EFFECTS OF DIFFERENT CONCENTRATIONS OF OAT ON POSTPRANDIAL PLASMA GLUCOSE AND TRIGLYCERIDES LEVEL IN HEALTHY INDIVIDUALS

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A Thesis Submitted in Partial Fulfillment of the Requirements
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การศึกษาผลของโอ๊ตที่ความเข้มข้นต่างกันต่อระดับน้ำตาลและไตรกลีเซอไรด์หลังอาหาร ในอาสาสมัครที่มีสุขภาพดี

นางสาวเม็ง จี ลี

วิทยานิพนธ์นี้เป็นส่วนหนึ่งของการศึกษาตามหลักสูตรปริญญาวิทยาศาสตรมหาบัณฑิต

สาขาวิชาอาหารและโภชนาการ ภาควิชาโภชนาการและการกำหนดอาหาร

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Thesis Title	EFFECTS OF DIFFERENT CONCENTRATIONS OF OAT ON POSTPRANDIAL PLASMA GLUCOSE AND TRIGLYCERIDES LEVEL IN HEALTHY INDIVIDUALS
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เม็ง จี ลี:การศึกษาผลของโอ๊ตที่ความเข้มข้นต่างกันต่อระดับน้ำตาลและไตรกลีเซอไรด์หลังอาหารในอาสาสมัครที่มีสุขภาพดี (EFFECTS OF DIFFERENT CONCENTRATIONS OF OAT ON POSTPANDIAL PLASMA GLUCOSE AND TRIGLYCERIDES LEVEL IN HEALTHY INDIVIDUALS) อ.ที่ปรึกษาวิทยานิพนธ์หลัก: ผศ.ดร.สุวิมล ทรัพย์วโรบล, อ.ที่ปรึกษาวิทยานิพนธ์ร่วม: อ.ดร.ณัฐธิดา โชติช่วง, 53 หน้า

การศึกษาจำนวนมากรายงานเกี่ยวกับผลในต้านของคลอเลสเตอรอลสูงอย่างมีประสิทธิภาพของเส้นใยอาหารที่ละลายน้ำได้ (เบต้า-กลูแคน) พบได้ในโอ๊ต (ชื่อวิทยาศาสตร์: *Avena sativa*)แต่อย่างไรก็ตามผลของความเข้มข้มที่แตกต่างกันต่อระดับน้ำตาลและไตรกลีเซอไรด์ในเลือดยังไม่มีการศึกษาที่แน่ชัด วัตถุประสงค์ของงานวิจัยในครั้งนี้เพื่อเปรียบเทียบผลของโอ๊ตที่ความเข้มข้นต่างกันต่อระดับน้ำตาลกลูโคสหลังรับประทานอาหารและระดับไตรกลีเซอไรด์ในเลือด การศึกษานี้เป็นแบบrandomized controlled และ crossoverโดยมีอาสาสมัครเข้าร่วมวิจัย จำนวน 11 คน อายุระว่าง 18-25 ปี ซึ่งจะถูกแบ่งกลุ่มแบบสุ่มให้ได้รับอาหารทดสอบจำนวน 3กลุ่ม (กลุ่มที่ได้รับโอ๊ตร่วมกับน้ำ 169, 210 และ 335 มิลลิลิตร ตามลำดับ) การทดสอบแต่ละครั้งจะสลับกันไปและห่างกันอย่างน้อย 1 สัปดาห์ อาสาสมัครได้รับการเหนี่ยวนำให้เกิดน้ำตาลในเลือดสูงและไตรกลีเซอไรด์โดยอาหารคาร์โบไฮเดรตสูงและอาหารที่มีไขมันสูง (HCHF) ซึ่งประกอบด้วยคาร์โบไฮเดรต 101.7กรัม (49% ของพลังงานทั้งหมด) และไขมัน 41.9กรัม (46% ของพลังงานทั้งหมด) เก็บตัวอย่างเลือดของอาสาสมัครเพื่อไปวิเคราะห์ระดับน้ำตาลกลูโคสในเลือดและการวิเคราะห์ซีรั่มไตรกลีเซอไรด์ ที่เวลา 0 (พื้นฐาน), 1, 3 และ 5 ชั่วโมง ผลของอาหารที่ทดสอบที่มีต่อระดับน้ำตาลกลูโคสหลังอาหารและไตรกลีเซอไรด์ การเปลี่ยนแปลงระดับน้ำตาลกลูโคสหลังอาหารและไตรกลีเซอไรด์ พื้นที่ใต้กราฟ (AUC), การเปลี่ยนแปลงของพื้นที่ใต้กราฟ (iAUC) จะถูกวิเคราะห์โดยใช้การวิเคราะห์ทางสถิติแบบ one way repeated ANOVAข้อมูลที่ได้จะนำมาเปรียบเทียบ Multiple comparison โดยใช้ Bonferroni correction ค่าแตกต่างอย่างมีนัยสำคัญที่ $P<0.05$.พื้นที่ใต้กราฟ (AUC) ของระดับพลาสมากลูโคสหลังจากที่รับประทานโอ๊ตร่วมกับน้ำ 169, 210 และ 335 มิลลิลิตร ไม่แตกต่างกันทางสถิติ (500.09, 535.09 และ 535.73 *มิลลิกรัม*/เดซิลิตรตามลำดับ) เมื่อเทียบแต่ละกลุ่ม นอกจากนี้ยังไม่พบความแตกต่างทางสถิติของความเข้มข้นที่ต่างกันของโอ๊ตต่อระดับไตรกลีเซอไรด์ (343.82, 304.55 และ 331.73 *มิลลิกรัม*/เดซิลิตรตามลำดับ) คำอธิบายที่เป็นไปได้สำหรับไม่มีผลของโอ๊ตที่ความเข้มข้นต่างๆ กันต่อระดับน้ำตาลกลูโคสหลังรับประทานอาหารและระดับไตรกลีเซอไรด์ในเลือด อาจจะเนื่องมาจากปริมาณเส้นใยอาหารปริมาณน้อย โดยเฉพาะอย่างยิ่งเส้นใยอาหารที่ละลายน้ำ (เบต้า-กลูแคน) ในการทดลอง นอกจากนี้ในการศึกษาต่อของผลกระทบในระยะยาวเป็นสิ่งจำเป็นที่จะต้องมีผลการทดลองที่สนับสนุนมากขึ้น

