCHUATHAICHAROEN 4536920 RANU/M
M.Sc. (NUTRITION)
THESIS ADVISORS: SURAT KOMINDR, M.D., F.R.C.P.(T)
MANOP SUPHANTHARIKA, Ph.D. (BIOCHEMICAL ENGINEERING)

ABSTRACT

Dietary control is necessary for diabetic patients to reduce the risk of complications. The consumption of fruits to substitute for desserts has been encouraged because of the resulting increased intake of fiber, micronutrient, and antioxidants. However, fruits contain various types and amounts of carbohydrate which variously affect blood glucose, and lipids. This study determined the acute responses of blood glucose, lipids and viscosity after ingestion of various amounts and types of fruits and also their glycemic indexes.

Sixteen DM type 2 patients were randomly divided into 2 groups to consume different test fruits. The test fruits consisted of 30 g and 50 g CHO of tangerine, rambutan, pomelo, rose apple, banana (Kai variety), and papaya, and 50 g glucose drink. All subjects underwent 6 tests of fruit consumption and a 50 g glucose drink. On the test day, each subject underwent an overnight fast and consumed one kind of test fruits. Baseline and postprandial blood samples were collected to determine plasma glucose, serum lipids and blood viscosity.

The results showed that bananas gave the lowest plasma glucose response curve in 30 g and 50 g CHO. The mean percentage change of plasma glucose and area under the glucose response curve (AUC) after eating 30 g CHO of tangerines tended to be higher than other fruits. Comparing the different amounts of CHO, a significant difference of AUC was seen in rambutan, pomelo, banana, and papaya. Glycemic indexes of banana, tangerine, papaya, rambutan, rose apple, and pomelo were 26, 30, 38, 47, 50, and 59, respectively. The mean triglyceride levels at 5 h after ingestion of all kinds of fruits tended to be increased from baseline especially both doses of tangerine, rambutan, and pomelo and 50 g CHO of banana. Total cholesterol levels did not change after ingestion of different amounts and kinds of fruits. The HDL-C levels were significantly decreased after ingestion of both doses of pomelo and 50 g CHO of rambutan. The LDL-C levels were significantly decreased after ingestion of 50 g CHO of rambutan, papaya, and pomelo. The percentage reduction of LDL-C was significantly larger after the 50 g CHO of pomelo than the 30 g CHO dose. There was no significant increase of whole blood viscosity at 5 hr after ingestion of different amounts of these fruits.

It can be mentioned that hyperlipidemic type 2 DM may ingest either three servings (50 g CHO) of rose apple or banana or papaya at a time without causing deleterious effect on blood glucose, serum lipids, and whole blood viscosity. However, high doses (50 g CHO) of tangerine, rambutan, and pomelo may produce hyperglycemia and dyslipidemia.

KEYWORDS : FRUIT/ DIABETES/ HYPERLIPIDEMIA/ BLOOD VISCOSITY

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ดัชนีน้ำตาลของส้ม, กล้วย, มะละกอ, เงาะ, ส้มโอ และ ชมพู่ และการตอบสนองของไขมัน และความหนืดเลือดในผู้ป่วยเบาหวานชนิดที่ 2 (GLYCEMIC INDEXES OF TANGERINE, BANANA, PAPAYA, RAMBUTAN, POMELO, AND ROSE APPLE AND ACUTE RESPONSES OF SERUM LIPID AND BLOOD VISCOSITY IN DM TYPE 2 PATIENTS)

ภาสินี ชาติเชื้อไทยเจริญ 4536920 RANU/M
วท.ม. (โภชนศาสตร์)
คณะกรรมการควบคุมวิทยานิพนธ์: สุรัตน์ โคมินทร์, M.D., F.R.C.P. (T), มานพ สุพรรณธริกา, Ph.D. (BIOCHEMICAL ENGINEERING)

บทคัดย่อ

การควบคุมอาหารเป็นสิ่งจำเป็นสำหรับผู้ป่วยเบาหวานเพื่อลดความเสี่ยงต่อการเกิดโรคแทรกซ้อน ผู้ป่วยได้รับการแนะนำให้รับประทานผลไม้แทนขนมหวานเพื่อช่วยควบคุมเบาหวาน, เพิ่มใยอาหาร, สารอาหาร และสารต้านอนุมูลอิสระ อย่างไรก็ตามในผลไม้มีองค์ประกอบของคาร์โบไฮเดรตหลายชนิดในสัดส่วนที่แตกต่างกันซึ่งส่งผลต่อระดับน้ำตาล และไขมันในเลือด ได้แตกต่างกันด้วย การศึกษานี้จึงต้องการศึกษาการตอบสนองของน้ำตาล, ไขมัน และความหนืดของเลือดภายหลังจากการรับประทานผลไม้ชนิดต่างๆในปริมาณที่ต่างกัน รวมทั้งหาค่าดัชนีน้ำตาลของผลไม้เหล่านี้ด้วย อาสาสมัครเป็นผู้ป่วยเบาหวานชนิดที่ 2 จำนวน 16 คน ถูกแบ่งออกเป็น 2 กลุ่มเท่าๆกัน โดยกลุ่มที่ 1 รับประทานส้ม, เงาะ, ส้มโอ กลุ่มที่ 2 รับประทานชมพู่, มะละกอ, กล้วย ที่มีปริมาณคาร์โบไฮเดรต 30 และ 50 กรัม และทุกคนดื่มน้ำตาลกลูโคสปริมาณ 50 กรัม เพื่อใช้ประเมินหาค่าดัชนีน้ำตาล อาสาสมัครงดอาหาร 10 ชั่วโมงจนถึงเช้า จากนั้นถูกเจาะเลือดที่เวลาเริ่มต้นก่อน และหลังจากรับประทานผลไม้แล้วที่เวลา 30, 60, 120, 180, 240 และ 300 นาที

ผลการศึกษาพบว่าพื้นที่ใต้กราฟของการเปลี่ยนแปลงระดับน้ำตาลในเลือดภายหลังการรับประทานกล้วยที่มีปริมาณคาร์โบไฮเดรต 30 และ 50 กรัมน้อยกว่าผลไม้ชนิดอื่นในขนาดเดียวกัน การเปลี่ยนแปลงดังกล่าวภายหลังการรับประทานส้มที่มีปริมาณคาร์โบไฮเดรต 30 กรัมมีแนวโน้มสูงกว่าผลไม้ชนิดอื่น และการเปลี่ยนแปลงดังกล่าวภายหลังการรับประทาน ส้มโอ, เงาะ, กล้วย และมะละกอที่มีปริมาณคาร์โบไฮเดรต 50 กรัมสูงกว่าผลไม้ชนิดเดียวกันที่มีปริมาณคาร์โบไฮเดรต 30 กรัม ค่าดัชนีน้ำตาลของกล้วย, ส้ม, มะละกอ, เงาะ, ชมพู่ และส้มโอเท่ากับ 26, 30, 38, 47, 50 และ 59 ตามลำดับ ระดับไตรกลีเซอไรด์มีแนวโน้มสูงขึ้นภายหลังจากการทานผลไม้ทุกชนิดโดยเฉพาะส้ม, เงาะ, ส้มโอที่มีปริมาณคาร์โบไฮเดรต 30 และ 50 กรัม และกล้วยที่มีปริมาณคาร์โบไฮเดรต 50 กรัม ไม่พบความแตกต่างของระดับโคเลสเตอรอลในเลือดภายหลังจากการรับประทานผลไม้แต่ละชนิดแม้ในปริมาณที่แตกต่างกัน ภายหลังการรับประทานส้มโอที่มีปริมาณคาร์โบไฮเดรต 30 และ 50 กรัม และเงาะที่มีปริมาณคาร์โบไฮเดรต 50 กรัมพบว่ามีการลดลงของระดับเอช ดี แอล โคเลสเตอรอลอย่างมีนัยสำคัญ ระดับแอล ดี แอล โคเลสเตอรอลลดลงภายหลังการรับประทานมะละกอ, เงาะ และส้มโอที่มีปริมาณคาร์โบไฮเดรต 50 กรัม นอกจากนี้ยังพบว่าระดับแอล ดี แอล โคเลสเตอรอลภายหลังการรับประทานส้มโอที่มีปริมาณคาร์โบไฮเดรต 50 กรัมลดลงมากกว่าที่เกิดจากปริมาณคาร์โบไฮเดรต 30 กรัม ถึงแม้ว่าจะมีการเพิ่มขึ้นของระดับน้ำตาลและไตรกลีเซอไรด์ แต่ไม่พบการเพิ่มขึ้นของระดับความหนืดของเลือดภายหลังการรับประทานผลไม้แต่ละชนิดและปริมาณที่ต่างกัน จากการศึกษานี้แสดงให้เห็นว่าผู้ป่วยเบาหวานชนิดที่ 2 สามารถรับประทานชมพู่, กล้วย และมะละกอได้ 3 ส่วน โดยไม่ทำให้เกิดผลเสียต่อระดับน้ำตาล,ไขมัน และความหนืดของเลือด แต่เราควรหลีกเลี่ยงการรับประทานส้ม, เงาะ และส้มโอขนาด 3 ส่วน เพราะอาจทำให้เกิดภาวะระดับน้ำตาลขึ้นสูง และระดับไขมันผิดปกติ

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LIST OF ABBREVIATIONS

Acetyl CoA	Acetyl coenzyme A
ADA	American diabetes mellitus
AUC	Areas under the curve
BMI	Body mass index
BW	Body weight
cP	Centipoise
º C	Celsius
CAD	Coronary artery disease
CHD	Coronary heart disease
CHO	Carbohydrate
cm	Centimeter
CVD	Cardiovascular disease
D	Shear stress
DF	Total dietary fiber
EDTA	Ethylenediamine tetraacetic acid
F	Fructose
F-1-P	Fructose-1-phosphate
F-6-P	Fructose-6-phosphate
F-1,6-P	Fructose-1,6-phosphate
g	Gram
G-6-P	Glucose-6-phosphate
G	Glucose
GI	Glycemic index
GL	Glycemic load
Glycerol-3-P	Glycerol-3-phosphate
HbA1C	Hemoglobin A 1 C
Hct	Hematocrit

LIST OF ABBREVIATIONS (CONTINUED)

HDL-C	High density lipoprotein-cholesterol
h	Hour
IHD	Ischemic heart disease
IGT	Impaired glucose tolerance
ISF	Insoluble fiber
Kcal	Kilocalory
Kg	Kilogram
m	Meter
MI	Myocardial infarction
min	Minute
mmol	Millimole
µg	Microgram
µL	Microliter
mg	Milligram
mg/dL	Milligram per deciliter
mm	Millimeter
mPa.s	Milli pascal second
MUAC	Mid upper arm circumference
NEFAs	Nonesterified fatty acids
NIDDM	Non-insulin dependent diabetes mellitus
nm	Nanometer
S	Sucrose
SD	Standard deviation
Sec^{-1}	Per second
SF	Soluble fiber
TC	Total cholesterol
TG	Triglyceride
TSF	Tricep skinfold thickness
U	Unit

LIST OF ABBREVIATIONS (CONTINUED)

UAMC	Upper arm muscle circumference
VLDL	Very low density lipoprotein
WHO	World health organization
WBV	Whole blood viscosity
η	Viscosity
τ	Shear rate

CHAPTER I
INTRODUCTION

Diabetes mellitus is a group of metabolic disorders and associated with severe microvascular complications (e.g., kidney disease and eye disease) and macrovascular complications (e.g., stroke and ischemic heart disease) (1-4). These complications can result in severe long-term complications (e.g., amputation, disability, and blindness) and account for a substantial economic burden (5).

Most diabetes patients have health problems or risk factors such as high blood pressure and dyslipidemia (increased of triglyceride, cholesterol, very low density lipoprotein and decreased high density lipoprotein) that increase the risk for heart disease and stroke. Diabetes is associated with a 2- to 4- fold increased risk of coronary heart disease (CHD) (6). Additionally, diabetic patients have an increased case fatality rate after myocardial infarction than in non-diabetic subjects and cardiovascular complications occur at an earlier age and often result in premature death (7). According to the Framingham Heart Study (8) cardiovascular disease (CVD) incidence rate among diabetic patients was 146.9 per 10,000 person-year and among participants without diabetes were 54.3 per 10,000 person-year in the 1977-1995.

The cause of diabetes mellitus is from the inadequacy of insulin hormone (9-11). In the absence of sufficient insulin, glucose cannot cross the cell membrane and accumulates in the blood, leading to hyperglycemia and glucosuria. Insulin also affects the performance of the intracellular lipase and lipoprotein lipase, by inhibiting the activity of intracellular lipase which breaks down triglycerides in adipose tissue (12, 13) into glycerol and non esterified fatty acids (NEFAs) and by stimulating the activity of lipoprotein lipase which breaks down very low density lipoprotein (VLDL) triglycerides. Therefore, either insulin resistance or deficiency can lead to increased circulating NEFAs released from the adipose tissues resulting in over production of hepatic triglycerides and over secretion of VLDL from the liver and defective VLDL lipolysis which leads to hyperlipidemia and consequently atherosclerosis and ischemic heart disease (IHD). There have been several reports relating blood viscosity to pathogenesis of ischemic heart disease (14-26). Several studies (20-22) also suggested that diabetic patients which had insulin resistance and hyperlipidemia had high blood viscosity and were associated with a high risk of atherosclerosis and ischemic heart disease. Blood viscosity is an important determinant of blood flow (14-17, 25) and the rate of oxygen transport and delivery in the circulation and coronary blood flow. If there is a decrease in the blood flow from hyperviscosity, this could worsen the hypoxia and further accelerate the process of vascular occlusion (18, 19).

Dietary control is necessary for diabetic patients to gain good diabetic control (10, 11, 27). The American Diabetes Association (ADA) (28) has suggested that the carbohydrate intake should be up to 55-66 % of total calories and food containing complex carbohydrate with fibers should be substituted for highly refined carbohydrates. In Thai society, one of the major sources of dietary carbohydrate is fruit. The carbohydrates of the fruits include monosaccharides (glucose, fructose), disaccharide (sucrose) and complex carbohydrates (starch and non starch) (29). Several studies (30-33) indicated that fruits can protect hypertention, stroke, cancer and cardiovascular disease; however, various amount of simple carbohydrates in the fruits may result in worsening of diabetic control. Though fructose is absorbed more slowly from the gastrointestinal tract than glucose (34, 35) but it can elevate serum triglycerides (36-39).

Different foods containing the same amount of carbohydrates may give a different glycemic response. For this reason the glycemic index (GI) of food has been proposed as the ratio of blood glucose area under the curve of tasted food to that of a reference food (40-42). The United Nation Joint Food and Agriculture Organizations do recognize and promote the use of GI in diabetes dietary management (43). The Diabetes Nutrition Study Group of the European Association for the Study of Diabetes states “carbohydrate containing foods which are rich in dietary fiber or have a low glycemic index are especially recommended” (43, 44). Low glycemic index diets have been shown to lower urinary c-peptide excretion in healthy subjects, improve glycemic control in diabetic subjects, and reduce serum lipids in hyperlipidemic subjects. The consumption of low glycemic index diets has been associated with higher HDL-cholesterol concentrations and in large cohort studies, with decreased risk of developing diabetes, cardiovascular disease and other complications (45-47). Glibertson et al. (48) have recently shown that the use of low glycemic index foods improved HbA_{1c} levels without increasing the risk of hypoglycemia in children with diabetes and that using a food pyramid based dietary instruction method enabled the children to successfully choose a low GI diet without impacting the dietary quality of the children's diet, nor their perception that their food choices were more limited. Komindr et al (49) suggested that in addition to strict dietary control, ingestion a low glycemic diet (mungbean noodle) without increasing fiber intake, can improve diabetic control and protein conservation in type 2 diabetes.

Thus, with the evidence supporting the value of low GI foods in the management of diabetic or hyperglycemic patients it is important to know the glycemic indexs and the response of blood after ingestion of fruits. So this study determined the glycemic index of Thai fruits and response of blood lipids and blood viscosity after ingestion of various amount and type of fruits.

Objectives

1. To determine the glycemic indexes of tangerine, banana, papaya, pomelo, rose apple, and rambutan.
2. To determine the acute responses of plasma glucose, serum lipids and blood viscosity in hyperlipidemic type 2 DM patients after ingestion of different amounts of fruits.
3. To determine the factors which affect glycemic index and glycemic response of fruits including type of sugar and fiber.

CHAPTER II
LITERATURE REVIEW

Epidemiology

Diabetes is already recognized as a public health problem of pandemic proportions. Type 2 diabetes mellitus has been rapidly emerging as a major health threat in populations from both developed and developing countries. The prevalence of diabetes for all age groups worldwide was estimated to be 2.8 % in 2000 and 4.4 % in 2030. The total number of people with diabetes is projected to rise from 171 million in 2000 to 366 million in 2030. In 2002, there are 18.2 million people in the United States, or 6.3% of the population, who have diabetes. A 16 % increase (50, 51) in diabetes prevalence was noted between 1980 and 1994 and in the 8-year period from 1990 to 1998, there was a 33 % increase. Prevalence is currently higher in developed than in developing countries, but the majority of people affected already resides in developing countries, and prevalence is increasing at a much more rapid rate in the developing countries. A study (52) showed the projected numbers of people in developed and developing countries with diabetes in the years 1995 and 2025. In the developing countries, a 48% increase in prevalence, from 3.3 to 4.9%, and a 170% increase in number, from 84 to 228 million, is projected. In the developed countries, increases in prevalence during this period will average; 27%, from 6 to 7.6%, thus increasing from 51 to 72 million people.

In Asian region, the incidence of diabetes mellitus was calculated to be 27.4 per 1000 person-years in Taiwan (53). The prevalence of diabetes was 9.1 % for men and 10.8 % for women. The prevalence of impaired glucose tolerance (IGT) was 12.0 % for men and 16.5 % for women in a rural area in Japan (54). However, in 1964 to 1992, the estimated of prevalence of diabetes mellitus was 9.6-11.9 % in Japanese adults 40 years old or over. In the Kashmir valley (55) of the Indian subcontinent, the prevalence of diabetes and IGT were 1.89% and 8.09 %, respectively. The prevalence of diabetes mellitus raises from 4.7 % in 1984, 8.6 % in 1992 and 9.0 % of Singaporean (56) adults 18-69 years old in 1998. In Thailand (57) the estimated national prevalence of diabetes in adults was 9.6 % (2.4 million people) in 2000. The incidences of diabetic complications in various organs (58) were 7.5 %-46.2 %. Moreover type 2 diabetes mellitus (DM) is being diagnosed more frequently in children and adolescents. The results of study of patients with DM in the Division of Pediatric Endocrinology, Faculty of Medicine, Siriraj Hospital (59) demonstrate that type 2 DM in Thai children and adolescents has increased from 5% during 1986-1995 to 17.9% during 1996-1999. Mean age was 11.6 years. Mean BMI was 27.8 kg/m^2. The period of increase in type 2 DM is associated with an increase of obesity prevalence from 5.8% in 1990 to 13.3% in 1996.

Diabetic Dyslipidemia and Cardiovascular Disease

Diabetes is a major risk of cardiovascular disease. In the United States (6), diabetes is associated with 2- to 4- fold increase in the risk of coronary artery disease. As the population in the United States has aged, the incidence of the deadly quartet (obesity, glucose intolerance, and dyslipidemia) has markedly increased, culminating in the current epidemic of type 2 diabetes mellitus. Of all individuals who have diabetes, approximately 85 % to 90 % have type 2; it typically affects older patients, especially those over the age of 50. Diabetes mellitus is now considered a “cardiovascular (CV) risk equivalent” because patients with type 2 diabetes and no prior history of CV disease have the same future risk of having a myocardial infarction (MI) as non-diabetics with a prior history of MI. Lipid and lipoprotein abnormalities in diabetic are part of the reasons attributing to coronary heart disease (CHD). Thus the control of serum lipids is important for preventing CHD in diabetic.

The characteristic lipid profile of diabetes patients is elevated triglycerides, cholesterol (60, 61), small- dense low density lipoprotein (LDL) particles (6, 62) and VLDL, reduced HDL-C (63, 64) levels and elevated serum LDL-C (61) in some patients. Then pathophysiologic mechanisms contributing to macrovascular disease in type 2 diabetes mellitus include hypertriglyceridemia, high small-dense LDL and low HDL-C levels (61, 62, 65).

Result from the WHO multinational study indicated that ischemic heart disease (IHD) was more strongly associated with serum triglyceride than cholesterol levels, especially in obese type 2 diabetic patients (66). The risk of CHD (67) was doubled in type 2 diabetic patients with high total triglycerides level > 204 mg/dL (2.3 mmol/l).

Blood Viscosity and Diabetes Mellitus

Blood viscosity is an important determinant of blood flow (17, 68, 69) and the rate of oxygen transport and delivery in the circulation and coronary blood flow. The rate of coronary blood flow is equal to pressure divided by resistance. An increase in blood viscosity will cause an increase in flow resistance and thus a reduction in blood flow at a time period. If there is a decrease in the blood flow from hyperviscosity, this could increase the hypoxia and further accelerate the process of vascular occlusion (18, 19).

Viscosity is a property of fluid (15) related to the internal friction of adjacent fluid layers sliding past one another as well as the friction generated between the fluid and the wall of the vessel. This internal friction contributes to the resistance to flow. For a moving fluid, the coefficient of viscosity (η) can be defined as the ratio of shear stress (τ) to shear rate (D):

$$\eta = \tau/D$$

where shear stress represents energy loss due to friction between adjacent fluid layers, and shear rate refers to the relative velocity of the fluid layers fluid with strong intermolecular attractions offer a high resistance to flow and have high coefficients of viscosity. The unit of viscosity is the poise, which is equivalent to 1 dyne.s/cm^2. Relative viscosity 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 and the relative viscosity of plasma is approximately 1.8 (15).

Blood is a non- Newtonian, shear thinning fluid; its viscosity is not constant, even with unchanged composition, blood viscosity changes with the speed at which it flows. The shear dependence of blood viscosity is a function of red blood cell aggregation at low shear rates and red blood cell deformation at high shear rates (70, 71). Blood viscosity should be expressed in absolute units (mPa sec, = cP) at a stated shear rate (or shear stress), temperature (usually 37°c) and hematocrit (14).

Factors influencing blood viscosity are the concentration of red blood cells (hematocrit, Hct), the amount of free fibrin (fibrinogen that has been transformed due to part activation of the coagulation system), plasma viscosity, the flexibility of red blood cells, the tendency for elements in the blood (such as red blood cells) to form larger groups and to a lesser extent, the concentration of white blood cells and platelets (14, 70, 72).

A substantial number of epidemiological studies (18, 73, 74) have evaluated the relationship between blood viscosity and cardiovascular disease. The risk of a major cardiovascular event (death, acute myocardial infarction or acute need for cardiovascular surgery) increases with elevated blood viscosity. In the Edinburgh Artery Study (74), blood viscosity has been strongly associated both with asymptomatic peripheral arterial disease and with the risk of subsequent cardiovascular events (ischemic heart disease or stroke). This result suggests that blood viscosity may be important risk factors for the development of early atherosclerosis. Elevated blood viscosity has been found in a number of conditions associated with increased risk of cardiovascular disease, such as hypertension, smoking, diabetes and elevated LDL cholesterol.

High blood viscosity was found in diabetes patients (75, 76). Several studies also suggested that diabetic patients with insulin resistance and high blood viscosity were associated with a high risk of atherosclerosis and ischemic heart disease (77, 78). Brun et al (20) suggested that blood hyperviscosity was a symptom of insulin resistance while Moan et al (22) reported that changes in blood viscosity, hematocrit and other hemorrheologic factors were correlated to insulin sensitivity. The relationship between type 2 DM and cardiovascular heart disease is well known and the early rheological impairment could be the factor that links both pathologies.

Some factors involved in hyperglycemia (77, 78) and dyslipidemia (79-82) are also suspected of impairing hemorrheology promoting changes in the microcirculation and they are independent risk factors for coronary heart disease. Several studies found that plasma viscosity was found to have a positive linear association with chylomicrons, very low density lipoprotein (VLDL), low density lipoprotein (LDL), total cholesterol and apolipoprotein B and negative linear association with HDL-cholesterol and with apolipoprotein A-1. Moreover, Mellinghoff et al (24) found that well controlled diabetic patients had significantly lower value of viscosity and density of plasma than poorly controlled patients.

Dietary Management for Diabetic Patients

The management of diabetes includes dietary management, exercise and used hypoglycaemic agent or insulin for glycemic control (83). Due to DM is metabolic disorder. So dietary management is attract because it is lower cost and directly effect to blood glucose and insulin level. In diabetic control, the choice of quantification and qualification of food improves glycemic control and decreased the risk of acute and long term complication particularly cardiovascular disease.

In the role of diet, particularly carbohydrates, the diabetes associations of many countries around the world reviewed their dietary recommendation and advised that diabetic patients should consume high carbohydrate (complex carbohydrate eg. whole grains, fruits, vegetable) with low fat, low glycemic index, high fiber and restrict simple sugars (84).

Glycemic Index and Diabetes Mellitus

The glycemic index concept was initially proposed in 1981.The glycemic index is the incremental area under the curve of blood glucose produced by a standard amount of carbohydrate in a food, usually 50 g, relative to the incremental area produced by the same amount of carbohydrate from a standard source, usually white bread or glucose. The concept of the glycemic index can also be applied to whole meals or overall diet (43) so it is being recommended as a guide to food choices for the control of postprandial glycemia (85).

Both metabolic and epidemiologic evidence (46, 47, 86-88) suggests that replacing high glycemic index forms of carbohydrate with low glycemic index carbohydrates will reduce the risk of type 2 diabetes in normal subjects and reduce the risk for the chronic complication of diabetes in diabetes patients.

Compare to the effect of the intake of a low glycemic index food (43), high glycemic index food with the same equivalent in carbohydrate content induces a higher peak of circulating glucose and a large area under the curve over the early postprandial period. As a consequence of the induced insulin response, intake of a high glycemic index food results in lower blood glucose concentrations over the late (2-3 hour) postprandial period than that of a low glycemic index food. High glycemic index foods are those with high carbohydrate content and foods that are rapidly digested. Specific factors that affect increased glycemic index include; high refined carbohydrate content; high glucose and/or starch content relative to lactose, sucrose and fructose content; low soluble fiber content and finally, soft overcooked highly processed or over ripened food textures (89-94).

Conversely, low glycemic index foods cause a lower peak glucose (43), demonstrate a smaller area under the curve for the increment in blood glucose in the 2 hour postprandial period, and have a lower risk of causing relative hypoglycaemia (because of the reduced responses of counter regulatory hormones).

The rapid increase in blood glucose after the ingestion of rapidly digestible, high glycemic index carbohydrates (43) may increase serum triglycerides, satiety and suppress appetite and food intake in the short term, whereas the consumption of slowly digestible, low glycemic index carbohydrates (43, 95, 96), which results in slow, prolonged glucose disposal, may be more effective in sustaining satiety in the long term.

Insulin resistance may also occur with a high glycemic index diet because of the direct effects of hyperglycemia (97), counter-regularatory hormone secretion and increased late postprandial serum free fatty acids levels (98).

Regular consumption of low glycemic index meals compared with isoenergetic and nutrient controlled high glycemic index meals (46, 99), results in reduction 12-h blood glucose, total serum triglyceride, serum cholesterol, insulin secretion, serum fructosamine, 24-h urinary c-peptide levels.

Low glycemic index foods have helps in reducing early post prandial hyperglycemia and decreasing risk for post absorptive hyperglycemia, serum cholesterol, triglyceride, triacylglycerol, free fatty acids and LDL-C among both diabetic and nondiabetic, especially among people with somewhat elevated triglycerides (46, 47, 86).

In addition to, under weight maintenance conditions (100), low glycemic index diets shown lower postprandial and daylong glucose level, glycated haemoglobin, triglyceride and total cholesterol concentration as compared to high glycemic index diets.

Carbohydrate in Fruit

The consumption of fruits should continue to be encouraged because of the resulting increased intake of fiber, micronutrients, and antioxidants (101, 102). In addition fruits are the major sources of dietary carbohydrate. Epidemiologic data (101, 103, 104) support the association between high intake of vegetables and fruit might slow or prevent the onset of chronic diseases. A high consumption of fruits and vegetables is associated with a significantly lower risk of coronary artery disease and stroke. Singh et al (105) reported that consumption of 0.5-1.0 kg guava could decrease blood pressure, serum cholesterol and triglycerides. Sablé-Amplis et al showed that consumption raw apple reduced serum cholesterol. However, in diabetic control the choice of the right type and quantity of fruit is important because fruits contained high simple carbohydrates and may result in high blood glucose, lipid and viscosity. The rise of blood glucose and lipid in type 2 diabetic patient after ingestion of fruits varies markedly and depends on many factors, including the composition of carbohydrate and amount of carbohydrate consumed.

The carbohydrates of the fruit include simple sugars (glucose, fructose and sucrose) and complex carbohydrates (starch and non-starch)

Simple sugars (106-108)

The major simple sugars in fruit are the monosaccharide glucose and fructose and the disaccharides sucrose. Glucose is derived from the free glucose in fruits and from the hydrolysis of starch and disaccharide (maltose and sucrose). Glucose is most rapidly absorbed by passing into the peripheral circulation and entering the peripheral tissues under the influence of insulin and induced hyperglycemia. Fructose is absorbed more slowly than sucrose and does not require insulin for entering into cells. Sucrose is disaccharides (glucose and fructose). Disaccharides are too large to cross the mucosal cell membrane and must, therefore, be hydrolyzed into monosaccharides before absorption. The absorption of fructose from the intestine is greatly facilitated by the presence of free glucose. Moreover the absorption of fructose in the presence of di-, tri-, and oligosaccharides from starch hydrolysis might not have been as rapid as it would have been in the presence of free glucose. Derived glucose from the hydrolysis of disaccharides is more rapidly transported into cells than free glucose (109).

Complex carbohydrates

Starch is polysaccharides, which will be turned into mono-, di, and oligosaccharides after hydrolysis. In general, complex carbohydrates break down more slowly than simple sugars (108, 110, 111).

Non-starch polysaccharides are soluble (such as gum and pectin) and insoluble fibers (such as cellulose, hemicellulose and lignin). Fiber has no calories because the body cannot absorb it so it low fat and low calories. Current evidence has suggested that high fiber diet, about 20-35 grams of dietary fiber per day, especially of the soluble fiber supplements offered some improvement in carbohydrate metabolism, lower total cholesterol and LDL-C (112).

In study patients with type II diabetes (113, 114) , soluble fiber decreased insulin levels in the blood, keep cholesterol under control by removing bile acids that digest fat and regulate blood sugar. This is accomplished by coating the gut's lining and delaying stomach emptying. As a result, they can slow sugar absorption after a meal and may reduce the amount of insulin needed. Tinnarat et al (115) defined that blood glucose concentration was flattened in diabetic patients after one month of high dietary fiber intake. On the other hand, insoluble fiber had little or not effect on plasma glucose and serum lipid. It helps to prevent constipation and possibly colon cancer.

Metabolism of Glucose and Fructose

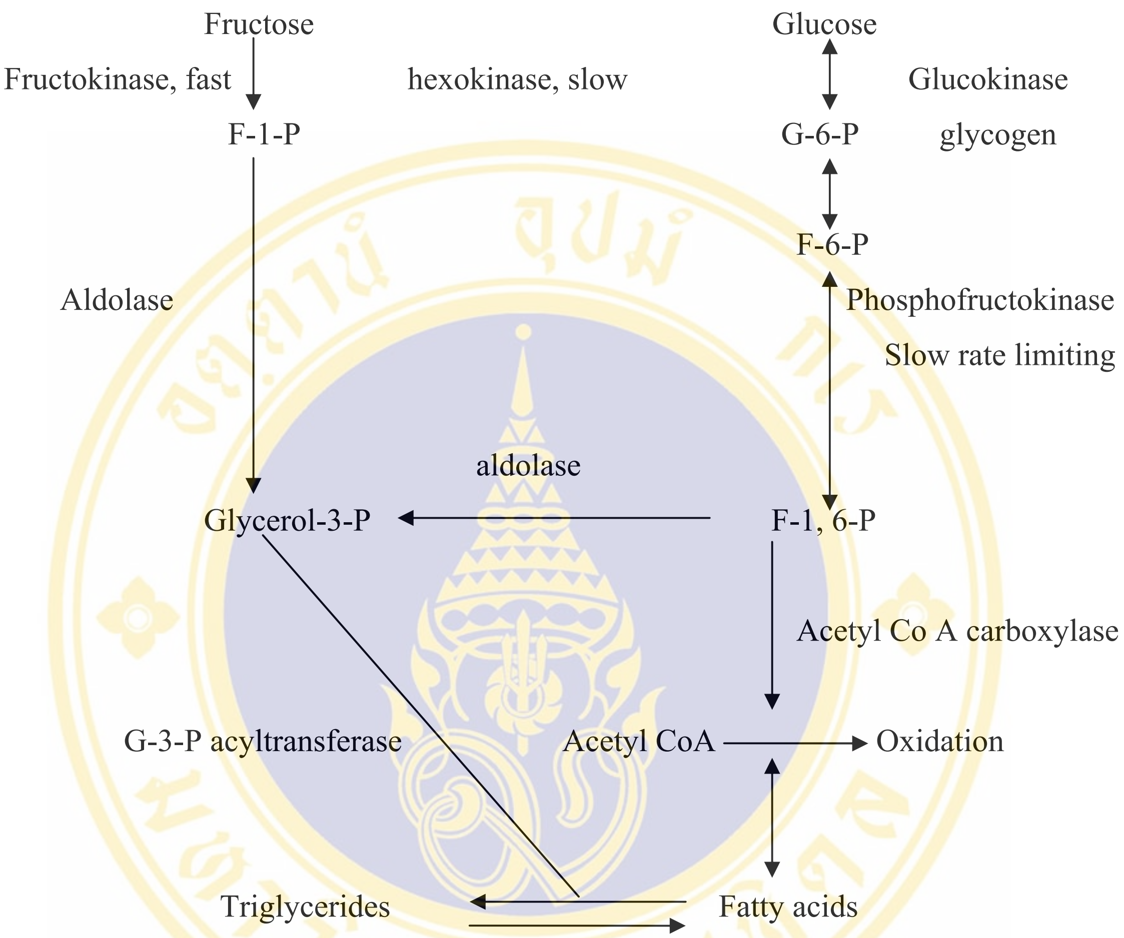


Figure 2.1 Metabolism of glucose and fructose

Although glucose and fructose are similar molecules, they are metabolized differently within the cell. The monosaccharides pass in to the liver. The metabolism of both sugars begins with phosphorylation (109). Glucose is phosphorylated to glucose-6-phosphate by enzymes hexokinase or glucokinase. Glucose-6-phosphate is used for the synthesisof glycogen or subsequently isomerized to fructose-6-phosphate and further then to fructose-1,6-phosphate which enters into the glycolytic pathway and other, fructose-1, 6-phosphate is then split into glycerol-3-phosphate and continue in the lipogenesis.

The hepatic metabolism of fructose begins with its phosphorylation by one or two enzymes, hexokinase or fructokinase. However, fructose is mainly metabolized in the liver; fructose is converted to fructose-1-phosphate rapidly than fructose-6phosphate because liver fructokinase activity is greater than the combined activity of hepatic hexokinase and glucokinase. The fructose-1-phosphate is broken down to glycerol-3-phosphate and is used for the synthesis of triglyceride (109, 116). The products of fructose metabolism in the glycolytic pathway of the liver are glucose, glycogen and lactate.

The hepatic metabolism of glucose is limited by the capacity to store glucose as glycogen and, more importantly, by the inhibition of glycolysis and further glucose uptake resulting from the effects of citrate and ATP to inhibit phosphofructokinase. In contrast, fructose uptake by the liver is not inhibited at the level of phosphofructokinase, fructose consumption results in large increases of circulating lactate than does consumption of comparable amount of glucose (35, 117).

Blood Glucose and Lipids Response to Simple Sugars and Starch in Diabetic Patients

Allen et al (118) studied in diabetic dogs showing more rapid rise in blood glucose concentrations and greater glucosuria after ingestion of glucose than after complex carbohydrate starch. Swann et al (112) showed that the blood glucose and insulin responses were higher following a glucose load than following a starch load. Moreover, the elevations of blood lipid concentrations also respond to large amount of dietary sugars. Consumption of carbohydrates is associated with increased plasma triglyceride concentration but no effect in plasma cholesterol concentrations. In human, increased triglycerides are observed with very high intake of over 35 % of energy from sucrose or over 20 % of energy from fructose (119). Nikkila and Kekki (120) demonstrated that increased sugar intake caused increased blood triglyceride levels in nondiabetic subjects.

The absorption of fructose from the intestine (121) is greatly facilitated by the present of free glucose but the present of di-, tri-, and oligosaccharides from starch hydrolysis might not have been as rapid as it would have been in the presence of free glucose (122). Patricia M. et al. (123) found that fructose must be consumed before a starchy food in order to reduce postprandial glycemia. This finding may have practical applications because the small amount of fructose used in their study (10 g fructose) could easily be obtained in the diet via fruits (eg. an apple has 9-10 fructose). Fructose was considered beneficial because it was thought to stimulate lower postprandial plasma concentrations of glucose; for example, Bantle et al and Osei et al (124) found that fructose reduced blood glucose concentrations in comparison with either isocaloric sucrose or isocaloric diabetic diets in patients with diabetes. Crapo et al (125) examined the acute effect of the ingestion of 50 g load of glucose, sucrose and fructose in DM type 2 patients. Fructose ingestion resulted in significantly lower plasma glucose and insulin responses than did sucrose or glucose although; some investigators showed that starch and glucose assimilated at the same rate. Furthermore, Rosalie et al (126) showed that type 2 diabetes subjects drank equienergetic preloads of glucose (75 g), fructose (75 g) and water 3 hours before an ad libitum buffet lunch. The results appeared oral fructose ingestion produced a lower postprandial plasma glucose response than equienergetic glucose. Moreover, this study found that 1) fructose ingestion produces greater increases in plasma insulin concentration in type 2 diabetics than nondiabetics and 2) glucose and fructose have equivalent short-term satiating efficiency in both type 2 diabetics and nondiabetics.

But undesirable results of fructose from experimental studies in animal model and from short-term feeding trials among humans suggest that higher fructose intake contributes to insulin resistance, impaired glucose tolerance and hyperinsulinemia. Furthermore, studies showed that fructose intake stimulates lipogenesis and increases plasma triacylglycerol concentration compare with other types of sugar and fructose intake resulted in weight gain. Taken together, these results suggest high intake of fructose overtime might deteriorate insulin sensitivity; for example, Sleder J. et al. (127) showed that rats fed diets containing 66 % glucose or fructose, 22 % casein and 12 % lard for 1 week. The effect of fructose diets on plasma triglyceride, glucose, and insulin concentrations were higher than other foods. This study indicated that fructose-induced hypertriglyceridemia is associated with significant hyperinsulinemia. However, other studies (34, 128) showed that small to moderate amount of fructose may be beneficial in improving glycemic control and does not cause hypercholesterolemia, hypertriglyceridemia ,or abnormalities in triglyceride turnover in type 2 diabetes and mild-to-moderate hypertriglyceridedmia subjects. In conclusion, diabetes patients can consume fructose in fruits, vegetables and other foods but uses of added fructose as a sweetening agent may cause deteriorating effects.

CHAPTER III
MATERIALS AND METHODS

A. Experimental Design

Subject selection

Sixteen hyperlipidemic DM type 2 patients were enrolled. Their ages ranged from 31-70 years. Their fasting plasma glucose levels were over 120 mg/dL, serum triglycerides levels were over 150 mg/dL and/or serum cholesterol levels were over 200 mg/dL (129). All subjects were not suffered from severe kidney or liver disease, anemia or any hormonal disturbances. There was no change in the amount of insulin or hypoglycemic agents used during the study.

The study was performed at the Clinical Research Center, Ramathibodi Hospital, Bangkok, Thailand. The objectives and details of the study were explained to all subjects and written consents were signed.

Fruit selection

Six kinds of Thai fruit were selected according to their popularity score lisred by the diabetic patients. They are banana-Kai variety (Musa sapientum, Linn.), tangerine-Bangmod variety (Citrus nobills), papaya-Khak-dam variety (Carica papaya), pomelo-Khawnampueng variety (Citrus spp.), rambutan-Rong-rean variety (Nophelium lappaccum, Linn) and rose apple -Tubtimjan variety (Eugenia spp.)

Selection of dosage of fruits

All fruits were purchased as fresh fruits from a local grocery at the same market. They were not too raw or too ripe. Two test amounts of fruits were chosen according to the amount normally eaten and the amount used for glycemic index test as follow 30 and 50 g which equalled to 2 and 3 serving sizes of the food exchange (130-132) portions of fruits calculated from Thai food table (133) (Table 3.1).

Table 3.1 Actual weight of fruits containing different carbohydrate portions

Kinds of fruit	Weight (g)	
	30 g CHO	50 g CHO
Tangerine	333.33	555.56
Pomelo	241.94	403.23
Rambutan	172.41	287.36
Rose apple	454.55	757.58
Papaya	254.24	423.73
Banana-Kai variety	86.21	143.68

Analysis of carbohydrate content in fruits

The composition of carbohydrate content of the fruits was analyzed by Food and Nutrition Technical Services, Institute of Nutrition, Mahidol University, Thailand (Table 3.2).

Table 3.2 The methods used for analyzing the composition of carbohydrate content of the fruits

Nutrient	Method
Carbohydrate	Calculation
Glucose	HPLC
Sucrose	HPLC
Fructose	HPLC
Total dietary fiber	Enzymatic-gravimetric
Soluble fiber	Enzymatic-gravimetric
Insoluble fiber	Enzymatic-gravimetric

The composition of carbohydrate content of different fruits was described in Table 3.3. Banana and rambutan contained 4-5 time higher carbohydrate content than rose apple and tangerine.