ภาควิชา.....โภชนาการและการกำหนดอาหาร..... ลายมือชื่อนิสิต.....

สาขาวิชา.....อาหารและโภชนาการ..... ลายมือชื่อ อ.ที่ปรึกษาวิทยานิพนธ์หลัก.....

ปีการศึกษา.....2556..... ลายมือชื่อ อ.ที่ปรึกษาวิทยานิพนธ์ร่ว.....

##5476853337: MAJOR FOOD AND NUTRITION
KEYWORDS: OAT/ POSTPRANDIAL HYPERGLYCEMIA/ HYPERLIPIDEMIA

MENG JIE LI: EFFECTS OF DIFFERENT CONCENTRATIONS OF OAT ON POSTPRANDIAL PLASMA GLUCOSE AND TRIGLYCERIDES LEVEL IN HEALTHY INDIVIDUALS. ADVISOR: ASST. PROF. SUWIMOL SAPWAROBOL, Dr.P.H., CO-ADVISOR: NATTIDA CHOTECHUANG, Ph.D., 53 pp.

Numerous studies reported potent anti-hypercholesterolemic effect of soluble fiber (β-glucan) found in oat (*Avena sativa*). However, the effect of different concentrations of oat on blood glucose and triglycerides is not yet well established. The objective of present study was to evaluate one serving oat flakes (35 g) in the various concentrations on postprandial plasma glucose and serum triglycerides level. In a randomized, controlled, crossover study, 11 participants aged 18-25 years were randomly allocated into 3 test meals (1 serving oat in 169, 210 and 335 ml water). Each test was cross-over on a separate day and at least one week apart. Participants were induced hyperglycemia and hypertriglyceridemia by a high carbohydrate and high fat meal (HCHF) which composed of 101.7g carbohydrate (49% of total energy) and 41.9g fat (46 % of total energy). Blood samples were collected for plasma glucose and serum triglycerides analysis at 0 (baseline), 1, 3 and 5 hours. Effect of the test meals on postprandial glucose and triglycerides, incremental glucose and triglycerides level, AUC, iAUC of glucose and triglycerides were assessed by one way repeated ANOVA. Statistical significance, with Bonferroni correction for multiple comparisons, was defined as $p < 0.05$. AUC plasma glucose after oat 1 serving with 169, 210 and 335 ml water were not significantly different (500.09, 535.09 and 535.73 mg/ dL, respectively) compared to each group. There was also no significant effect of different oat on serum triglycerides (343.82, 304.55 and 331.73 mg/ dL, respectively). Possible explanation for the insignificant effect of oatmeal in various concentrations on postprandial blood glucose and triglycerides may be due to low dose of dietary fiber in the intervention meals. In addition, more frequent time intervals and longer experimental period for the blood sample collection are needed to warrant the results.

Department: ... Nutrition and Dietetics.... Student's Signature.....
Field of Study: .....Food and Nutrition..... Advisor's Signature.....
Academic Year: .....2013..... Co-advisor's Signature.....

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CHAPTER I

INTRODUCTION

Metabolic syndrome is a common disorder characterized by a series of complex disorder, it directly contribute to the risk of coronary heart disease, cardiovascular atherosclerotic disease as well as type 2 diabetes mellitus. According to the World Health Organization criteria (Alberti and Zimmet, 1998), metabolic syndrome is characterized by insulin resistance and any two of the following criteria: high triglycerides or/ and low High-density lipoprotein-cholesterol (HDL-c), dysregulated glucose homeostasis, abdominal obesity, high blood pressure, and microalbuminuria. Diabetes Mellitus(DM) is generally to be described as the most common endocrine metabolic disorders while all forms of diabetes mellitus share the hyperglycemia with disturbances of carbohydrate, fat and protein metabolism results from defection of insulin production or/and insulin action. Long term hyperglycemia from whatever type 1 or type 2 DM can lead to microvascular and macrovascular complications involve in retinopathy, nephropathy, neuropathy as well as the cardiovascular disease. (American Diabetes Association [A.D.A.], 2010) The Third National Health Examination Survey 2004 reported the prevalence of Diabetes and Impaired Fasting Glucose in Thailand weighted to the national 2004 population is 6.7% and 12.5%, respectively (Wichai Aekplakorn et al., 2007). The mortality of cardiovascular disease and diabetes in non-communicable diseases reported by World Health Organization in 2008 has grown to 280 per 100,000 female and 343 per 100,000 male in Thai populations (World Health Organization [W.H.O.], 2011: Online).

Postprandial state is used to describe the period that comprises and follows a meal. Duration of the postprandial state depends on the nature of meals. After consumption a meal consists mainly of carbohydrates, 2–3 hours need to return to the basal state while a mixed meal takes 3–5 hours; And after a fat-rich meal the return to the basal state may take as long as 8–10 hours. (Heine et al., 2004; Aronoff et al., 2004) The postprandial state involves numerous regulated motor, enzymatic, secretory, hormonal and metabolic events (Heine et al., 2004; Aronoff et al., 2004; Jackson, Poppitt and Minihane, 2011). Postprandial hyperglycemia and hyperlipidemia can give rise to endothelium dysfunction,

finally place the risk for atherogenesis (Heine et al., 2004; Gerich, 2003). Postprandial hyperglycemia has become known as an independent and clinically significant risk factor for cardiovascular disease in non-diabetics and diabetics. It is acknowledged as a potential therapeutic target to prevent microvascular as well as macrovascular complications then reduce cardiovascular mortality (Peter et al., 2009). Postprandial lipidemia (postprandial triglycerides concentration) also has risen as a clinically significant cardiovascular disease risk factor and it can be modulated by various lifestyle (Jackson et al., 2011).

Nutrition intervention has been generally acknowledged as the primary preventive approach to reduce the risk factors for coronary atherosclerosis (National Cholesterol Education Program Expert Panel, 2002)**.** Whole grains and cereal are recommended by National Cholesterol Education Program Expert Panel as one of the first accomplishment to increase complex carbohydrate and dietary fiber intake in variety of fiber-containing foods to achieve to metabolic control and improvement in diabetes mellitus and cardiovascular disease. For healthy people, the grain intake based on the calories requirement should be 6 servings per day and at least 1/3 should be the whole grain in one day. Fourteen gram per 1000 kcal dietary fiber is recommended for healthy population to reach the adequate amount based on calorie intake. (National Academy of Science and the Institutes of Medicine, 2002) The Adult Theraputic Program (ATP) III panel recommends that the therapeutic diet should be enriched by foods that provide total dietary fiber intake 20-30 g daily and even up to 50 g total dietary fiber per day can be beneficial **(**National Cholesterol Education Program Expert Panel, 2002; Roth et al., 2008)**.** However, an average intake of dietary fiber by the Thai adults in central Thailand was below 9 g/d (Nattinee Jitnarin et al., 2008).