Table 3.3 The composition of carbohydrate content of the test fruits

Kinds of fruit	g / 100 g fruit weight						
	CHO	G	F	S	DF	SF	ISF
Tangerine	9	1.5	1.3	4	1.5	0.7	0.8
Pomelo	12.4	1.1	1.3	5.9	0.8	0.6	0.2
Rambutan	17.4	2.2	2.3	7.4	1.5	0.4	1.1
Rose apple	7.6	3.2	3.3	nd	0.9	0.2	0.7
Papaya	11.8	3.1	2.6	nd	1.2	0.6	0.6
Banana-Kai	34.8	8.3	7.6	4.8	2	0.6	1.4

CHO = Carbohydrate, G = Glucose, F = Fructose, S = Sucrose,
DF = Total dietary fiber, SF = Soluble fiber, ISF = Insoluble fiber, nd = Not detectable

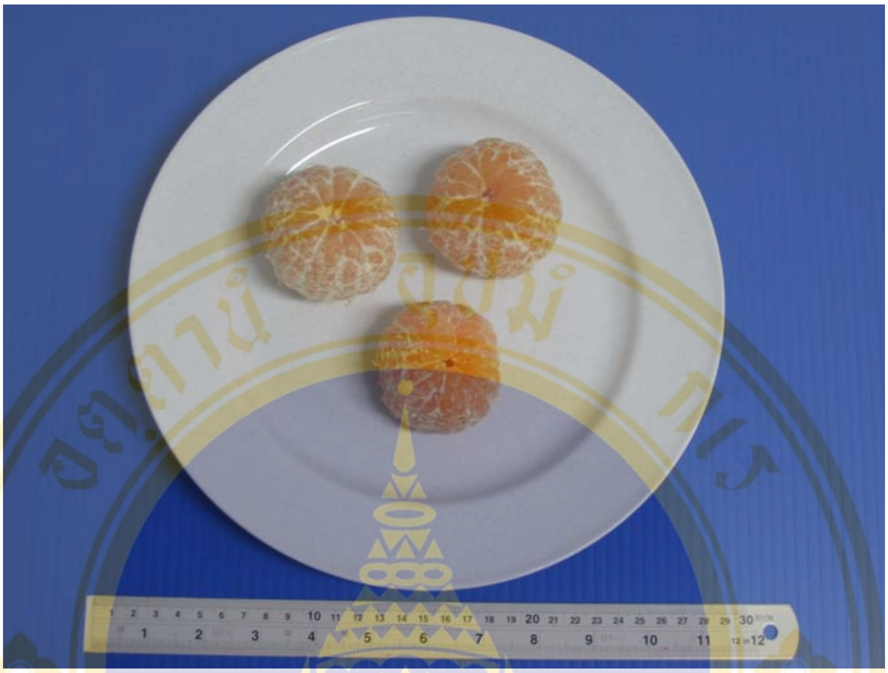
Table 3.4 Carbohydrate composition in the 2 test-doses of 6 Thai fruits according to their actual intake

Kinds of fruit	g/30 g CHO							g/50 g CHO						
	Total sugar	G	F	S	DF	SF	ISF	Total sugar	G	F	S	DF	SF	ISF
Tangerine	22.66	5	4.33	13.33	5	2.33	2.67	37.77	8.33	7.22	22.22	8.33	3.89	4.44
Pomelo	20.08	2.66	3.15	14.27	1.94	1.46	0.48	33.47	4.44	5.24	23.79	3.22	2.42	0.80
Rambutan	20.52	3.79	3.97	12.76	2.59	0.69	1.9	34.19	6.32	6.61	21.26	4.31	1.15	3.16
Rose apple	29.55	14.55	15	nd	4.09	0.91	3.18	42.76	21.05	21.71	nd	5.92	1.32	4.60
Papaya	14.49	7.88	6.61	nd	3.06	1.53	1.53	24.15	13.13	11.02	nd	5.08	2.54	2.54
Banana-Kai	17.85	7.16	6.55	4.14	1.72	0.52	1.20	29.75	11.93	10.92	6.9	2.87	0.86	2.01

G = Glucose, F = Fructose, S = Sucrose,

DF = Total dietary fiber, SF = Soluble fiber, ISF = Insoluble fiber, nd = Not detectable

Characteristics and portions of different fruits

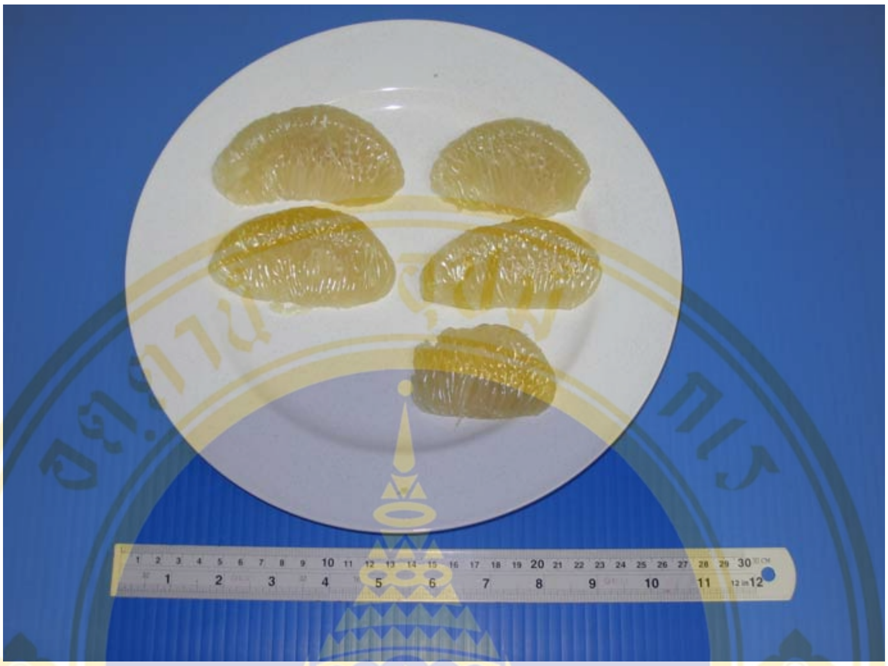


(A)

(B)

Figure 3.1 Tangerine : 30 g CHO (A) and 50 g CHO (B)

Characteristics and portions of different fruits (continued)

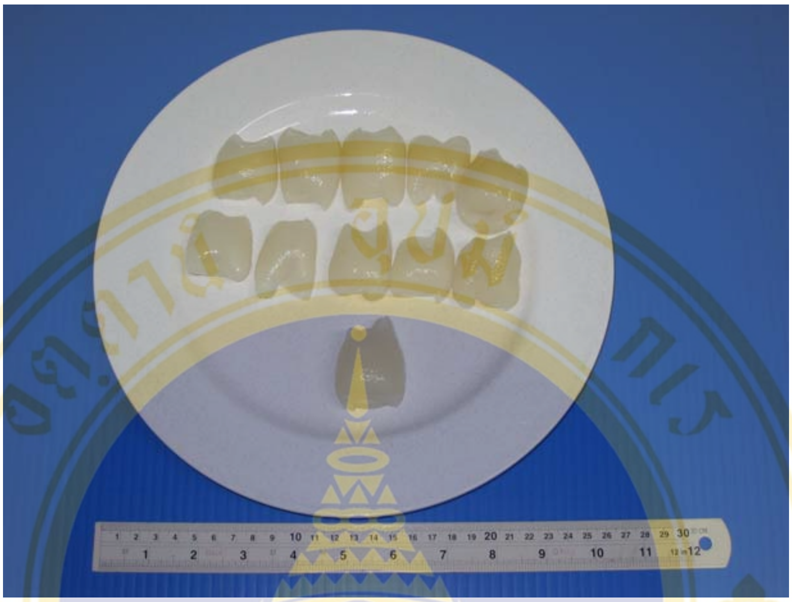


(A)

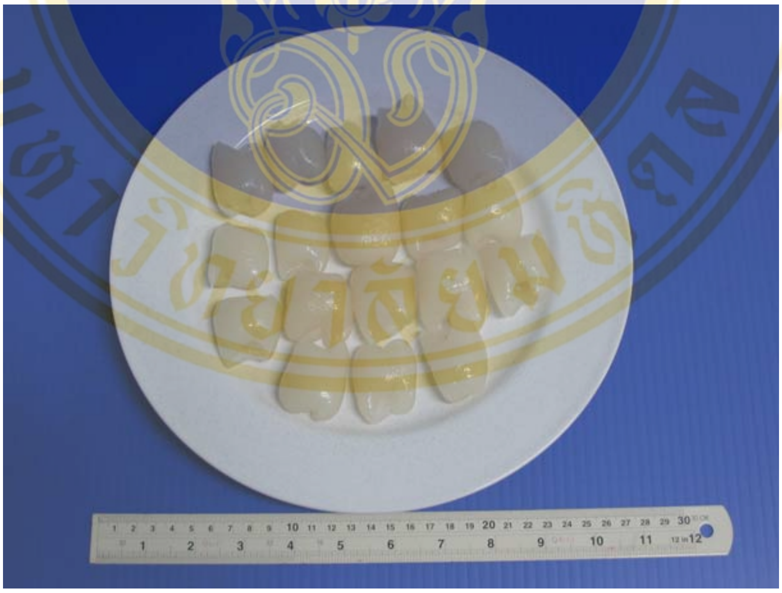
(B)

Figure 3.2 Pomelo : 30 g CHO (A) and 50 g CHO (B)

Characteristics and portions of different fruits (continued)



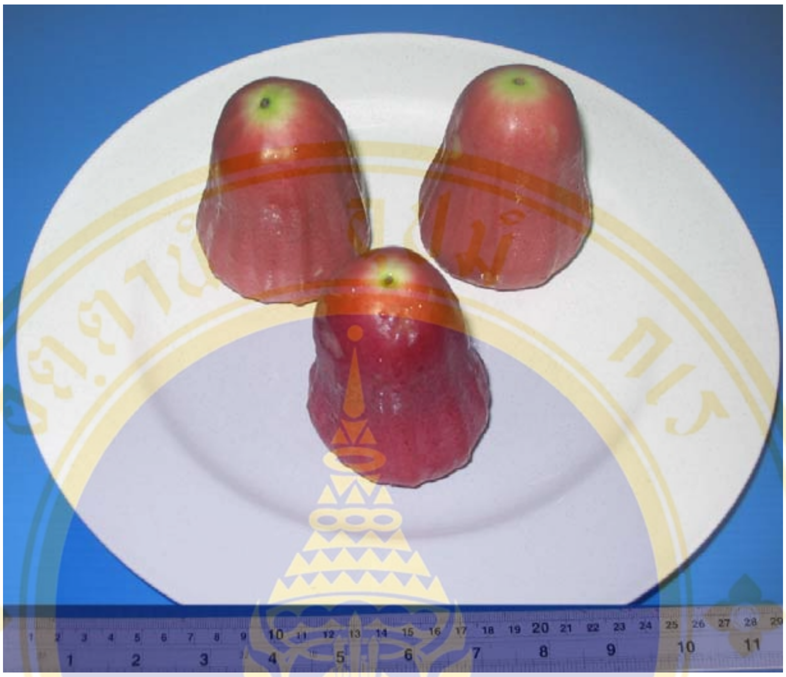
(A)



(B)

Figure 3.3 Rambutan : 30 g CHO (A) and 50 g CHO (B)

Characteristics and portions of different fruits (continued)

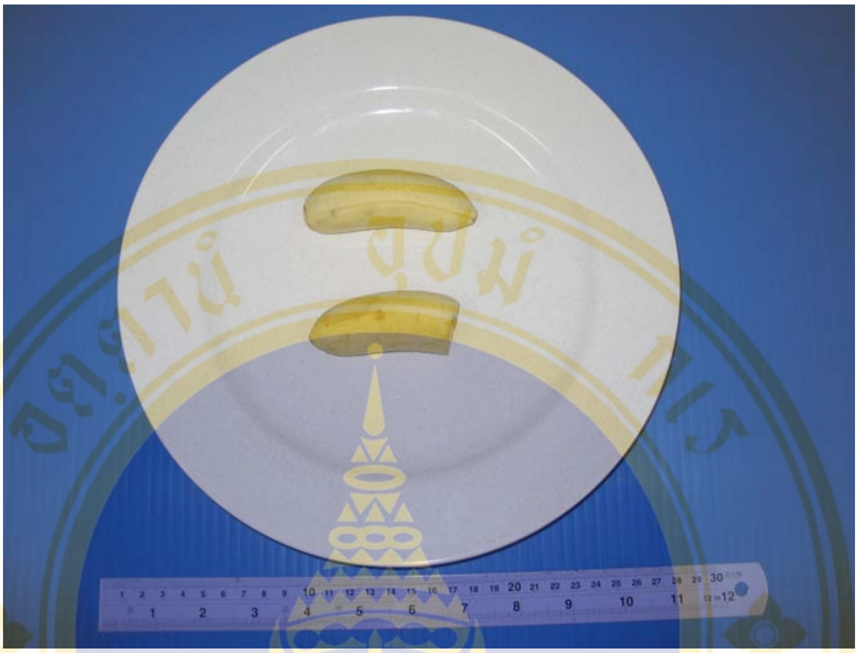


(A)

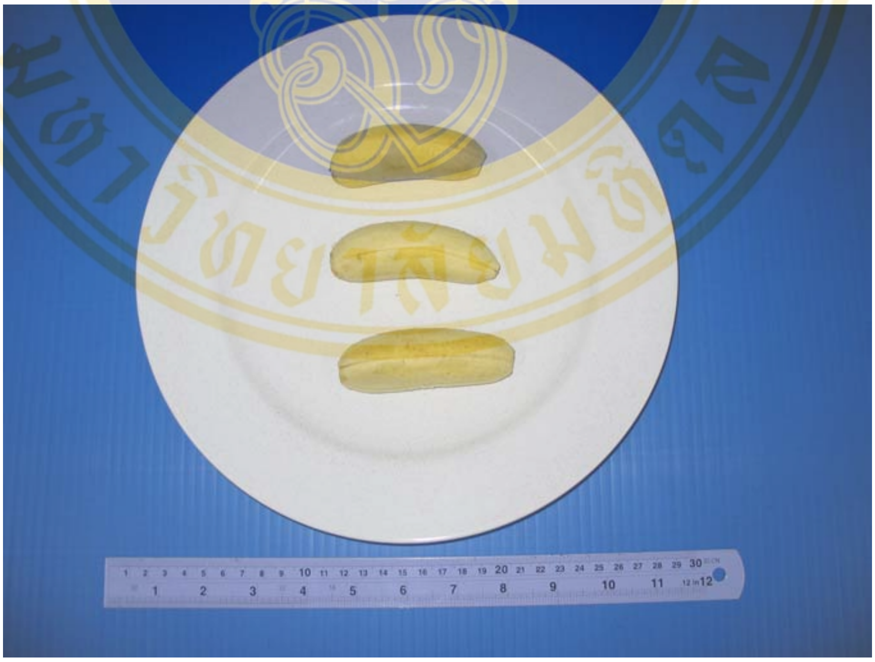
(B)

Figure 3.4 Rose apple : 30 g CHO (A) and 50 g CHO (B)

Characteristics and portions of different fruits (continued)



(A)



(B)

Figure 3.5 Banana-Kai variety : 30 g CHO (A) and 50 g CHO (B)

Characteristics and portions of different fruits (continued)

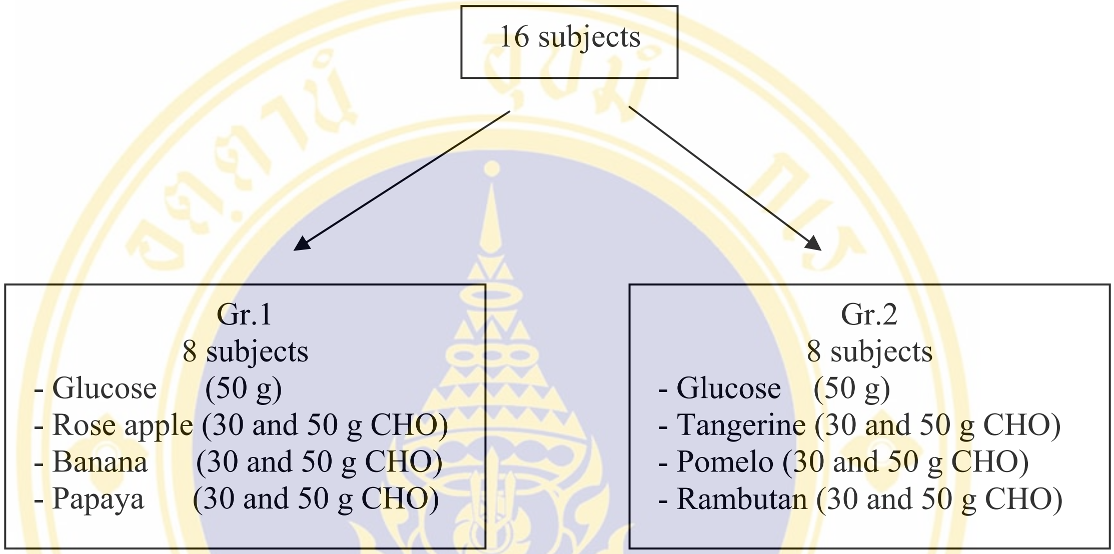
(A)

(B)

Figure 3.6 Papaya : 30 g CHO (A) and 50 g CHO (B)

Study design

The subjects were randomly divided into 2 groups. All subjects underwent 6 tests of fruit consumption and a 50 g glucose drink (a reference standard for determination of the glycemic index) in 150 ml water, in random sequence.



Each subject underwent an overnight fast for 8 to 10 hours before each test. They did not take their ordinary morning medication on each test day. The test loads were given at 8 o'clock in the morning after an overnight fast. The subjects underwent a test load every 3-4 weeks. Each test lasted 300 min.

Time (min)	0	30	60	120	180	240	300
Time (o' clock)	8	8.3	9	10	11	12	13
Plasma glucose	*	*	*	*	*	*	*
Serum lipid	*						*
Blood viscosity	*		*				*

* Blood collection

On the test day, a plastic catheter was placed in a forearm vein for blood sample collect and kept patent with 50 units of heparin after each blood drawn. The heparin in the catheter and 2 ml of blood were aspirated and discarded prior to collecting each blood sample. The test meals consisted of different kinds and amounts of fruits (30 and 50 of carbohydrate) and a 50 g glucose drink which were given according to the randomization. Fasting blood samples were drawn before taking the test fruits.

Each subject consumed one test fruit at a time and finished up within 15 minutes and followed by ingestion of 150 ml of water. Postprandial blood samples were taken at 30, 60, 120, 180, 240 and 300 minutes. All blood samples were taken for analysis of glucose. Blood sample at 0, 60 and 300 minutes were taken for analysis of whole blood viscosity. Serum lipids at 0 and 300 minutes were analysed.

B. Biochemical Analysis

Plasma glucose and serum lipid levels were analysed by laboratory of the Clinical Chemistry Department, Ramathibodi hospital.

Determination of glucose

After collection of the whole blood containing anticoagulant, NaF (Sodium fluoride) the levels of glucose in plasma were determined using the Dimension© Clinical chemistry system of Dade Behring company which was an in vitro diagnostic test intended for the quantitative determination of glucose in serum, cerebrospinal fluid, plasma and urine.

The glucose method is an adaptation of the hexokinase-glucose-6-phosphate-dehy -drogenase method, presented as a general clinical laboratory method by Kunst et al. This method is more specific than general reducing methods and will give results lower than those obtained by such reducing method.

Principles of procedure:

Glucose + ATP $\xrightarrow[\text{Hexokinase, } Mg^{2+}]{}$ Glucose -6-Phosphate + ADP

Glucose-6-Phosphate dehydrogenase

Glucose -6-Phosphate + NADP $\longrightarrow$ Glucose - 6 - Phosphoglucolactone + NADPH

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 :

Glucose concentration = (A*100) / As

where A is the absorbance of the specimen reaction tube and As was the standard reaction tube containing 100 mg per deciliter standard glucose. The amount of glucose concentration was expressed as mg/dL. Appropriate dilution was made if the specimen contained high level of glucose. When a series of hourly plasma glucose of each individual were analyzed, the area under the curve plotted between the glucose concentrations against the time of blood withdrawals can be calculated using computer integration.

Calculation of glycemic index (GI)

Glycemic index of food is the ratio of blood glucose area under the curve for test food compared with a reference food (bread or glucose). The GI is usually defined as the area under the glycemic response curve during a 2-hour period after consumption of 50 g CHO from a test food, and values are expressed relative to the effect of either white bread or glucose (40, 134). Originally the reference was glucose contained 50 g CHO (135).

In the fruit, the GI was calculated as

$$GI = \frac{\text{Blood glucose response area of fruit}}{\text{Blood glucose response area of glucose}} \times 100$$

Determination of serum total cholesterol

Determination of total cholesterol level using Dimension ® Clinical Chemistry System by Cholesterol Flex ™ Reagent Cartridge.

Principle of procedure

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{Cholesterol esterase}} \text{cholesterol} + ROOH$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{Cholesterol oxidase}} \text{cholest-4-ene-3-one} + H_2O_2$$

$$2\ H_2O_2 + \text{4- aminophenazone} + \text{phenol} \xrightarrow{\text{Peroxidase}} \text{4-(p-benzoquinone-mono-imino) phenazone} + 4\ H_2O$$

4-(p-benzoquinone-mono-imino) phenazone is chromophore that absorbs at 540 nm

Calculation

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

Determination of serum triglycerides

The TGL Flex® used on the Dimension ® Clinical Chemistry System is an in vitro diagnostic test is tented for the quantitative determination of triglyceride in serum and plasma.

Principle of procedure

TG method is based on an enzymatic procedure in which a combination of enzyme is employed for the measurement of serum or plasma triglycerides. 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-phos- phate oxidase (GPO) oxidize glycerol-3-phosphate to dihydroacetone phosphate and hydrogen peroxide. The catalytic reaction of peroxidase (POD) produces quinoneimine chromophore.

$$\text{Triglyceride} \xrightarrow{\text{LPL}} \text{Glycerol + fatty acids}$$

$$\text{Glycerol + ATP} \xrightarrow{\text{GK}} \text{Glycerol -3-phosphate + ADP}$$

$$\text{Glycerol-3-phosphate} + O_2 \xrightarrow{\text{GPO}} \text{Dihydroxyacetone phosphate} + H_2O_2$$

$$2H_2O_2 + \text{Aminoantipyrine+ 4-chlorophenol} \xrightarrow{\text{POD}} \text{Quinoneimine + HCl} + 4\,H_2O$$

Wavelenges biochromic end point : 510 and 700 nm

Determination of high density lipoprotein cholesterol (HDL –C)

The automated HDL cholesterol assay is a homogeneous method for directly measuring HDL levels without the need for off-line pretreatment or centrifugation steps.

The method is in a two-reagent format and depends on the properties of a unique detergent, which solubilizes only the HDL lipoprotein particles, thus releasing HDL cholesterol to react with cholesterol esterase to produce color. In addition to selectively disrupting the HDL lipoprotein particle, this detergent also inhibits the reaction of the cholesterol enzymes with LDL, VLDL and chylomicron lipoproteins by adsorbing to their surfaces. A polyanion is contained in the first reagent to assist with complexing LDL, VLDL and chylomicron lipoproteins further enhancing the selectivity of the detergent and enzymes for HDL cholesterol.

$$\text{LDL,VLDL,Chylomicrons} \xrightarrow{\text{Detergent}} \text{Stable complexes polyanion}$$

$$\text{HDL cholesterol} \xrightarrow[\text{Cholesterol oxidase}]{\text{Detergent}} \Delta\text{'cholestenone} + H_2O_2$$

$$H_2O_2 + \text{DSBmT} + 4\ \text{AA} \xrightarrow[\text{Peroxidase}]{} \text{Odor development}$$

Estimation of low density lipoprotein cholesterol (LDL-C)

LDL-C level was calculated from Friedwald's formula (136) as follow :

$$\text{LDL-C} = \text{total cholesterol} - \frac{\text{(triglyceride)}}{5} - \text{HDL-C}$$

C. Hematological Assessment

Viscosity measurement

After collection of the whole blood containing anticoagulant, ethylenediamine tetraacetic acid (EDTA) (1 mg EDTA for 1 c.c. of blood) and viscosity of blood was measured by the Brookfield DV-II+ viscometer (137).

Viscosity is a measure of a fluid's resistance to flow. The principle of operation of the DV-II+ is to drive a spindle (which is immersed 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. The measurement range of a DV-II+ is determined by the rotational speed of the spindle, the size and shape of the calibrated spring. The blood samples for whole blood viscosity were measured at 37° c; 8 ml of blood was used into the chamber which had fixed shear rate at 264 sec^{-1}.

D. Assessment of Nutritional Status

Anthropometric measurement

Anthropometry is an indirect method of nutritional assessment by body composition. Anthropometric assessment includes weight, height, triceps skin-fold thickness (TSF), mid-upper arm circumference (MUAC) and mid-upper arm muscle circumference (UAMC) were performed according to standard methodology (138-141).

1.1 Height (Ht)

The subject was asked to stand straight barefoot on a horizontal platform with his heel together, stretching upward to the fullest extension. The back was as straight arm of the height meter was in contact with the subject's head. The reading was read to the nearest millimeter.

1.2 Weight (Wt)

Weight was taken by standing barefoot on the accurate weighting scale. The subject was asked to wear the light clothing.

1.3 Body mass index (BMI)

BMI was calculated from the following formula:

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

1.4 Tricep skinfold thickness (TSF)

TSF is an indirect estimate of body fat or caloric stores. The measurement of the tricep skinfold was performed at the midpoint of the upper left arm, between the Acromion process and the tip of the Olecranon, with the arm hanging relaxed. Use the mean of three consecutive readings. The instrument used was a Harpenden skinfold caliper (British Indicator Ltd., England).

1.5 Mid-upper-arm-circumference (MUAC)

MUAC has been used as an indicator of the inadequacy of both caloric and protein store. The MUAC was measured in centimeters at the same level as the triceps skinfold thickness. The measurement was taken at the midpoint of the upper left arm between the Acromion process of the scapula and tip of Olecranon. After locating the midpoint, the left arm was extended so that it was hanging loosely by the side, with the palm facing inwards. The tape was wrapped gently but firmly around the arm at the midpoint.

1.6 Upper arm muscle circumference (UAMC)

The muscle circumference of the mid-upper-arm is derived from measurements of both MUAC and TSF. UAMC is used to assess protein-energy malnutrition, as the size of the muscle mass is an index of protein reserves. UAMC was calculated using the following equation.

$$UAMC, cm = (MUAC, cm) - (0.314 \times TSF, mm)$$

E. Statistic Analysis

1. The results were expressed as mean ± SD
2. Comparisons of area under the curves of the glycemic index, actual serum lipids and percentage change of serum lipids of fruits were performed by Kruskal-Wallis H test.
3. Comparisons of plasma glucose responses after ingestion of different amounts of fruit between time periods by Friedman's test and multiple comparisons by Bonferroni adjusted.
4. Comparisons of areas under the curves of plasma glucose, blood viscosity before and after ingestion of various kinds and different amount of fruits by Friedman's test and serum lipids by Wilcoxon test. P-values of less than 0.05 were considered significant.

CHAPTER IV
RESULTS

Part I Characteristics of Subjects

Anthropometric measurement

Table 4.1 shows the anthropometric measurements of the subjects. The mean body weight and height were 67.29 ± 11.60 kg and 1.56 ± 0.06 m, respectively. The mean BMI was 27.53 ± 4.20 kg/m^2. The means of waist/hip ratio, TSF, MUAC and UAMC were 0.91 ± 0.08, 28.66 ± 9.67 mm, 29.94 ± 3.20 cm and 21.07 ± 1.08 cm, respectively.

Clinical characteristics of subjects

Table 4.2 describes the initial metabolic characteristics (mean + SD) of sixteen subjects. The mean levels of fasting plasma glucose, serum triglycerides cholesterol, LDL-C and HDL-C were 161.88 ± 61.13, 149.13 ± 49.42, 203.38 ± 29.48, 44.44 ± 7.11 and 129.38 ± 23.43 mg/dL, respectively. All of them did not have severe diabetic complications.

Table 4.1 Anthropometric measurement of subjects

Characteristic	Mean ± SD
Age (years)	56.00 ± 11.60
Weight (Kg)	67.29 ± 11.60
Height (m)	1.56 ± 0.06
BMI (Kg/m^2)	27.53 ± 4.20
Waist (cm)	89.00 ± 11.81
Hip (cm)	98.20 ± 11.41
Waist/Hip ratio	0.91 ± 0.08
MUAC (cm)	29.94 ± 3.20
TSF (mm)	28.66 ± 9.67
UAMC (cm)	21.07 ± 1.08

Table 4.2 Clinical characteristics of subjects

Clinical characteristic	Mean ± SD
Fasting plasma glucose (mg/dL)	161 ± 61.13
Triglyceride (mg/dL)	149 ± 49.42
Cholesterol (mg/dL)	203 ± 29.48
HDL-C (mg/dL)	44 ± 7.11
LDL-C (mg/dL)	129 ± 23.43

Part II Fruit Selection from Questionnaire

Tangerine, guava, banana, and watermelon are the habitually consumed fruits. In fact, the diabetic patients would like to consume durian, longan, and rambutan but these fruits are usually prohibited. In their opinions, durian, longan, and grape consumption would increase their plasma glucose whereas guava, rose apple, papaya, watermelon, tangerine, pomelo and dragon fruits ingestion would not. Furthermore, they believed that durian, coconut and longan consumption would raise serum lipid levels.

In the previous study, Somnuk S. et al (142) had determined the glycemic indexes of durian, guava, longan, dragon fruit, mango, and pineapple. Therefore, durian, guava, longan, and dragon fruit were excluded from our study. For watermelon, the amount contained in 50 g CHO is very large, too much to be consumed in one short period. Therefore, we selected the tangerine, banana, pomelo, rambutan, rose apple, and papaya for our study.

Part III Biochemical Parameters

Plasma glucose levels

Table 4.3–4.4 and Figure 4.1-4.4 compare the mean ± SD and the percentage change of plasma glucose which was recorded up to 300 minutes. The glucose levels rose within 30 minutes, reached the peaks at 60 minutes in most fruits and dropped to below baseline levels within 180-240 minutes in both amount of CHO (30 and 50 g); however, plasma glucose levels of 30 g CHO of rambutan reached the peaks at 30 minutes.

Comparing between fruits, after ingestion of 30 g CHO of different kinds of fruits, (Table 4.3, Figure 4.1).The mean percentage change of plasma glucose at 60 minutes after eating tangerine tended to be higher than other fruits.

The mean percentage changes of plasma glucose after glucose drink were significantly higher ($p < 0.05$) than most fruits in both groups, they are tangerine, rambutan and pomelo at 60 and 120 minutes, and tangerine at 180 minutes in group 1, and banana at 60 and 120 minutes and papaya at 120 minutes in group 2, respectively.There was no difference in the mean percentage change of plasma glucose between rose apple and glucose drink.

Table 4.3 Plasma glucose (mean ± SD) responses and percentage changes after ingestion of 30 g CHO of different fruits

Kinds of fruit	Amount of CHO	0	30	60	120	180	240	300
					Mean ± SD, mg.dL^{-1}			
Tangerine	30 g	139 ± 30	224 ± 30*	239 ± 31*	180 ± 35*	135 ± 33	111 ± 30	106 ± 23*
Rambutan	30 g	136 ± 29	213 ± 29*	204 ± 26*	152 ± 32	119 ± 29	106 ± 19	107 ± 14
Pomelo	30 g	139 ± 35	208 ± 49*	224 ± 46*	168 ± 46	132 ± 48	115 ± 45	110 ± 39*
Rose apple	30 g	184 ± 95	223 ± 89*	243 ± 109*	203 ± 101	177 ± 101	162 ± 95	154 ± 90
Banana	30 g	185 ± 93	205 ± 91	219 ± 95*	190 ± 97	166 ± 92	153 ± 88*	150 ± 82*
Papaya	30 g	184 ± 98	208 ± 106	213 ± 106	192 ± 107	171 ± 99	155 ± 84	152 ± 87
					% change, %			
Tangerine	30 g	-	63 ± 22	75 ± 21	31 ± 18	-1.8 ± 21	-19 ± 15	-23 ± 12
Rambutan	30 g	-	60 ± 20	53 ± 19	13 ± 17	-12 ± 16	-21 ± 14	-19 ± 13
Pomelo	30 g	-	51 ± 20	62 ± 14	21 ± 19	-5.9 ± 19	-19 ± 14	-22 ± 10
Rose apple	30 g	-	28 ± 20	36 ± 26	10 ± 11	-6.7 ± 13	-15 ± 9	-18 ± 12
Banana	30 g	-	15 ± 15	22 ± 9	3 ± 5	-11 ± 9	-19 ± 8	-14 ± 16
Papaya	30 g	-	15 ± 9	17 ± 7	3 ± 13	-8.2 ± 14	-16 ± 13	-17 ± 14

Time (min) spans columns 0–300.

* Significant difference from baseline

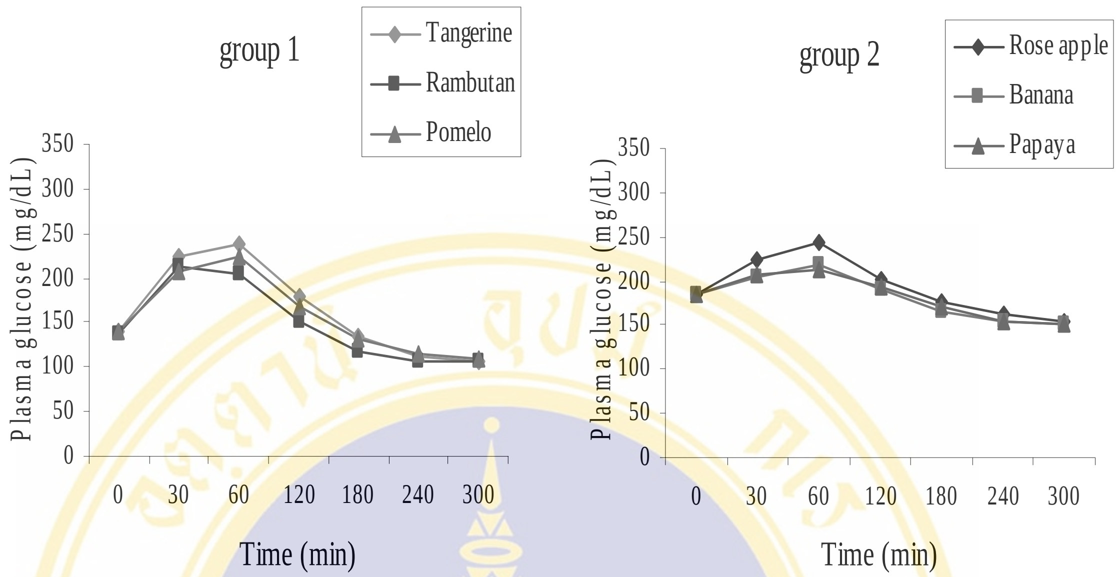


Figure 4.1 Mean plasma glucose after ingestion of 30 g CHO of different fruits

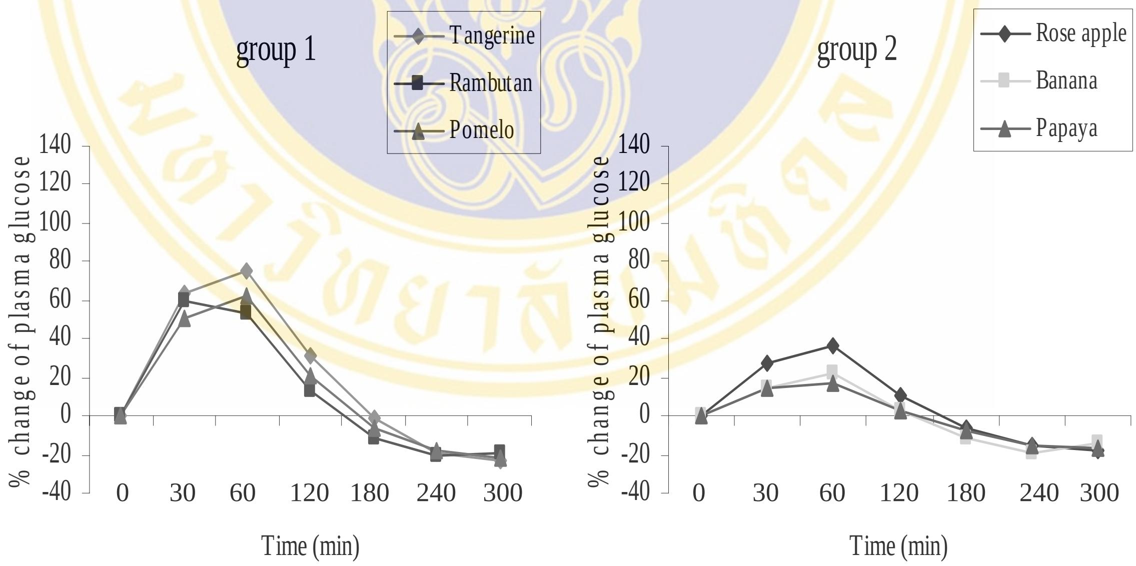


Figure 4.2 Mean percentage change of plasma glucose after ingestion of 30 g CHO of different fruits

Table 4.4 Plasma glucose (mean ± SD) responses and percentage changes after ingestion of 50 g CHO of different fruits

Kinds of fruit	Amount of CHO	0	30	60	120	180	240	300
					Mean ± SD, $mg.dL^{-1}$			
Glucose 1	50 g	144 ± 25	252 ± 40*	327 ± 58*	275 ± 45*	200 ± 56	146 ± 41	122 ± 31
Tangerine	50 g	144 ± 28	213 ± 31*	227 ± 35*	158 ± 38	121 ± 29	102 ± 20	101 ± 19*
Rambutan	50 g	149 ± 31	232 ± 54*	264 ± 56*	190 ± 51	149 ± 48	132 ± 40	123 ± 28*
Pomelo	50 g	157 ± 28	240 ± 40*	270 ± 35*	221 ± 47*	180 ± 43	145 ± 36	126 ± 32
Glucose 2	50 g	165 ± 72	241 ± 80*	291 ± 84*	261 ± 98*	194 ± 101	159 ± 96	142 ± 78
Rose apple	50 g	196 ± 110	250 ± 123	272 ± 125*	240 ± 136	197 ± 122	176 ± 114	157 ± 101
Banana	50 g	166 ± 81	195 ± 73	214 ± 86*	181 ± 83	157 ± 84	137 ± 69 *	137 ± 68
Papaya	50 g	168 ± 77	215 ± 102	232 ± 101*	194 ± 93	169 ± 90	149 ± 85	139 ± 76
					% change, %			
Glucose 1	50 g	-	77 ± 25	130 ± 35	94 ± 29	38 ± 31	1.2 ± 23	-15 ± 15
Tangerine	50 g	-	51 ± 24	60 ± 25 ^{G1}	12 ± 28 ^{G1}	-15 ± 19 ^{G1}	28 ± 12	-30 ± 6
Rambutan	50 g	-	54 ± 13	80 ± 10 ^{G1}	27 ± 23 ^{G1}	-2 ± 23	-14 ± 17	-17 ± 9
Pomelo	50 g	-	54 ± 21	73 ± 13 ^{G1}	42 ± 25 ^{G1}	14 ± 20	-7.9 ± 15	-20 ± 14
Glucose 2	50 g	-	54 ± 38	88 ± 44	62 ± 19	15 ± 25	-8.9 ± 26	-16 ± 16
Rose apple	50 g	-	36 ± 32	47 ± 37	24 ± 30	0.6 ± 16	-12 ± 16	-21 ± 14
Banana	50 g	-	23 ± 22	34 ± 18	9.5 ± 9 ^{G2}	7.4 ± 10	-17 ± 7	-17 ± 9
Papaya	50 g	-	27 ± 10	37 ± 26	15 ± 15 ^{G2}	-2 ± 12	-15 ± 11	-19 ± 12

* Significant difference from baseline

$^{G1, G2}$ Significant difference from glucose 1 and glucose 2 at the same time period at $p < 0.05$ level

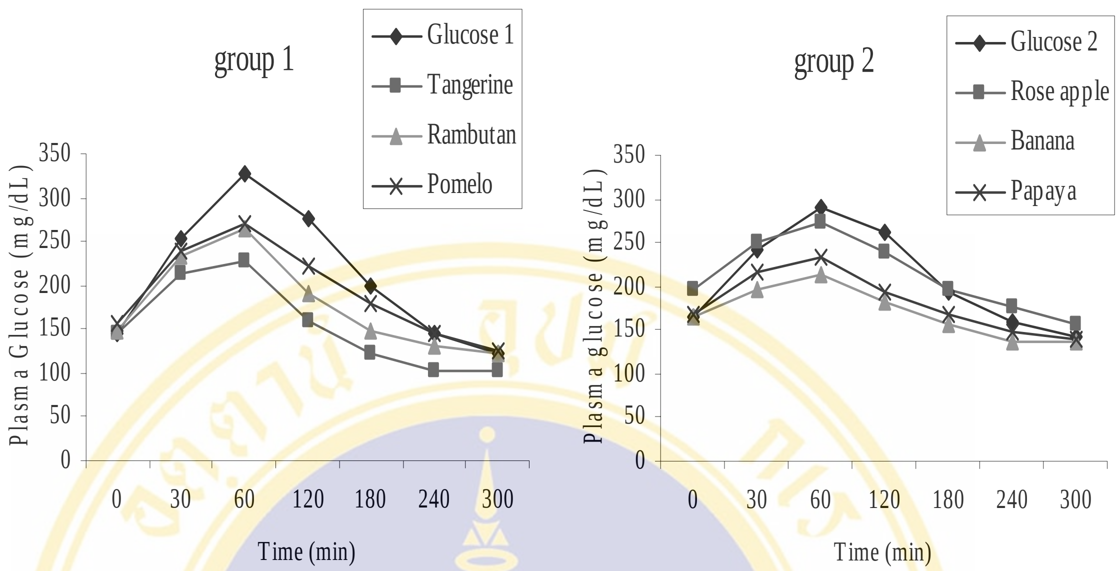


Figure 4.3 Mean plasma glucose responses after ingestion of 50 g CHO of different fruits

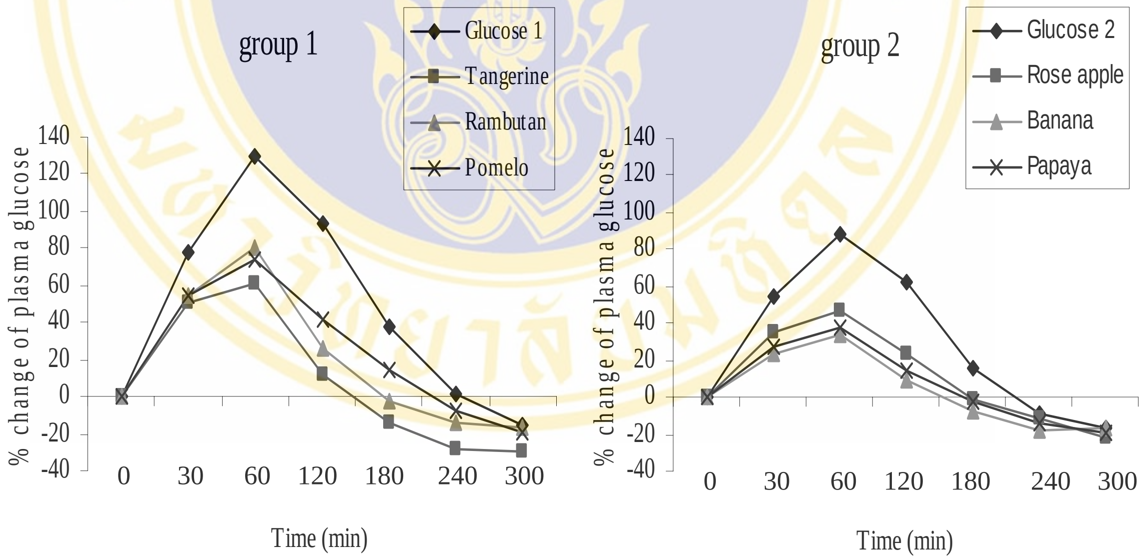


Figure 4.4 Mean percentage change of plasma glucose responses after ingestion of 50 g CHO of different fruits

Areas Under the Plasma Glucose Responses Curve of Fruits and their Glycemic Indexes

The areas under the plasma glucose response curves (AUC) above baseline for each fruit are shown in Table 4.5 and Figure 4.5-4.6. After ingestion of 30 g CHO of different fruits, tangerine showed the highest AUC. After ingestion of 50 g CHO, pomelo showed the highest AUC. Moreover, AUC after both amounts ingestion of banana was less than other fruits.