Oat (*Avena sativa*) belongs to the Poaceae family is unique among the cereal with its various components, multifunctional properties and nutritional profile. Oat is not suitable for bread making since the lack of gluten, so it is often served as breakfast cereal, porridge, oat flakes, cookies made from rolled oats. The valuable nutritional components of oat include high level protein, unsaturated fatty acid, dietary fiber, antioxidative components for example vitamin E (tocopherols and tocotrienols), phenolic compounds

such as the oat-specific polyphenols avenanthramides, as well as phytic acids, sterols, and flavonoids(Sadiq Butt et al., 2008; Andersson and Hellstrand, 2012).

Beta-glucan is the main soluble dietary fiber component in the endosperm cell walls of oat. It has been generally considered due to its contribution of the cholesterol lowering and glycemic control properties as well as other component of metabolic syndrome such as blood pressure, obesity and satiety improvement (Khoury et al., 2012). The mechanisms suggested to explain the glucose lowering effects of β-glucan include the ability delayed gastric emptying followed by formation viscous solutions in the small intestine and decreased enzyme diffusion, decreased glucose transport to enterocytes, slowing subsequent digestion and absorption. Short-chain fatty acids from the anaerobic bacterial fermentation of *β*-glucan in the colon provided another possible explanatory mechanism for the effect of *β*-glucan on glucose homeostasis. However, the hypotriglycerides effect of *β*-glucan is still inconsistent and the mechanisms have not been fully determined (Khoury et al., 2012). Few mechanisms may contribute to the hypotriglycerides effect: possibly delayed absorption of triglycerides in the small intestine (Ebihara and Schneeman, 1989) as well as reduced absorption of the glucose (Liljeberg and Bj¨orck, 2000). Inhibition of lipogenesis by soluble fiber such as extracted β-glucan is also been taken as a possible explanation for the hypotriglycerides effect in vitro study (Drozdowski et al., 2010). The increased cleaning of chylomicron particles and chylomicron particles remnants from oat bran offers another possibility for the hypotriglycerides effect of *β*-glucan (Cara et al., 1992).

Besides the soluble fiber β-glucan in oat, other component such as oat-specific polyphenols avenanthramide extract was invested to reduce serum levels of total cholesterol, Low-density lipoprotein-cholesterol (LDL-c), triglycerides and increase HDL-c in humans (Liu et al., 2011). Oat is also a good source of insoluble fiber. In large cohort studies, the insoluble cereal fiber has been demonstrated to relate negatively to cardiovascular disease and diabetes, however, mechanisms linked to this component still show inconsistent effect on postprandial glycemia or lipidemia (David et al., 2000). Insoluble fiber was suggested to increase the rate of nutrients passage through the GI tract, then expected to reduce digestion and absorption of nutrients, increased insulin sensitivity

by insoluble dietary fiber might be as another contributor to the effect on glycemic improvement (Lattimer and Haub, 2010; Weickert and Pfeiffer, 2008).

The aim of the current study is to evaluate one serving of oat (35 g) in various concentrations (prepared with 169, 210 and 335 ml water) on postprandial plasma glucose and serum triglyceride levels with a high carbohydrate and high fat meal in healthy individuals.

CHAPTER II

BACKGROUND and LITERATURE REVIEW

2.1 Metabolic syndrome

The term metabolic syndrome has been refer to a series of complex disorder by intrinsically linked factors, it directly contribute to the risk of coronary heart disease, cardiovascular atherosclerotic disease as well as type 2 diabetes mellitus. According to the World Health Organization criteria (Alberti and Zimmet, 1998), metabolic syndrome is characterized by insulin resistance and any two of the following criteria: abdominal obesity, high triglycerides or/ and low high-density lipoprotein-cholesterol, high blood pressure, microalbuminuria and dysregulated glucose homeostasis. Diabetes Mellitus is generally to be described as the most common endocrine metabolic disorders while all forms of diabetes mellitus share the hyperglycemia with disturbances of carbohydrate, fat and protein metabolism results from defection of insulin production or/and insulin action. Long term hyperglycemia from whatever type 1 or type 2 diabetes can lead to microvascular and macrovascular complications involve in retinopathy, nephropathy, neuropathy as well as the cardiovascular disease. (A.D.A., 2010) The Third National Health Examination Survey 2004 reported the prevalence of Diabetes and Impaired Fasting Glucose in Thailand weighted to the national 2004 population is 6.7% and 12.5%, respectively (Wichai Aekplakorn et al., 2007).The mortality of cardiovascular disease and diabetes in noncommunicable diseases reported by World Health Organization in 2008 has grown to 280 per 100,000 female and 343 per 100,000 male in Thai populations (W.H.O., 2011: Online).

2.2 Postprandial hyperglycemia and lipidemia

Postprandial state is the period that comprises and follows a meal. Duration of the postprandial state depends on the nature of meals. After consumption a meal consists mainly of carbohydrates, 2–3 hours need to return to the basal state while a mixed meal takes 3–5 hours; And after a fat-rich meal the return to the basal state may take as long as 8–10 hours. (Heine et al., 2004; Aronoff et al., 2004) The postprandial state involves numerous regulated motor, secretory, hormonal and metabolic events (Heine et al., 2004; Aronoff et al., 2004; Jackson et al., 2011).

Hyperglycemia can lead to increased tissue glucose uptake and finally increase the risk of atherogenesis with endothelium dysfunction through several possible pathways, such as polyol and glucosamine pathway, increased nonenzymatic glycation products, glycation of certain protein, activation of diacylglycerol (DAG) and protein kinase C (PKC), decreased production of nitric oxide and increased oxidative stress (Gerich, 2003).

Postprandial hyperlipidemia can induce inflammation and oxidative stress, coagulation and fibrinolysis, vascular function and reactivity by increasing vascular tone and result in reduction in vascular reactivity involve in smooth muscle relaxation and arterial dilation (Jackson et al., 2011).

Postprandial hyperglycemia and hyperlipidemia can give rise to endothelium dysfunction, finally place the risk for atherogenesis (Heine et. al, 2004; Gerich, 2003). Postprandial lipaemia (postprandial triglycerides concentrations) also has risen as a clinically significant cardiovascular disease risk factor and it can be modulated by various lifestyle (Jackson et al., 2011).

2.3 Whole grains and dietary fiber

Nutrition intervention has been generally acknowledged as the primary preventive approach to reduce the risk factors for coronary atherosclerosis (National Cholesterol Education Program Expert Panel, 2002). The Therapeutic Lifestyle Changes Diet is a multifactorial lifestyle approach to reduce risk of coronary heart disease. Whole grains and cereal are recommended by National Cholesterol Education Program Expert Panel as one of the first accomplishment to increase complex carbohydrate and dietary fiber intake in variety of fiber-containing foods to achieve to metabolic control improvement in diabetes mellitus and cardiovascular disease. For healthy people, the grain intake based on the calories requirement should be 6 servings per day and at least 1/3 should be the whole grain in one day. Fourteen gram dietary fiber per 1000 kcal is recommended for healthy population to reach the adequate amount based on calorie intake, 38 g/d and 25 g/d for men and women ages between 19 and 50 are recommended for total dietary fiber. (National Academy of Science and the Institutes of Medicine, 2002) The Adult Teraputic Program (ATP) III panel recommends that the therapeutic diet should be enriched by foods that provide total dietary fiber intake 20-30 g daily and even up to 50 g total dietary

fiber per day can be beneficial. (National Cholesterol Education Program Expert Panel, 2002; Retelny, Neuendorf and Roth, 2008) However, the dietary fiber intake among Thai population is much lower than the recommended amount above- below 9 g/d represented by the Thai adults in central Thailand (Nattinee Jitnarin et al., 2008).

2.4 Oat and its components

Oat (*Avena sativa*) belongs to the Poaceae family is unique among the cereal with its various components, multifunctional properties and nutritional profile. Oat is not suitable for bread making since the lack of gluten, so it often served as breakfast cereal, porridge, oat flakes and cookies made from rolled oats. The valuable nutritional components include high level protein, unsaturated fatty acid, dietary fiber, antioxidative components for example vitamin E (tocopherols and tocotrienols), phenolic compounds such as the oat-specific polyphenols avenanthramides, as well as phytic acids, sterols, and flavonoids(Butt et al., 2008; Andersson and Hellstrand, 2012).

2.4.1 Established mechanism of Oat β-glucan on glycemic and lipidemic control

Oat β-glucan which is the main soluble dietary fiber component in the endosperm cell walls of oats has been generally considered due to its contribution of the cholesterol lowering and glycemic control properties as well as other component of metabolic syndrome such as blood pressure and obesity and satiety improvement (Khoury et al., 2012).