Comparing between the different amounts of CHO (30 and 50 g), the significant difference of AUC was seen between rambutan, pomelo, banana and papaya (Table 4.5, Figure 4.6).

The glycemic indexes of 6 Thai fruits are shown in Table 4.5 and Figure 4.7. The GI of banana, tangerine, papaya, rambutan, rose apple and pomelo were 26, 30, 38, 47, 50 and 59, respectively.

Table 4.5 Area under the glucose- response curves (mg.hr.dL^{-1}) after ingestion of different amounts of fruits and their glycemic indexes

Kinds of fruit	30 g CHO		50 g CHO		GI
	Mean ± SD	Glycemic rank	Mean ± SD	Glycemic rank	
Glucose gr.1			23740 ± 7190		
Tangerine	9545 ± 2461	1	7087 ± 3251	5	30
Pomelo	7905 ± 2619	2	13136 ± 3895 *	1	59
Rambutan	6556 ± 2649	3	11107 ± 4043 *	3	47
Glucose gr. 2			16275 ± 5073		
Rose apple	5053 ± 2181	4	8563 ± 5452	2	50
Papaya	3018 ± 2503	5	6327 ± 3476 *	4	38
Banana	2455 ± 643	6	3964 ± 1100 *	6	26

* significant difference from the same fruit of different dose

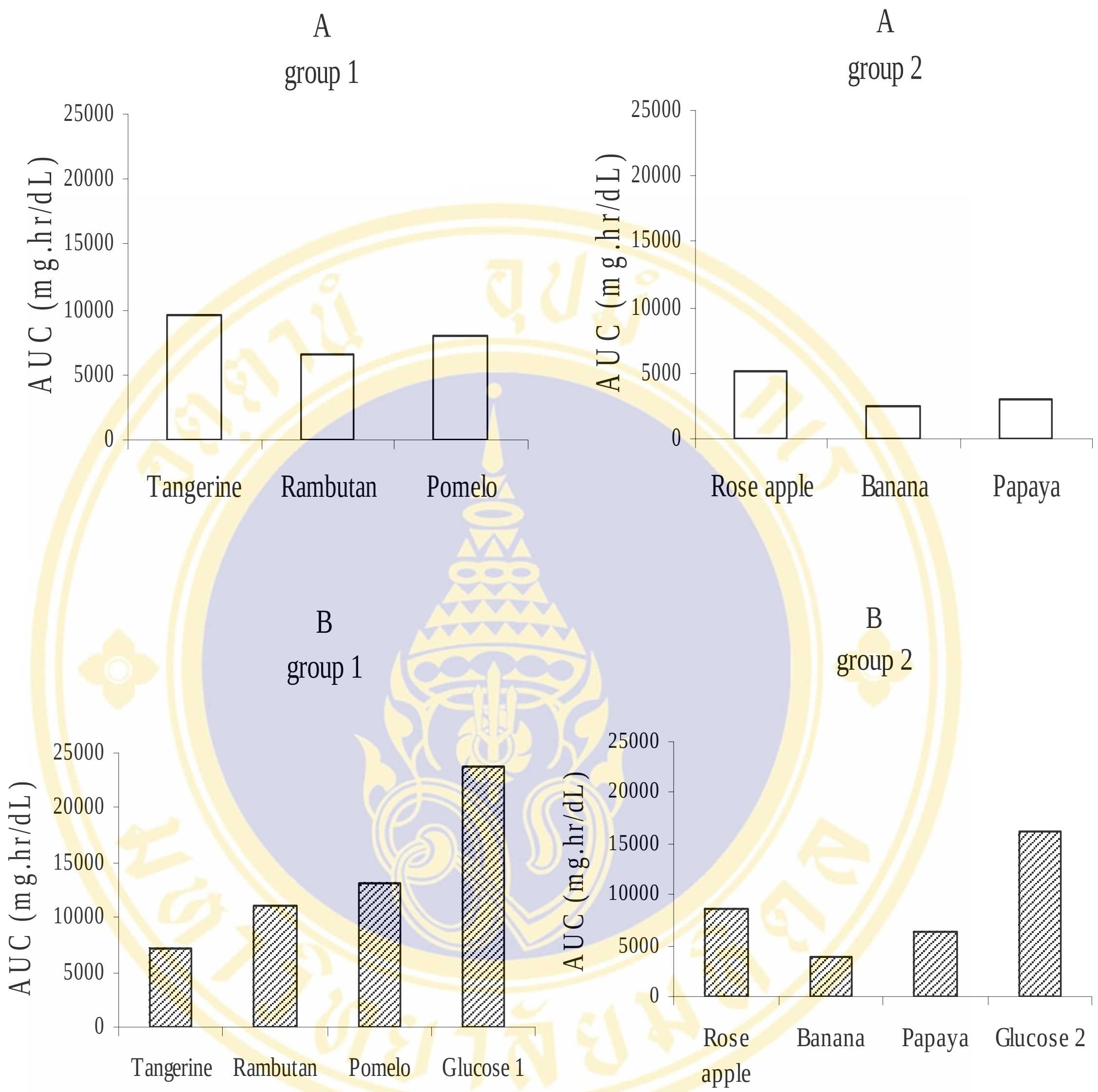
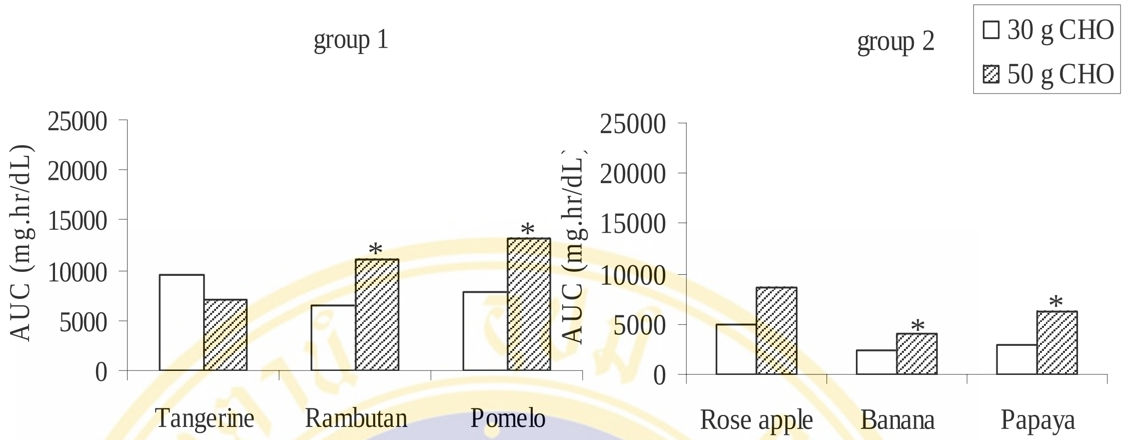


Figure 4.5 Mean areas under the glucose response curves after ingestion of 30 g CHO (A) and 50 g CHO (B) of different fruits



* significant difference between 30 g and 50 g CHO

Figure 4.6 Mean areas under the glucose response curves after ingestion of 30 g and 50 g CHO of different fruits

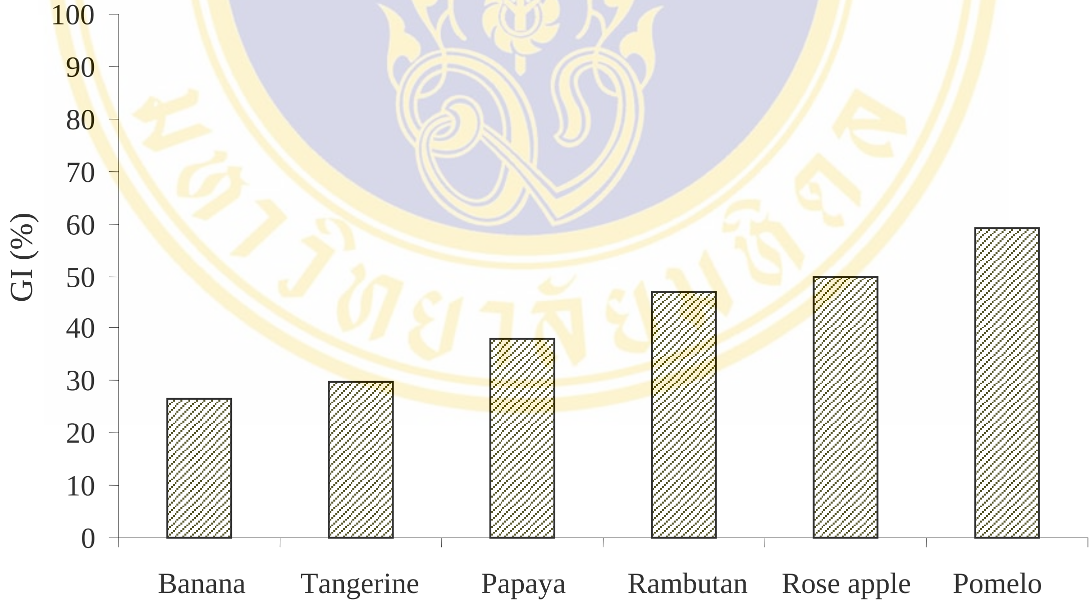


Figure 4.7 Glycemic indexes of 6 Thai fruits (50 g CHO)

Serum Lipids

Serum triglycerides levels

Table 4.6 and Figure 4.8-4.9 show the serum triglyceride levels and the percentage changes at 300 minutes after ingestion of different amounts of fruits. After ingestion of 30 g CHO of tangerine, rambutan and pomelo, mean triglyceride levels at 300 minutes were significantly increased ($p < 0.05$) from baseline. After ingestion of 50 g CHO of tangerine, rambutan, pomelo, and banana, mean triglyceride levels at 300 minutes were significantly increased ($p < 0.05$) whereas no significant elevation of triglyceride was detected after ingestion of both amounts of papaya and rose apple.

There was no significant difference in the percentage changes of serum triglycerides among all fruits after ingestion of the same carbohydrate doses. However, the percentage change of 50 g CHO doses of banana and pomelo were significantly higher ($p < 0.05$) than their 30 g CHO doses (Figure 4.9)

Serum cholesterol levels

Table 4.7 and Figure 4.10-4.11 show the serum cholesterol levels and the percentage changes at 300 minutes after ingestion of different amounts of fruits. At 300 minutes, after ingestion 30 and 50 g CHO of different fruits, there was only slight variation of the cholesterol levels (Figure 4.10- 4.11).

The percentage changes of serum cholesterol at 300 minutes between all fruits (Figure4.11) after ingestion 30 and 50 g CHO were not significantly different ($P < 0.05$).

Comparing between the doses (30 g and 50 g CHO), there was no significant differences in the percentage changes of serum cholesterol after ingestion of different amounts of all fruits (Figure 4.11).

Serum HDL-C levels

Table 4.8 and Figure 4.12-4.13 show the serum HDL cholesterol levels and the percentage changes at 300 minutes after ingestion different amounts of fruits. After ingestion 30 g and 50 g CHO of pomelo, serum HDL-C levels were significantly ($p < 0.05$) decreased. Moreover serum HDL-C levels after 50 g CHO of rambutan were significantly decreased from baseline.

The percentage changes of serum HDL-C at 300 minutes between all fruits after ingestion of 30 and 50 g CHO were not significantly different ($P < 0.05$) (Table 4.8).

Comparing between the doses (30 g and 50 g CHO), there was no significant difference in the serum HDL-C after ingesting different amounts of all fruits (Figure 4.13).

Serum LDL-C levels

Table 4.9 and Figure 4.14-4.15 show the serum LDL cholesterol levels and the percentage changes at 300 minutes after ingestion of different amounts of fruits. After ingestion 50 g CHO of rambutan, pomelo and papaya serum LDL-C levels were significantly ($p < 0.05$) decreased.

The percentage changes of serum LDL-C at 300 minutes between all fruits after ingestion of 30 and 50 g CHO (Table 4.15) were not significantly different ($P < 0.05$) between them.

Comparing between the doses (30 g and 50 g CHO), there was a significant ($P < 0.05$) reduction in the percentage change of serum LDL-C after ingestion of the bigger dose of pomelo (Table 4.9).

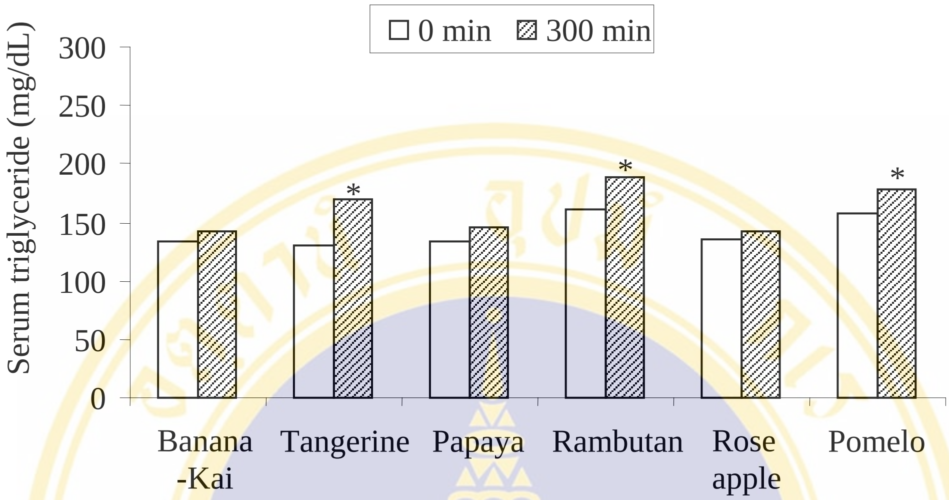
Table 4.6 Actual levels of serum triglyceride at baseline and 300 minutes and percentage changes from baseline at 300 minutes after ingestion of different amounts of fruits

Kinds of fruit	Amounts of CHO (g)	$mg.dL^{-1}$ at 0 min	$mg.dL^{-1}$ at 300 min	% change at 300 min
Banana-Kai	30	134 ± 58	143 ± 59	7.89 ± 25
Tangerine	30	131 ± 50	170 ± 51 *	35.3 ± 26
Papaya	30	134 ± 55	146 ± 62	10.8 ± 24
Rambutan	30	161 ± 65	188 ± 59 *	20 ± 23
Rose apple	30	135 ± 46	143 ± 42	12.3 ± 27
Pomelo	30	158 ± 57	178 ± 52 *	14.6 ± 12
Glucose 1	50	144 ± 38	169 ± 41	18.3 ± 13
Glucose 2	50	138 ± 52	161 ± 62	18.9 ± 21
Banana-Kai	50	123 ± 55	156 ± 63 *	31.6 ± 21 ^{Ba}
Tangerine	50	153 ± 42	195 ± 41 *	29.5 ± 16
Papaya	50	131 ± 59	155 ± 64	26 ± 31
Rambutan	50	167 ± 76	205 ± 63 *	28 ± 19
Rose apple	50	157 ± 57	173 ± 62	17.6 ± 38
Pomelo	50	121 ± 32	153 ± 37 *	27.4 ± 15 ^{P}

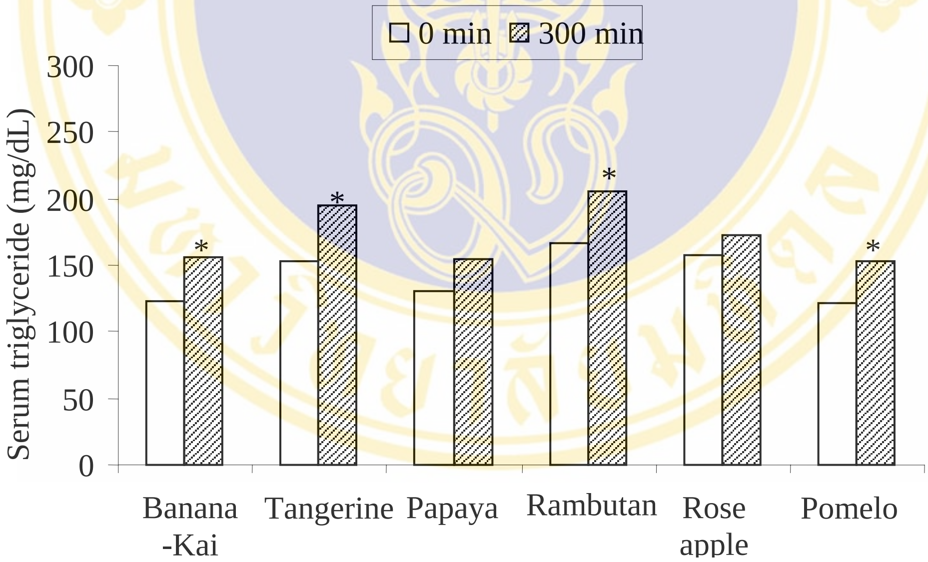
* significant difference from baseline at $p < 0.05$ level

^{P,Ba} significant difference from 30 g CHO of pomelo and banana at $p < 0.05$ level

(A)

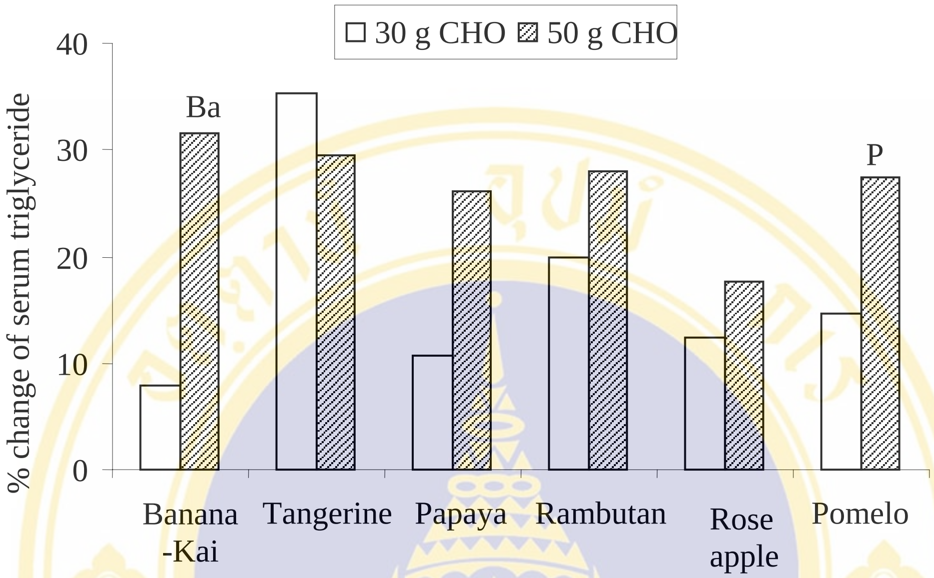


(B)



* significant difference from baseline

Figure 4.8 Mean serum triglyceride level before and 5 h after ingestion of 30 g CHO (A) and 50 g CHO (B) of different fruits



[Ba,P] significant difference from 30 g CHO of banana and pomelo

Figure 4.9 Mean percentage changes of serum triglyceride levels at 5 h after ingestion of 30 g and 50 g CHO of different fruits

Table 4.7 Actual levels of serum cholesterol at baseline and 300 minutes and percentage changes from baseline at 300 minutes after ingestion of different amounts of fruits

Kinds of fruit	Amounts of CHO (g)	$mg.dL^{-1}$ at 0 min	$mg.dL^{-1}$ at 300 min	% change at 300 min
Banana-Kai	30	193 ± 36	195 ± 44	0.35 ± 4
Tangerine	30	201 ± 31	197 ± 40	-2.70 ± 9
Papaya	30	191 ± 33	195 ± 42	1.41 ± 6
Rambutan	30	239 ± 34	238 ± 37	-0.50 ± 6
Rose apple	30	185 ± 34	186 ± 43	-0.10 ± 6
Pomelo	30	205 ± 34	209 ± 34	1.89 ± 4
Glucose 1	50	203 ± 33	200 ± 40	-1.90 ± 6
Glucose 2	50	189 ± 22	192 ± 28	1.77 ± 5
Banana-Kai	50	185 ± 27	189 ± 29	2.55 ± 4
Tangerine	50	192 ± 30	199 ± 34	3.55 ± 5
Papaya	50	184 ± 24	180 ± 22	-2.40 ± 3
Rambutan	50	240 ± 37	235 ± 37	-1.70 ± 3
Rose apple	50	204 ± 29	204 ± 30	0.08 ± 3
Pomelo	50	181 ± 28	181 ± 31	-0.20 ± 4

No significant difference between baseline and postprandial serum cholesterol at $p < 0.05$ level

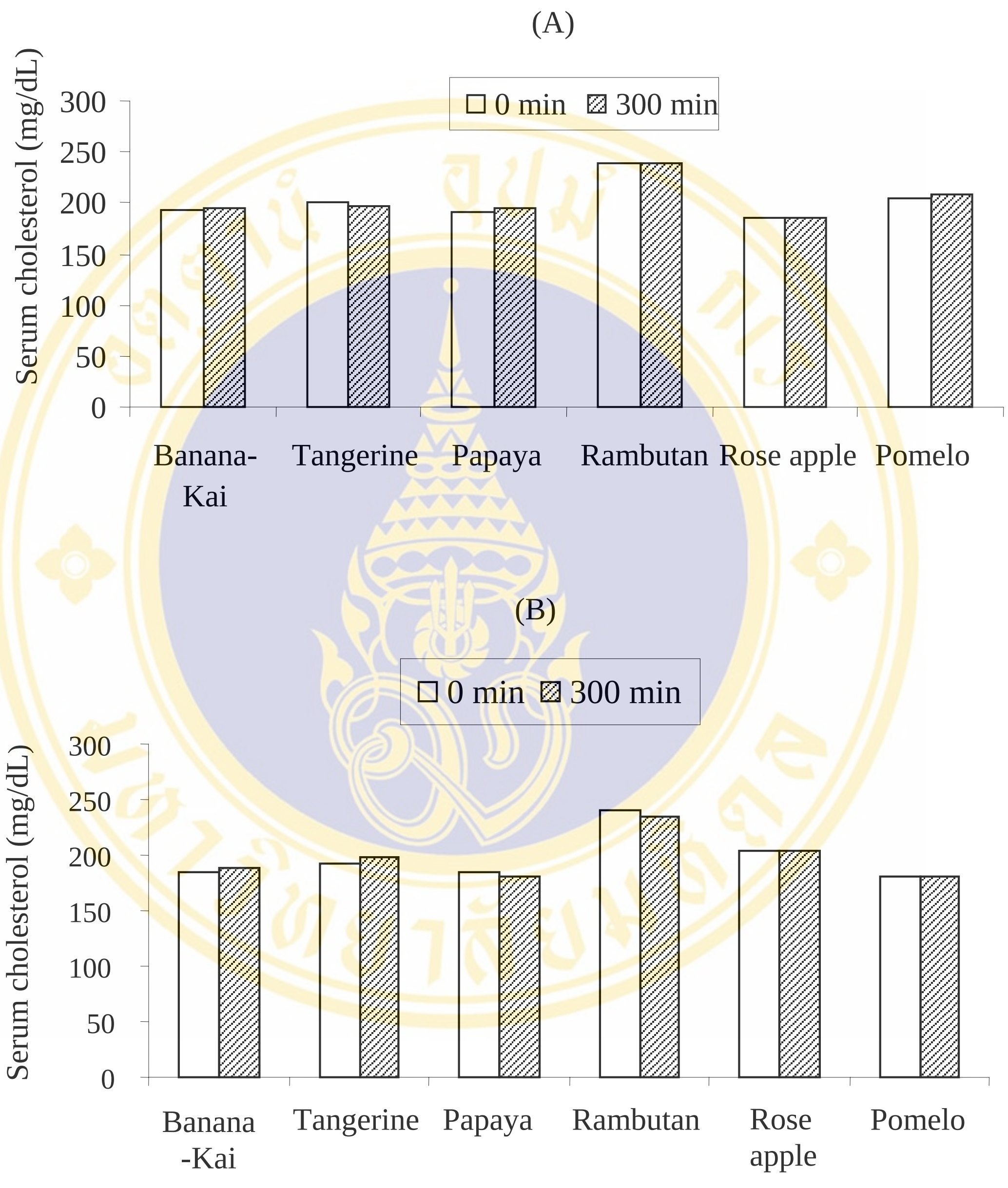


Figure 4.10 Mean serum cholesterol levels before and 5 h after ingestion of 30 g CHO (A) and 50 g CHO (B) of different fruits

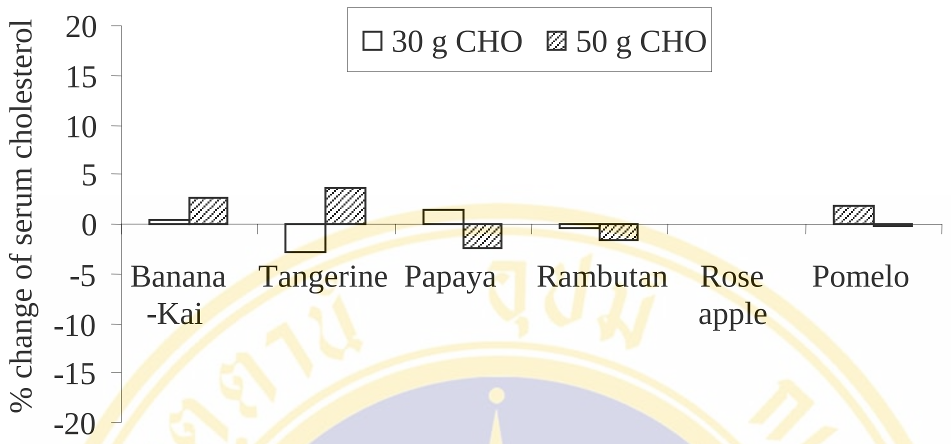
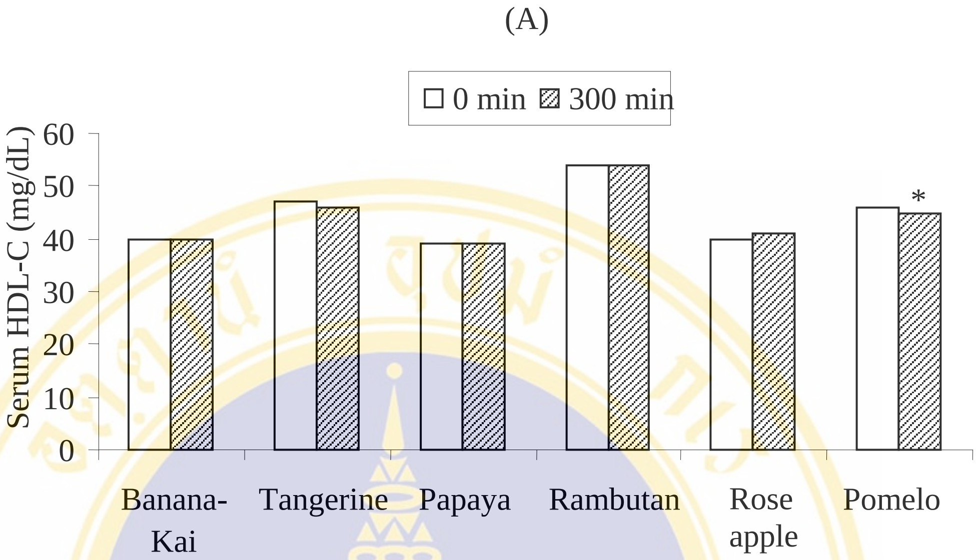


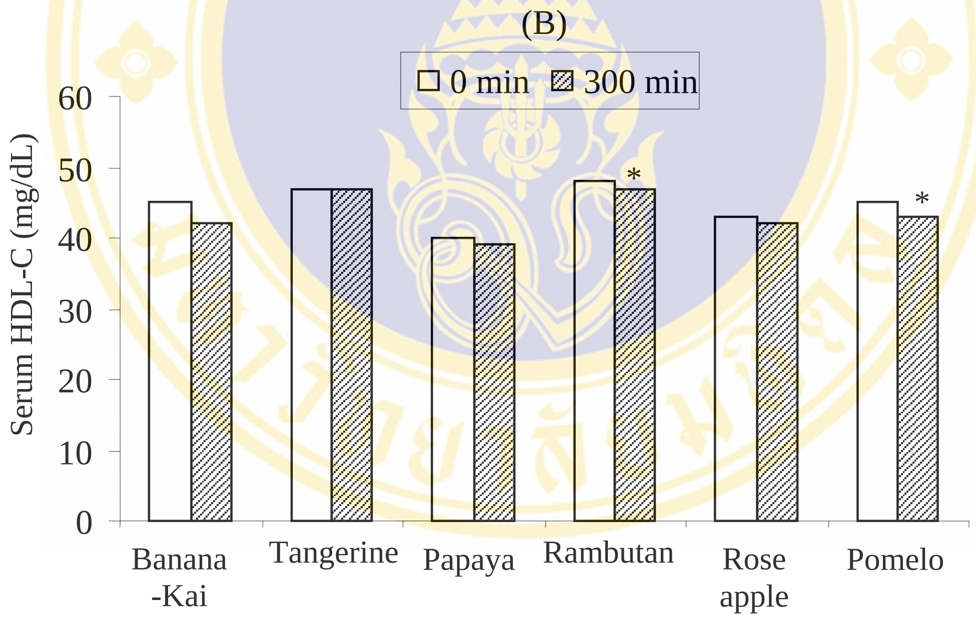
Figure 4.11 Mean percentage changes of serum cholesterol levels at 5 h after ingestion of 30 g and 50 g CHO of different fruits

Table 4.8 Actual levels of serum HDL-C at baseline and 300 minutes and percentage changes from baseline at 300 minutes after ingestion of different amounts of fruits.

Kinds of fruit	Amounts of CHO (g)	$mg.dL^{-1}$ at 0 min	$mg.dL^{-1}$ at 300 min	% change at 300 min
Banana-Kai	30	40 ± 8	40 ± 10	-0.07 ± 8
Tangerine	30	47 ± 7	46 ± 5	-1.03 ± 6
Papaya	30	39 ± 7	39 ± 8	0.92 ± 5
Rambutan	30	54 ± 10	54 ± 10	-0.28 ± 4
Rose apple	30	40 ± 5	41 ± 6	2.69 ± 9
Pomelo	30	46 ± 8	45 ± 7 *	-3.90 ± 4
Glucose 1	50	46 ± 8	42 ± 7	-6.89 ± 5
Glucose 2	50	43 ± 8	43 ± 7	0.89 ± 9
Banana-Kai	50	45 ± 10	42 ± 10	-5.44 ± 8
Tangerine	50	47 ± 7	47 ± 8	0.46 ± 7
Papaya	50	40 ± 11	39 ± 10	-1.63 ± 5
Rambutan	50	48 ± 15	47 ± 15 *	-4.18 ± 4
Rose apple	50	43 ± 8	42 ± 9	-1.68 ± 6
Pomelo	50	45 ± 7	43 ± 7 *	-4.68 ± 4

* significant difference from baseline at $p < 0.05$ level





*Significant difference from baseline

Figure 4.12 Mean serum HDL-C level before and 5 h after ingestion of 30 g CHO (A) and 50 g CHO (B) of different fruits

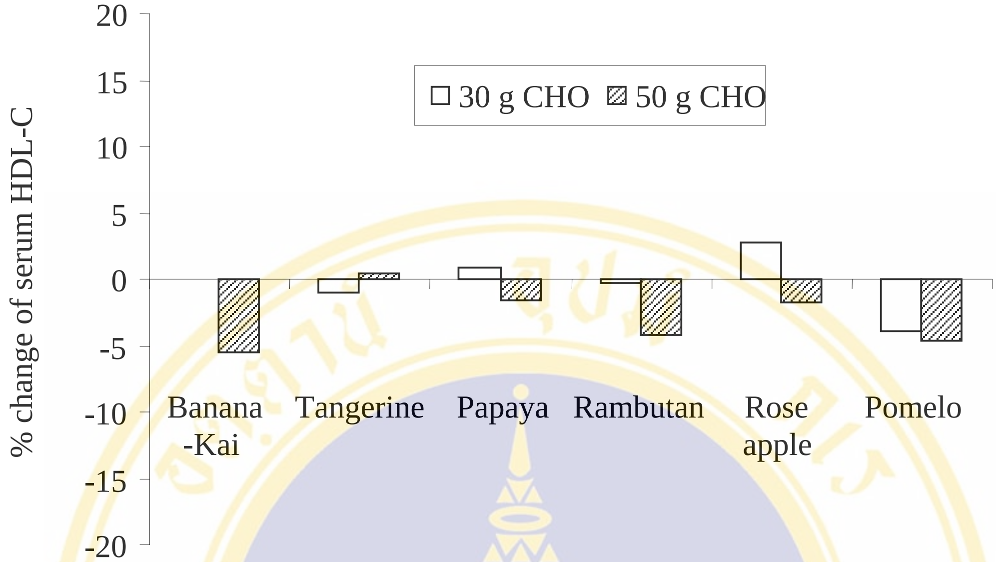


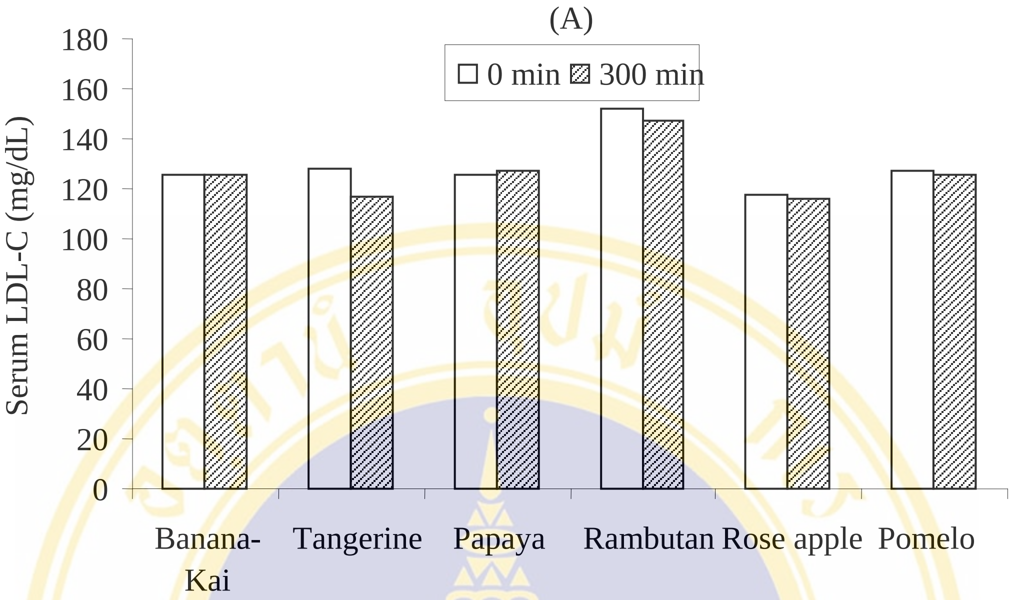
Figure 4.13 Mean percentage changes of serum HDL-C level at 5 h after ingestion of 30 g and 50 g CHO of different fruits

Table 4.9 Actual levels of serum LDL-C at baseline and 300 minutes and percentage changes from baseline at 300 minutes after ingestion of different amounts of fruits

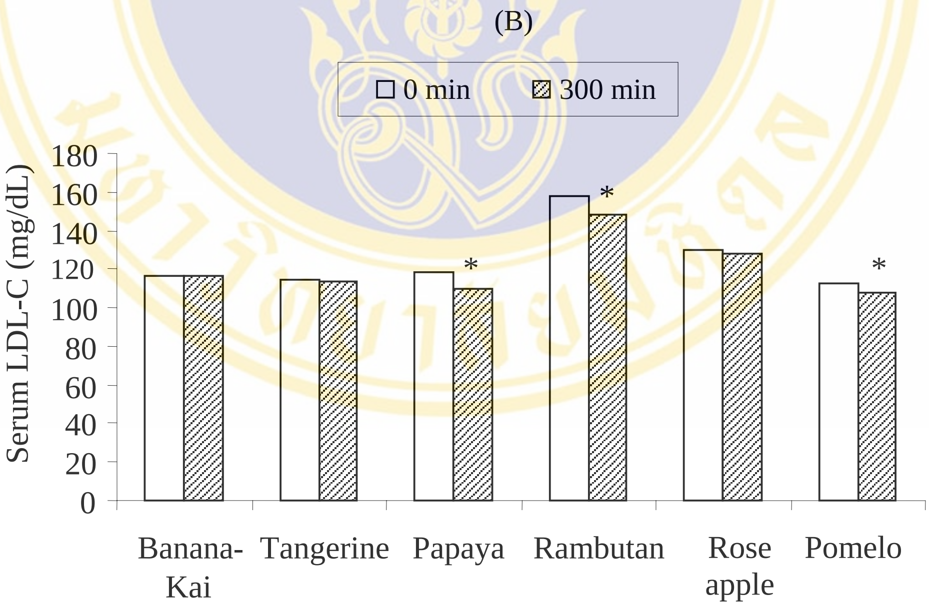
Kinds of fruit	Amounts of CHO (g)	$mg.dL^{-1}$ at 0 min	$mg.dL^{-1}$ at 300 min	% change at 300 min
Banana-Kai	30	126 ± 30	126 ± 37	-0.93 ± 6
Tangerine	30	128 ± 32	117 ± 42	-11.1 ± 16
Papaya	30	126 ± 27	127 ± 33	0.4 ± 9
Rambutan	30	152 ± 28	147 ± 28	-3.49 ± 9
Rose apple	30	118 ± 32	116 ± 39	-2.11 ± 11
Pomelo	30	127 ± 31	126 ± 30	-0.38 ± 5
Glucose 1	50	129 ± 30	125 ± 34	-3.85 ± 10
Glucose 2	50	118 ± 17	117 ± 21	-0.54 ± 8
Banana-Kai	50	116 ± 18	116 ± 15	0.89 ± 8
Tangerine	50	115 ± 31	114 ± 33	-1.47 ± 6
Papaya	50	118 ± 17	110 ± 18 *	-6.86 ± 8
Rambutan	50	158 ± 32	148 ± 30 *	-6.64 ± 4
Rose apple	50	130 ± 18	128 ± 19	-1.44 ± 9
Pomelo	50	113 ± 31	108 ± 32 *	-4.86 ± 5 [P]

* significant difference from baseline at $p < 0.05$

[P] significant difference from 30 g CHO of pomelo at $p < 0.05$ level

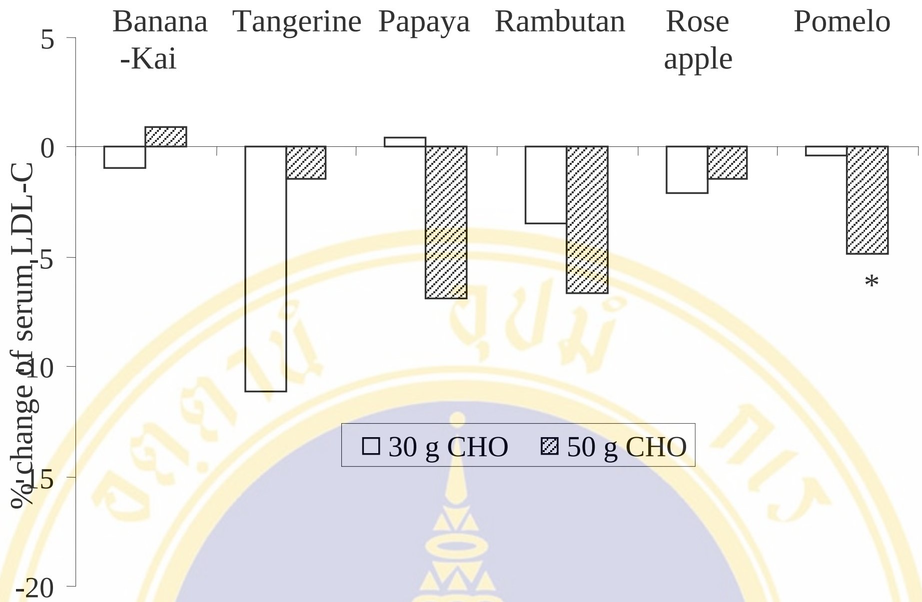


No significant difference between baseline and postprandial serum lipid



* Significant difference from baseline at P < 0.05 level

Figure 4.14 Mean serum LDL-C level before and 5 h after ingestion of 30 g CHO (A) and 50 g CHO (B) of different fruits



* significant difference between 30 g and 50 g CHO of pomelo

Figure 4.15 Mean percentage changes of serum LDL-C level at 5 h after ingestion of 30 g and 50 g CHO of different fruits

Part III Blood Rheology

Table 4.10-4.11 and Figure 4.16-4.18 show actual values of blood viscosity and percentage changes after ingestion of different amounts of fruits at the shear rate of 264 sec^{-1} at 37 °C during the peaks of hyperglycemia and hyperlipidemia at 60 and 300 minutes, respectively.

Whole blood viscosity tended to be decreased at 60 minutes after ingestion of 30 g CHO of every fruit; especially after eating tangerine, rambutan, pomelo and papaya which reached statistically different ($p < 0.05$) from baseline and returned to baseline levels at 300 minutes (Table 4.10, Figure 4.16). After ingestion of 50 g CHO of different fruits and glucose drink, blood viscosity showed the same trend. The significant reduction from baseline at 60 minutes was seen in tangerine, rambutan, pomelo, papaya and banana ($p < 0.05$) (Table 4.11, Figure 4.16). There was no dose difference in the changes of viscosity. There was no significant difference in the blood rheological changes between different kinds and amounts of fruits (Figure 4.17-4.18).

Table 4.10 Actual levels of blood viscosity (at 37°c) at baseline, 60 and 300 minutes and percentage changes after ingestion of 30 g CHO of different fruits

Kinds of fruit	Amounts of CHO (g)	Whole blood viscosity 0 min	60 min	300 min
			mPa.s	
Banana	30	3.56 ± 0.47	3.53 ± 0.53	3.64 ± 0.63
Tangerine	30	3.31 ± 0.21	3.11 ± 0.22 *	3.37 ± 0.29
Papaya	30	3.45 ± 0.41	3.25 ± 0.42 *	3.62 ± 0.49
Rambutan	30	3.50 ± 0.30	3.30 ± 0.27 *	3.52 ± 0.23
Rose apple	30	3.68 ± 0.40	3.46 ± 0.41	3.58 ± 0.46
Pomelo	30	3.50 ± 0.28	3.30 ± 0.17 *	3.53 ± 0.36
			% change	
Banana	30	-	-0.87 ± 3.55 [T]	1.98 ± 6.98
Tangerine	30	-	-6.15 ± 0.67	1.64 ± 4.24
Papaya	30	-	-5.89 ± 3.25	4.81 ± 6.37
Rambutan	30	-	-5.72 ± 2.61	1 ± 5.23
Rose apple	30	-	-5.93 ± 6.90	-2.78 ± 6.70
Pomelo	30	-	-5.38 ± 3.72	-0.81 ± 4.82

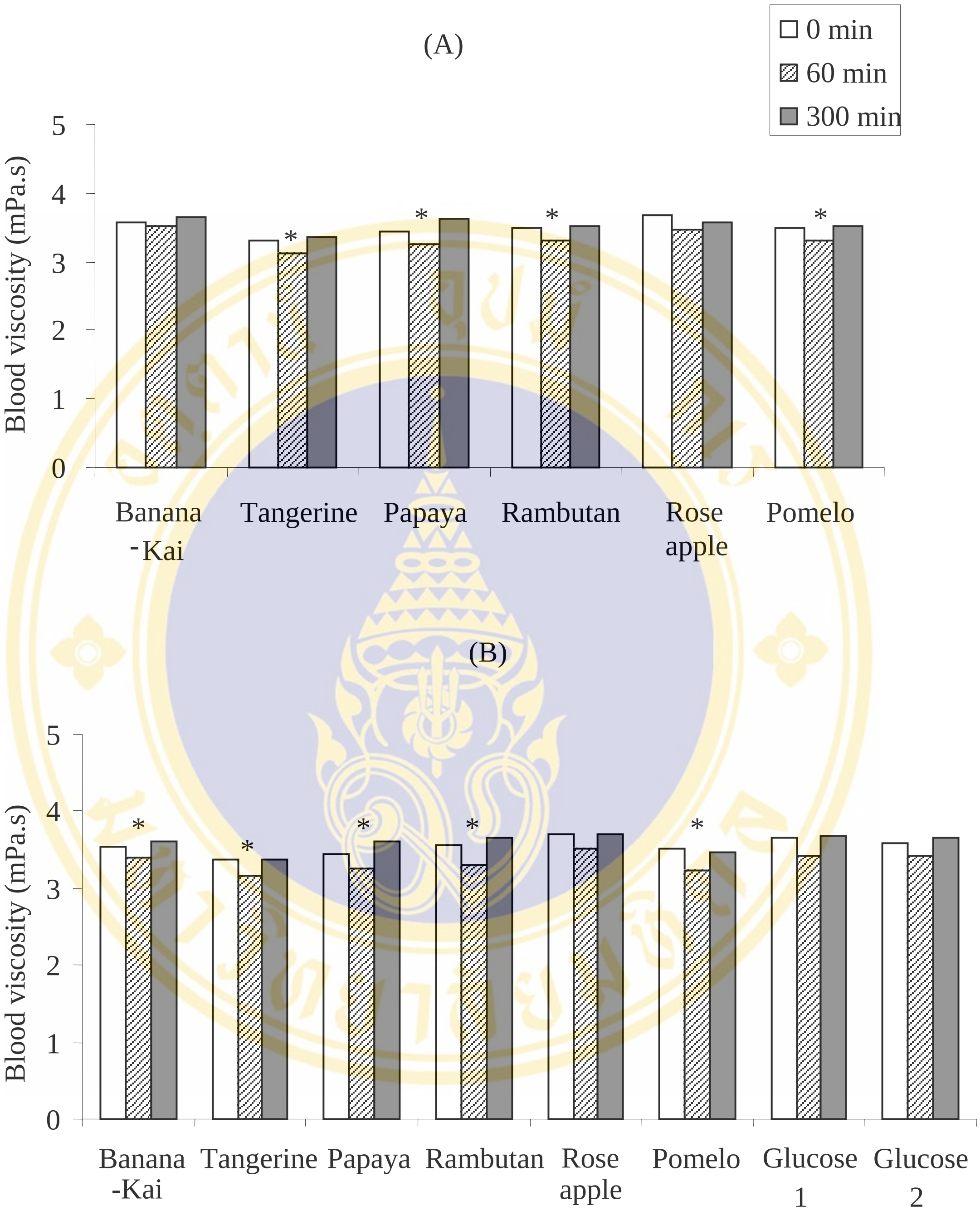
* Significant different from baseline at $p < 0.05$

[T] Significant difference from tangerine at the same time period at $p < 0.05$ level

Table 4.11 Actual levels of blood viscosity (at 37°c) at baseline, 60 and 300 minutes and percentage changes after ingestion of 50 g of different fruits

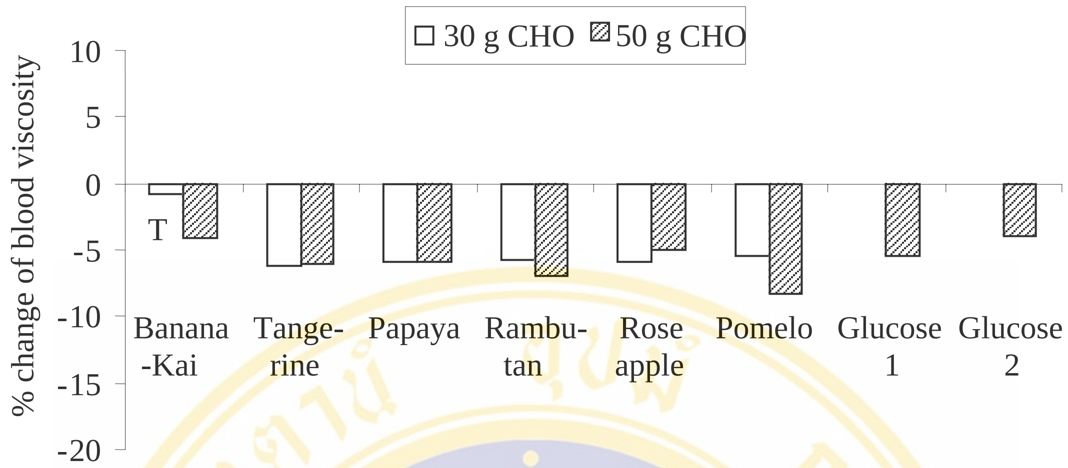
Kinds of fruit	Amounts of CHO (g)	Whole blood viscosity 0 min	60 min	300 min
			mPa.s	
Glucose 1	50	3.65 ± 0.45	3.43 ± 0.25	3.67 ± 0.30
Tangerine	50	3.38 ± 0.11	3.17 ± 0.19 *	3.38 ± 0.14
Rambutan	50	3.55 ± 0.26	3.31 ± 0.31 *	3.66 ± 0.58
Pomelo	50	3.52 ± 0.19	3.22 ± 0.20 *	3.47 ± 0.27
Glucose 2	50	3.58 ± 0.30	3.43 ± 0.28	3.65 ± 0.40
Rose apple	50	3.70 ± 0.49	3.51 ± 0.46	3.7 ± 0.43
Banana-Kai	50	3.54 ± 0.41	3.39 ± 0.35 *	3.62 ± 0.52
Papaya	50	3.45 ± 0.41	3.25 ± 0.42 *	3.62 ± 0.49
			% change	
Glucose 1	50	-	-5.44 ± 6.71	1.16 ± 7.53
Tangerine	50	-	-6.04 ± 3.43	0.21 ± 1.80
Rambutan	50	-	-6.91 ± 3.30	3.39 ± 15.06
Pomelo	50	-	-8.35 ± 2.57	-1.30 ± 2.75
Glucose 2	50	-	-3.95 ± 5.27	2.04 ± 8.23
Rose apple	50	-	-5.04 ± 6.27	0.36 ± 3.89
Banana-Kai	50	-	-4.16 ± 2.63	2.13 ± 7.54
Papaya	50	-	-5.89 ± 3.25	4.81 ± 6.37

* Significant different from baseline at $p < 0.05$ level



* significant difference from baseline

Figure 4.16 Mean whole blood viscosity levels before, 60 min and 300 min after ingestion of 30 g CHO (A) and 50 g CHO (B) of different fruits



[T] Significant difference between 30 g CHO of banana and 30 g CHO of tangerine at the same time period at $p < 0.05$ level

Figure 4.17 Mean percentage change of blood viscosity at 60 minutes after ingestion of different amount of fruits

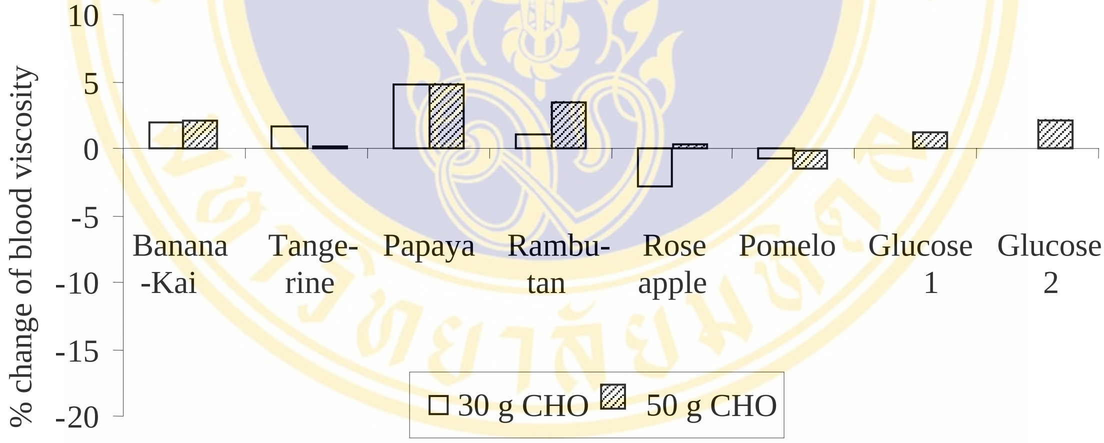


Figure 4.18 Mean percentage change of blood viscosity at 300 min after ingestion of different amount of fruits

CHAPTER V

DISCUSSION

The reasons for conducting this study was to assess the effects of sugar and carbohydrate combination as well as dietary fiber contained in some Thai fruits on postprandial plasma glucose and lipid concentrations, blood viscosity and their glycemic indexes. The difference between the test fruits were the type of sugars and dietary fibers they contained. This study was also designed to show the metabolic changes of glycemic load by comparing the two amounts of carbohydrate (30 g and 50 g CHO) from each kind of fruit.

Metabolic Responses of Fruits

Plasma Glucose Responses, Area Under the Glucose Response Curve After Ingestion of Different Amounts of Fruits.

The peaks of plasma glucose and AUC after 50 g CHO of glucose ingestion were higher than fruits. The reasons were fruits contained less glucose and more of other sugars with lower glycemic responses as shown in other studies (143-145) and confirmed by the sugar contents and dietary fiber contents in the fruit composition data analyzed (Table 3.3).

In the clinical setting, this lower response should be beneficial to the diabetic patients because rapidly absorbed carbohydrate (whether in the form of sugar and starches) produce large insulin responses (146); hence worsening of the glycemic control and is harmful to those individuals at risk for diabetes.

To rationalize the advice of fruits in diabetic patients, it is important to know their chemical composition and above all their biological responses. The study of Crapo et al. (125) as well as Wolever et al. (147) and Gannon et al. (144) suggested that different kinds of sugar created different plasma glucose responses. Glycemic indexes of glucose, sucrose and fructose are 100, 61, and 23, respectively.

Both doses of pomelo and rambutan gave high glycemic response and glycemic index than other fruits (Table 4.5 Figure 4.5 – 4.7) though pomelo and rambutan contained lower glucose and fructose with higher sucrose content (Table 3.4). These results may be explained by the more rapidly hydrolysed and transported of disaccharides than free glucose as shown by Southgate (109). In addition, the denser texture and higher total dietary fiber contained in rambutan (Table 3.4) as compared to pomelo reduced the digestion rate; thus glycemic response of rambutan was lower.

Whereas, tangerine contains almost the same amount of sucrose as pomelo and rambutan, it produced highest peak of plasma glucose response and area of glucose response and gave delayed return to baseline because it contained moderate amount of glucose, and sucrose which is rapidly hydrolysed. However, GI of tangerine was low; this was likely due to the effect of high total dietary fiber as compared to pomelo and rambutan (Table 3.4). These results demonstrated that total dietary fiber contained in the 50 g CHO of tangerine may be a proper amount needed to modulate the absorption of simple sugar; total dietary fiber of fruits may be more important than the amount of soluble fiber. In addition, the study design of this research was that all subjects in each group consumed one dose and one fruit in the test day. Tangerine of 50 g CHO test was less sweet than tangerine of 30 g CHO test and the interval of test day between of tangerine tests was longer than other fruit tests. The tangerines in 2 tests were come from different season. The different in the seasons of tangerine may be cause the variation of carbohydrate compositions in tangerines.

Several studies in type 2 DM subjects have found that fructose ingestion produced lower plasma glucose and insulin responses than sucrose and glucose (124, 126, 128, 148). Although rose apple contained highest fructose amount (Table 3.4) but it also contains an equivalent amount of glucose, resulted in moderate elevation of the glucose response. In addition, rose apple contains almost no starch in spite of its high dietary fiber content. This may explain why rose apple has rather high GI, 50.

Though tangerine contained highest total glucose equivalent (sucrose + glucose) as compared to pomelo and rambutan, its glycemic effect was not high (GI = 30). This was due to its high total dietary fiber (Table 3.4) which contrasted to rambutan and pomelo which had rather low total dietary fiber. Hence, pomelo produced the highest GI (= 59).

In our data, banana gave the lowest glycemic response and area under the glucose response curve in both amount of carbohydrate although it contained higher glucose than tangerine, papaya, rambutan, and pomelo and low dietary fiber especially soluble fiber content (Table 3.4). It is likely that this was due to its lower total sugars to starch ratio. Vaaler et al (149) exhibited apple and banana produced less glucose response than other fruits. They thought that small amount of starch in banana and apple might have contributed to their lower blood glucose response compared to the other no starch content fruits tested. This was supported by finding from Faisant et al (150) who showed that starch content of banana was hydrolysed slowly by alfa-amylase in human. Chaichumporn R. (151) also demonstrated that green mature mangoes in which total sugars to starch ratio lower than ripe mangoes produced plasma glucose response and glucose area lower than the response of ripe mangoes.

Ingestion of both doses of rose apple and tangerine, the areas of glucose responses were not significantly different. It can be mentioned that in practice 2 to 3 servings of these two fruits should not deteriorate the glycemic responses. However, area under the glucose response curves after ingestion of 2 and 3 servings of pomelo, rambutan, papaya, and banana were significantly different. Therefore, increased amount of these fruits did alter the glycemic response.

Foods were classified into the glycemic index classes suggested by Brand-Miller et al (152) as low GI less than 55, medium GI between 55-70, and high GI more than 70. In our study, GI of pomelo, rose apple, rambutan, papaya, tangerine, and banana were 59, 50, 47, 38, 30, and 26, respectively. From these results, rose apple, rambutan, papaya, tangerine, and banana were low GI foods while pomelo was a medium GI food.

Lunetta M (153) showed a significant positive correlation of glycemic indexes with total glucose content and an inverse correlation with fructose content of fruits and no correlation between fiber content and GI. However, Wolever et al (154, 155) and Guevin et al (140) found amount of total dietary fiber was significantly related to GI and uronic acids, insoluble fiber were most closely related to GI but soluble fiber was not related.

According to our study, glucose content did not seem to be the only factor responsible for the peak of plasma glucose increment and GI, other sugars and fiber content in fruits were also responsible. The presence of total dietary fibers in fruits may contribute to the lowering of the glycemic response. However, in our study there was no clear correlation between fiber content and GI. In addition, the presence or absence of starch, as unrefined carbohydrates in the fruit is also responsible for the outcome of glycemic response and GI.

Lipid Levels Responding to Different Amounts of Fruits

Recently, there has been increasing interest toward the liberalization of sucrose in the diets of individuals with diabetes mellitus. However, there is evidence from several well-controlled prospective studies demonstrating that the consumption of varying amounts of sucrose may result in hyperinsulinemia, hypertriglyceridemia, hypercholesterolemia and reduced HDL-C concentratons. For examples, Sleder J. (127) showed fructose-fed rats (66 % of fructose) developed significant hypertriglyceridemia. Reiser S. (156) found that fructose intake of hyperinsulinemic men increased total triglycerides and their lipoprotein distribution, total and VLDL-C, apoprotein B-100, C-II, C-III. Moreover Hallfrisch et al. (36) exhibited total plasma cholesterol and LDL-C were higher after the hyperinsulinemic men consumed 7.5 and 15 % fructose than when they consumed the 0 % fructose diet importantly, plasma triglyceride significantly increased in men and women subjects.

Our study found that consumption of all fruits and glucose drink elevated the plasma triglyceride concentration in particular tangerine, rambutan and pomelo. Tangerine, rambutan and pomelo has low amount of fructose but it contains high sucrose which is rapidly hydrolyzed (109, 157) and is also used for triglyceride synthesis. This effect can be explained by the metabolism of glucose and fructose as shown in Figure 1. Conversion of fructose to fructose-1-phosphate and to acetyl CoA is more rapidly than conversion of glucose to fructose-6-phosphate and to acetyl CoA because liver fructokinase activity is greater than the glucokinase. Consequently, fructose-1-phosphate is broken down to glycerol-3-phosphate and is being used to synthesis triglycerides.

In general, postprandial serum triglycerides rise about 3-4 hours after ingestion of food and then decline at about 6-8 hours (158).

Most data also support the effect of dose response of fruits on triglyceride concentration (142, 151). We found that the percentage changes of serum triglycerides of 50 g carbohydrate doses were greater than 30 g CHO doses. Our data also indicated that the amount of carbohydrate in these fruits (Table 3.4) has minimal effect on serum cholesterol (Table 4.7, Figure 4.11-4.13) and LDL-C concentration (Table 4.9, Figure 4.17-4.19). It is because fruit contains no cholesterol; therefore, it had to gain from de novo synthesis through acetyl coenzyme A.

Hollenbeck et al (159) demonstrated that long-term consumption of high sucrose in DM type 2 patients elevated plasma triglycerides and depressed HDL-C. Table 4.7 Figure 4.11-4.13 showed significant reduction from baseline of HDL-C in both doses of pomelo and 50 g CHO of rambutan as well. It might be that the increment of triglycerides accelerated the reverse cholesterol pathway (158). However, the degree of reduction between the 2 doses was not significantly different. In addition, HDL-C is influenced by many physiological variables such as sex hormone, diet and physical activity. These data need further investigation.

Blood Hemorrheologic Changes

The hemorrheology properties of blood are important for determination of blood flow. In diabetic patients the rheological abnormalities occur before the appearance of any severe degenerative complications (69, 73). Plasma and blood viscosity are consistency associated with both the prevalence and incidence of cardiovascular disease (73, 160).The factors affecting the blood viscosity are red cell concentration, blood components, the size of vessels and the velocity of blood flow (68, 161). Diabetic microangiopathy is associated with hyperviscosity (74). In addition, patients with hypertriglyceridemia and mixed hyperlipidemia have been found to have mean plasma viscosity higher than normal subjects (162, 163). Normal blood viscosity (68) is in the range 3-4 times more viscous than water. Our study disclosed that whole blood viscosity during fasting were within normal range. Results of Somnuk et al (142) and Visavajarn et al (164) as well as our data revealed that at the peak of hyperglycemia (60 minutes postprandial) the whole blood viscosity tended to be decreased in the entire test (Table 4.10-4.11 Figure 4.16-4.18). Moreover, percentage change of viscosity of 30 g CHO of banana was significantly less than 30 g CHO of tangerine at 60 minutes. This should be due to the degree of difference in the glucose responses and the percentage change of glucose at 60 minutes and AUC of banana and tangerine.

In addition, mean percentage change of blood viscosity at 60, 300 minutes after ingestion of 30 g and 50 g CHO of fruits were not significantly different. It is unlikely that consumption of 2-3 servings of fruit could affect whole blood viscosity.

Koeing et al (79), Simone et al (73) and Carallo et al (165) reported that triglyceride and cholesterol levels were positively related to whole blood viscosity but we did not see significant increments of whole blood viscosity in our subjects after consumption of different amount of certain fruits. The hyperviscosity effect was not demonstrable in our patients even though their triglycerides were increased. Though there were some fluctuations of whole blood viscosity, the differences were about 6 % only and were not statistically significant.

Application of Glycemic Index and Glycemic Load from this Study

A GI values show only how rapidly a particular carbohydrate turns into blood glucose. It doesn't show how much of that carbohydrate is in a serving of a particular food. Therefore, glycemic load comes in for explaining a food's effect on blood sugar. The glycemic load (GL) (166, 167) is a relatively new way to assess the impact of carbohydrate consumption that takes the glycemic index into account, but gives a fuller picture than does glycemic index alone. In watermelon, for example, glycemic index is pretty high, about 72. According to the calculations by the people at the University of Sydney's Human Nutrition Unit, in a serving of 120 grams it has 6 grams of available carbohydrate per serving, so its glycemic load is pretty low (about 4).

A GL of 20 or more is high, a GL of 11 to 19 inclusive is medium, and a GL of 10 or less is low (168). Foods that have a low GL almost always have a low GI. Foods with an intermediate or high GL range from very low to very high GI.

Calculating overall dietary GL is difficult outside a research setting. But knowing a food's GL can help you make comparisons that can improve the quality of your carbohydrate choices.

CHAPTER VI
CONCLUSION

From the viewpoints of glycemic response, serum lipid response and blood viscosity response it can be mentioned that type 2 DM patients may be able to consume high amount of rambutan, banana or papaya, (about 3 servings at a time) because of low GI and low peak of glycemic responses. Even, increased banana ingestion (about 3 servings) could bring about increased serum triglyceride and decreased HDL-C level while there was no significant change in serum cholesterol; and increased papaya ingestion (about 3 serving) could bring about increased blood glucose response and decreased LDL-C level. However, AUC of 50 g CHO of papaya and banana lower than AUC of 30 g CHO of pomelo, rambutan and tangerine. Moreover, glucose responses of them at 30 and 60 minutes slowly increased from baseline.

The glycemic index of banana, tangerine, papaya, rambutan, rose apple and pomelo were 26, 30, 38, 47, 50 and 59, respectively.

The hyperlipidemic type 2 DM may ingest three servings of rose apple. Although, glycemic index of rose apple rank second highest in the group but glucose responses were low and peak of glycemic response was not high. Besides, increased amount of consumption did not affect the glycemic response, serum lipid levels and blood viscosity. Hence, it is highly recommended for the diabetics especially the obese and poorly control.

Although, GI of tangerine and rambutan were ranked as low GI foods and GI of pomelo was not very high they produced very high peaks of postprandial glycemic response. Therefore, tangerine, rambutan and pomelo were not suitable for glycemic control but they may be the appropriate choices for correction of hypoglycemic episode.

Therefore, for diabetic patients, a cautious approach is necessary regarding the consumption quantity of tangerine, rambutan and pomelo. They should not consume more than 2 servings of fruit in a meal.

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APPENDIX

แบบสอบถามเรื่องทัศนคติที่มีต่อผลไม้ไทยในผู้ที่เป็นโรคเบาหวานชนิดที่ 2

(โปรดระบุชนิด และพันธุ์ของผลไม้)

1. ผลไม้ที่ท่านชอบรับประทาน

1...

2...

3...

2. ผลไม้ที่ท่าน**อยาก**รับประทาน แต่ถูกห้าม

1...

2...

3...

3. ผลไม้ที่ท่านคิดว่าเมื่อรับประทานแล้วจะทำให้น้ำตาลในเลือด**ขึ้นสูง**

1...

2...

3...

4. ผลไม้ที่ท่านคิดว่าเมื่อรับประทานแล้วจะทำให้ระดับไขมันในเลือด**ขึ้นสูง**

1...

2...

3...

5. ผลไม้ที่ท่านคิดว่าเมื่อรับประทานแล้วระดับน้ำตาลในเลือดขึ้น**ไม่สูง**

1...

2...

3...

Table 3 Plasma glucose in 16 subjects after ingestion different amounts of fruits

	50 g glucose (group 1)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
1	99	196	236	178	74	65	76
2	148	296	377	296	216	162	135
3	142	285	387	306	234	157	117
4	137	193	263	299	204	141	114
5	159	274	333	274	220	150	124
6	126	249	350	303	222	178	141
7	187	279	384	303	254	200	178
8	150	240	288	241	177	117	92

	50 g glucose (group 2)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
9	137	274	346	209	130	94	97
10	141	292	279	214	162	132	108
11	135	160	211	231	137	99	85
12	153	205	256	265	238	213	175
13	279	317	404	413	337	296	256
14	85	159	216	173	114	59	65
15	274	362	405	409	344	296	261
16	115	162	209	171	88	81	92

Table 3 Plasma glucose in 16 subjects after ingestion different amounts of fruits (continued)

	50 g CHO of tangerine (group 1)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
1	142	173	209	135	90	72	85
2	157	198	267	196	148	119	117
3	115	196	205	153	108	85	79
4	151	236	211	159	124	112	114
5	178	234	216	119	103	105	121
6	99	191	202	151	112	88	72
7	178	268	297	232	180	135	115
8	135	211	211	121	103	101	106

	30 g CHO of tangerine (group 1)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
1	142	234	249	175	105	76	85
2	198	259	301	265	211	177	159
3	103	162	198	151	126	101	88
4	142	252	249	177	132	106	99
5	137	218	250	160	106	97	103
6	101	207	214	169	133	99	99
7	146	218	232	173	132	117	114
8	146	238	222	169	132	117	101

Table 3 Plasma glucose in 16 subjects after ingestion different amounts of fruits (continued)

	50 g CHO of rambutan (group 1)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
1	97	141	184	105	68	68	72
2	160	231	281	175	130	115	117
3	153	225	267	218	198	173	144
4	119	184	227	198	155	126	114
5	186	268	348	213	162	142	144
6	162	290	296	231	177	151	139
7	184	299	308	252	207	186	155
8	128	214	204	126	97	92	101

	30 g CHO of rambutan (group 1)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
1	92	162	155	94	70	70	81
2	155	216	211	159	126	108	115
3	137	191	209	159	132	106	99
4	133	207	195	153	126	108	108
5	133	229	193	139	92	94	99
6	105	202	202	160	126	119	114
7	187	258	250	211	169	135	128
8	142	236	214	141	112	106	115

Table 3 Plasma glucose in 16 subjects after ingestion different amounts of fruits (continued)

	50 g CHO of pomelo (group 1)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
1	177	279	308	258	191	132	105
2	160	227	283	265	214	166	141
3	141	234	263	220	189	151	124
4	164	254	276	238	173	133	110
5	151	205	254	137	112	106	101
6	106	207	209	177	133	101	88
7	202	314	321	270	249	214	182
8	157	200	247	205	175	155	156

	30 g CHO of pomelo (group 1)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
1	108	139	166	117	85	74	77
2	151	258	250	191	170	152	145
3	121	160	182	146	123	108	92
5	135	211	223	180	130	103	96
6	141	191	258	133	85	86	94
7	108	189	193	169	123	90	85
8	216	288	305	265	231	211	193
4	133	229	213	142	110	99	99

Table 3 Plasma glucose in 16 subjects after ingestion different amounts of fruits (continued)

	50 g CHO of rose apple (group 2)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
9	155	220	193	142	130	112	119
10	211	241	348	268	193	150	123
11	126	171	166	132	103	94	92
12	169	204	268	250	218	202	173
13	305	317	351	373	297	272	238
14	90	187	205	164	99	79	58
15	409	526	510	490	443	407	364
16	103	137	132	97	96	92	90

	30 g CHO of rose apple (group 2)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
9	151	211	193	150	115	112	105
10	178	214	256	213	177	141	119
11	135	186	195	162	130	110	103
12	177	207	263	222	189	162	141
13	300	315	343	322	310	301	299
14	85	141	159	94	61	63	72
15	353	391	440	371	341	315	294
16	96	121	97	90	92	88	96

Table 3 Plasma glucose in 16 subjects after ingestion different amounts of fruits (continued)

	50 g CHO of banana-Kai variety (group 2)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
9	133	186	180	126	112	110	114
10	168	209	232	191	148	117	101
11	106	117	132	119	99	96	94
12	157	155	184	196	169	142	137
13	238	267	287	268	250	196	204
14	88	144	151	92	67	67	74
15	330	335	389	332	312	279	272
16	110	150	157	124	101	92	97

	30 g CHO of banana-Kai variety (group 2)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
9	159	204	198	150	126	117	119
10	177	177	211	173	133	121	114
11	133	144	166	141	115	103	99
12	160	166	193	173	157	139	133
13	314	341	348	337	306	287	281
14	88	123	124	94	74	67	77
15	341	350	380	344	314	297	278
16	105	132	132	110	101	94	97

Table 3 Plasma glucose in 16 subjects after ingestion different amounts of fruits (continued)

	50 g CHO of papaya (group 2)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
9	153	209	189	135	117	114	115
10	164	187	243	204	162	121	92
11	132	166	187	160	132	110	105
12	168	189	232	218	186	162	153
13	198	278	279	245	220	189	177
14	88	112	146	106	79	65	68
15	337	436	451	384	359	335	306
16	106	141	130	103	96	92	92

	30 g CHO of papaya (group 2)						
Subject	0 min	30 min	60 min	120 min	180 min	240 min	300 min
9	150	177	164	133	130	126	128
10	182	207	211	180	142	123	99
11	137	159	164	135	108	97	99
12	166	164	211	218	200	187	171
13	301	350	344	317	303	252	258
14	77	90	97	77	68	67	70
15	359	389	396	377	332	301	306
16	99	128	115	96	88	83	86

Table 4 Serum triglyceride in 16 subjects after ingestion different amounts of fruits

Subject	50 g CHO of tangerine		50 g CHO of rambutan		50 g CHO of pomelo	
	Before	After	Before	After	Before	After
1	183	219	338	323	184	219
2	117	143	201	262	126	138
3	161	222	128	186	97	128
4	142	188	104	120	107	128
5	126	204	112	176	81	120
6	240	265	174	227	131	196
7	129	165	140	182	139	170
8	124	153	139	166	105	125

Subject	30 g CHO of tangerine		30 g CHO of rambutan		30 g CHO pomelo	
	Before	After	Before	After	Before	After
1	213	258	208	244	266	275
2	167	204	141	173	213	225
3	134	197	127	188	130	157
4	62	120	152	145	104	122
5	97	113	109	173	111	142
6	171	192	128	133	142	188
7	113	149	121	142	177	182
8	93	128	303	304	124	132

Table 4 Serum triglyceride in 16 subjects after ingestion different amounts of fruits (continued)

Subject	50 g CHO of rose apple		50 g CHO of banana-Kai variety		50 g CHO of papaya	
	Before	After	Before	After	Before	After
9	186	192	214	273	212	279
10	156	230	84	139	138	142
11	192	202	150	146	135	140
12	139	128	137	155	100	116
13	137	248	121	160	63	117
14	165	127	81	123	192	171
15	239	194	156	199	165	206
16	41	63	37	51	46	71

Subject	30 g CHO of rose apple		30 g CHO of banana-Kai variety		30 g CHO of papaya	
	Before	After	Before	After	Before	After
9	126	191	161	196	212	222
10	103	120	94	150	109	99
11	142	135	95	96	142	145
12	146	142	132	151	127	93
13	152	120	152	120	135	162
14	204	196	225	212	101	151
15	158	172	176	183	204	236
16	46	71	37	33	43	56

Table 4 Serum triglyceride in 16 subjects after ingestion different amounts of fruits (continued)

Subject	50 g glucose (gr. 1)		Subject	50 g glucose (gr.2)	
	Before	After		Before	After
1	206	228	9	223	280
2	139	151	10	121	161
3	128	138	11	169	175
4	98	113	12	151	129
5	132	191	13	135	164
6	123	158	14	149	151
7	127	150	15	114	171
8	197	222	16	42	55

Table 5 Serum cholesterol in 16 subjects after ingestion different amounts of fruits

Subject	50 g CHO of tangerine		50 g CHO of rambutan		50 g CHO of pomelo	
	Before	After	Before	After	Before	After
1	202	198	226	216	172	176
2	181	171	293	294	168	154
3	161	174	234	225	155	160
4	141	151	203	194	145	141
5	229	247	214	214	228	231
6	225	243	281	276	202	206
7	194	194	195	202	175	169
8	205	215	270	262	202	210

Subject	30 g CHO of tangerine		30 g CHO of rambutan		30 g CHO pomelo	
	Before	After	Before	After	Before	After
1	199	188	243	266	263	253
2	206	217	295	281	213	227
3	180	180	211	219	169	171
4	152	122	213	217	158	161
5	237	233	256	252	232	248
6	233	251	235	224	212	219
7	175	174	190	171	208	206
8	227	210	265	271	184	184

Table 5 Serum cholesterol in 16 subjects after ingestion different amounts of fruits (continued)

Subject	50 g CHO of rose apple		50 g CHO of banana-Kai variety		50 g CHO of papaya	
	Before	After	Before	After	Before	After
9	199	190	204	216	207	190
10	238	241	191	194	183	176
11	186	193	159	165	152	149
12	172	175	183	190	195	185
13	221	220	196	214	163	162
14	211	205	162	156	194	195
15	242	246	232	227	218	219
16	163	163	149	152	161	161

Subject	30 g CHO of rose apple		30 g CHO of banana-Kai variety		30 g CHO of papaya	
	Before	After	Before	After	Before	After
9	205	201	212	218	216	224
10	191	191	198	202	177	183
11	154	145	156	158	142	142
12	172	154	203	199	170	173
13	258	279	258	279	224	237
14	170	171	167	156	197	205
15	179	190	202	203	238	251
16	152	156	146	141	163	142

Table 5 Serum cholesterol in 16 subjects after ingestion different amounts of fruits (continued)

Subject	50 g glucose (gr. 1)		Subject	50 g glucose (gr.2)	
	Before	After		Before	After
1	199	195	9	215	231
2	188	186	10	225	234
3	176	174	11	185	194
4	162	159	12	167	173
5	254	256	13	183	182
6	217	216	14	184	180
7	183	154	15	187	194
8	243	259	16	162	151

Table 6 Serum HDL-C in 16 subjects after ingestion different amounts of fruits

Subject	50 g CHO of tangerine		50 g CHO of rambutan		50 g CHO of pomelo	
	Before	After	Before	After	Before	After
1	39	39	14	12	39	39
2	52	46	55	53	37	36
3	58	63	61	60	56	56
4	44	45	53	50	46	42
5	53	54	57	57	54	51
6	41	42	51	49	39	37
7	44	41	44	43	45	40
8	44	47	50	49	47	45

Subject	30 g CHO of tangerine		30 g CHO of rambutan		30 g CHO pomelo	
	Before	After	Before	After	Before	After
1	41	43	41	45	35	34
2	44	47	64	61	48	47
3	56	53	65	65	57	54
4	56	51	58	58	48	46
5	54	51	64	65	57	54
6	41	39	51	50	39	38
7	43	43	41	39	43	44
8	42	44	50	49	44	39

Table 6 Serum HDL-C in 16 subjects after ingestion different amounts of fruits (continued)

Subject	50 g CHO of rose apple		50 g CHO of banana-Kai variety		50 g CHO of papaya	
	Before	After	Before	After	Before	After
9	36	33	36	36	32	34
10	52	49	53	54	41	39
11	29	29	31	32	27	27
12	42	43	56	49	38	34
13	49	53	44	44	51	50
14	39	36	34	27	26	26
15	51	51	56	53	51	49
16	46	45	49	44	50	51

Subject	30 g CHO of rose apple		30 g CHO of banana-Kai variety		30 g CHO of papaya	
	Before	After	Before	After	Before	After
9	34	39	37	36	32	34
10	44	43	50	48	43	43
11	31	33	31	31	29	27
12	40	39	39	38	36	38
13	44	51	44	51	39	41
14	37	33	26	23	35	33
15	44	44	45	46	41	43
16	46	46	47	48	53	52

Table 6 Serum HDL-C in 16 subjects after ingestion different amounts of fruits (continued)

Subject	50 g glucose (gr. 1)		Subject	50 g glucose (gr.2)	
	Before	After		Before	After
1	37	36	9	44	43
2	51	43	10	45	46
3	53	51	11	30	27
4	47	44	12	38	39
5	56	53	13	41	48
6	34	34	14	36	39
7	40	35	15	51	50
8	46	42	16	56	51

Table 7 Serum LDL-C in 16 subjects after ingestion different amounts of fruits

Subject	50 g CHO of tangerine		50 g CHO of rambutan		50 g CHO of pomelo	
	Before	After	Before	After	Before	After
1	126	116	145	139	96	93
2	106	96	199	188	106	90
3	71	68	147	128	80	79
4	68	69	129	120	78	74
5	151	153	135	122	158	156
6	137	148	196	182	137	130
7	124	121	123	123	102	96
8	136	138	193	180	150	147

Subject	30 g CHO of tangerine		30 g CHO of rambutan		30 g CHO pomelo	
	Before	After	Before	After	Before	After
1	116	94	161	173	175	164
2	129	129	203	186	123	115
3	97	88	121	117	86	86
4	84	47	125	131	89	91
5	164	160	171	153	153	166
6	158	173	158	148	145	143
7	110	102	125	104	130	126
8	167	140	155	163	115	119

Table 7 Serum LDL-C in 16 subjects after ingestion different amounts of fruits (continued)

Subject	50 g CHO of rose apple		50 g CHO of banana-Kai variety		50 g CHO of papaya	
	Before	After	Before	After	Before	After
9	127	119	125	126	133	102
10	155	146	122	112	115	109
11	119	125	98	104	98	94
12	103	107	100	110	137	128
13	144	118	128	139	99	89
14	139	144	112	105	130	134
15	143	157	146	134	134	129
16	109	105	93	97	101	96

Subject	30 g CHO of rose apple		30 g CHO of banana-Kai variety		30 g CHO of papaya	
	Before	After	Before	After	Before	After
9	146	124	143	143	142	146
10	126	124	129	124	112	120
11	95	85	106	108	85	85
12	103	86	137	131	109	117
13	183	204	183	204	157	165
14	92	100	96	91	142	142
15	104	112	122	121	157	161
16	97	96	92	86	101	79

Table 7 Serum LDL-C in 16 subjects after ingestion different amounts of fruits (continued)

Subject	50 g glucose (gr. 1)		Subject	50 g glucose (gr.2)	
	Before	After		Before	After
1	120	114	9	127	132
2	110	113	10	150	156
3	98	96	11	122	132
4	96	93	12	98	108
5	172	165	13	116	101
6	159	151	14	118	111
7	119	90	15	113	110
8	158	174	16	98	89

Table 8 Whole blood viscosity in 16 subjects after ingestion different amounts of fruits

Subject	50 g CHO of tangerine			30 g CHO of tangerine		
	0 min	60 min	300 min	0 min	60 min	300 min
1	3.27	2.97	3.24	3.15	2.94	3.10
2	3.36	3.33	3.51	3.25	3.05	3.22
3	3.47	3.34	3.48	3.20	3.00	3.18
4	3.34	2.97	3.30	3.25	3.04	3.32
5	3.43	3.16	3.44	3.2	3.00	3.18
6	3.21	2.96	3.19	3.64	3.44	3.97
7	3.57	3.43	3.57	3.13	2.91	3.36
8	3.35	3.22	3.33	3.66	3.48	3.60

subject	50 g CHO of rambutan			30 g CHO of rambutan		
	0 min	60 min	300 min	0 min	60 min	300 min
1	2.98	2.70	3.00	2.86	2.77	3.12
2	3.61	3.57	4.92	3.51	3.21	3.55
3	3.76	3.61	3.33	3.63	3.43	3.78
4	3.48	3.22	3.46	3.60	3.33	3.30
5	3.66	3.34	3.91	3.33	3.12	3.43
6	3.66	3.33	3.72	3.84	3.49	3.73
7	3.43	3.06	3.63	3.46	3.34	3.57
8	3.79	3.61	3.33	3.75	3.67	3.70

Table 8 Whole blood viscosity in 16 subjects after ingestion different amounts of fruits (continued)

Subject	50 g CHO of pomelo			30 g CHO pomelo		
	0 min	60 min	300 min	0 min	60 min	300 min
1	3.24	3.03	3.09	3.25	3.13	3.06
2	3.58	3.12	3.55	3.85	3.43	3.88
3	3.82	3.54	3.91	3.76	3.48	4.14
4	3.46	3.12	3.33	3.01	3.06	3.13
5	3.34	3.01	3.21	3.40	3.22	3.46
6	3.46	3.31	3.55	3.46	3.21	3.37
7	3.48	3.18	3.43	3.67	3.42	3.69
8	3.74	3.46	3.71	3.60	3.48	3.52

Subject	50 g CHO of rose apple			30 g CHO of rose apple		
	0 min	60 min	300 min	0 min	60 min	300 min
9	4.32	3.90	3.96	3.97	3.91	4.12
10	3.82	3.63	3.85	3.91	3.94	3.76
11	3.70	3.67	3.66	3.78	3.75	3.81
12	2.98	2.54	3.10	3.18	2.92	2.98
13	4.30	3.90	4.35	3.82	3.54	3.91
14	3.52	3.34	3.61	3.87	3.43	3.21
15	3.84	3.81	3.97	4.00	3.24	3.88
16	3.09	3.25	3.10	2.91	2.91	2.94

Table 8 Whole blood viscosity in 16 subjects after ingestion different amounts of fruits (continued)

Subject	50 g CHO of banana-Kai variety			30 g CHO of banana-Kai variety		
	0 min	60 min	300 min	0 min	60 min	300 min
9	4.03	3.69	3.75	4.27	4.26	4.53
10	3.85	3.67	4.15	3.84	3.93	3.96
11	3.45	3.4	3.58	3.3	3.28	3.36
12	3.13	3.07	3.13	3.36	3.24	3.18
13	3.85	3.66	4.5	3.82	4.03	4.42
14	3.43	3.18	3.37	3.7	3.48	3.45
15	3.78	3.7	3.61	3.52	3.43	3.55
16	2.83	2.76	2.88	2.67	2.62	2.68

Subject	50 g CHO of papaya			30 g CHO of papaya		
	0 min	60 min	300 min	0 min	60 min	300 min
9	4.24	4.12	4.57	4.24	4.12	4.57
10	3.6	3.36	3.48	3.6	3.36	3.48
11	3.24	2.97	3.27	3.24	2.97	3.27
12	3	2.89	3.13	3	2.89	3.13
13	3.49	3.34	4.09	3.49	3.34	4.09
14	3.48	3.4	3.6	3.48	3.4	3.6
15	3.61	3.18	3.6	3.61	3.18	3.6
16	2.97	2.76	3.22	2.97	2.76	3.22

Table 8 Whole blood viscosity in 16 subjects after ingestion different amounts of fruits (continued)

Subject	50 g glucose (gr. 1)			Subject	50 g glucose (gr.2)		
	0 min	60 min	300 min		0 min	60 min	300 min
1	3.39	3.36	3.39	9	3.52	3.3	3.84
2	4.59	3.78	4.14	10	3.73	3.5	3.69
3	3.82	3.57	3.82	11	4.12	3.67	3.96
4	3.28	3.19	3.27	12	3.09	3.01	3.1
5	3.78	3.56	3.79	13	3.58	3.78	4.23
6	3.31	3	3.36	14	3.75	3.63	3.73
7	3.24	3.42	3.81	15	3.46	3.48	3.57
8	3.78	3.55	3.78	16	3.36	3.07	3.06

BIOGRAPHY

NAME	Miss Pasinee Chartchuathaicharoen
DATE OF BIRTH	March 21, 1980
PLACE OF BIRTH	Bangkok, Thailand
INSTITUTION ATTENDED	Faculty of Allied Health Science
	Bachelor of Science (Medical Technology)
	Chulalongkorn University, 1997-2001
	Master of Science (Nutrition)
	Mahidol University, 2002-2006
HOME ADDRESS	120/732 Taybanmai, Muang district,
	Samutprakarn 10280