The potential mechanisms that oat *β*-glucan may lower blood cholesterol levels may also involve the viscous layer formation at the absorption surface in the small intestine which contribute to ameliorate intestinal and hepatic cholesterol and bile acid metabolism resulting in reduced circulation LDL-cholesterol level and hepatic cholesterol pool (Othman, Moghadasian and Jones, 2011). The increased fermentable production of short chain fatty acid may inhibit the cholesterol synthesis independent of bile acid. Besides cholesterol, it also may interfere the absorption of dietary fat, protein and carbohydrate.

The mechanisms suggested to explain the glucose lowering effects of β-glucan include the ability delayed gastric emptying followed by formation viscous solutions in

the small intestine and decreased enzyme diffusion, decreased glucose transport to enterocytes, slowing subsequent digestion and absorption. Short-chain fatty acids from the anaerobic bacterial fermentation of *β*-glucan in the colon provided another possible explanatory mechanism for the effect of *β*-glucan on glucose homeostasis. (Khoury et al., 2012).

However, the hypotriglycerides effect of *β*-glucan is still inconsistent and the mechanisms have not been fully determined (Khoury et al., 2012). Few mechanisms may contribute to the hypotriglycerides effect: possibly delayed absorption of triglycerides in the small intestine (Ebihara and Schneeman, 1989) as well as reduced absorption of the glucose (Liljeberg and Bj¨orck, 2000). Inhibition of lipogenesis by soluble fiber such as extracted β-glucan is also been taken as a possible explanation for the hypotriglycerides effect *in vitro* study (Drozdowski et al., 2010). The increased cleaning of chylomicron particles and chylomicron particles remnants from oat bran offers another possibility for the hypotriglycerides effect of *β*-glucan (Cara et al., 1992).

2.4.2 Established mechanism of other components in oat on glycemic and lipidemic control

Besides the soluble fiber β-glucan in oat, other component such as oat-specific polyphenols avenanthramide extract was investigated to reduce serum levels of total cholesterol, LDL cholesterol, and triglycerides and increase HDL-c in humans (Liu et al., 2011).

Even oat is also a good source of insoluble fiber. In a large cohort study, it has been demonstrated to relate negatively to cardiovascular disease and diabetes; however, mechanisms linked to this component still show inconsistent effect on postprandial glycemia or lipidemia (David et al., 2000). Insoluble fiber was suggested to increase the rate of nutrients passage through the gastrointestinal tract, then expected to reduce digestion and absorption of nutrients (Lattimer and Haub, 2010). Other contributors, for example, improved insulin sensitivity and the modulation of inflammatory markers, as well as direct and indirect influences on the gut microbiota are associated with reduced diabetes risk (Weickert and Pfeiffer, 2008).

2.5 Experimental of oat on postprandial glycemia and lipemia

2.5.1 Experimental of oat on postprandial glycemia and lipemia

Previous studies have evaluated the effect of oat flake and oat powder on postprandial glycemia (Granfeldt, Nyberg and Bjo¨rck, 2000; Ulmius, Johansson and O¨ nning, 2009). Granfeldt et al. studied the degree of gelatinization and the product thickness of rolled oats for postprandial glycemic response in healthy subjects (5 men and 5 women). Eight-two point six to ninety-four point eight gram raw or preheated (roasted or steamed) kernels thick (1.0 mm) and thin (0.5 mm) processed under conditions simulating commercial production rolled oats were made from roasted and steamed oat kernels were compared to 116.4 g white wheat bread. All test meals contain 50 g of starch, 12.0 g of protein and 6.8 g of fat also similar amount of 1621 KJ total energy. The thick oats illustrated lower peak postprandial glycemic values at 30-min values than the bread and the oat flakes processed under conditions simulating commercial processing while at 180 min, all oat products demonstrated higher glycemic responses than the white bread. All thin flakes were demonstrated with high glucose response with glycemic index (GI) 88–118 while it was not significantly different from white wheat bread ($P > 0.05$). Whereas all thick oat flakes showed significantly lower metabolic responses with GI, 70–78 than the reference bread ($P < 0.05$). However, in this study, the author did not mention the content of dietary fiber in the oats. Another experimental examined 62 g oat powder in spray dried oat drink as liquid matrix after heat treated with low amount soluble fiber (2.7 g) on postprandial glucose and triglycerides level compare to the rye bran, sugar beet fiber, mix group with three intervention above and control meal without fiber (Ulmius et al., 2009). All the test meals are in same amount of carbohydrate (75 g) adjusted and 7.9 g lipids, but the protein content (5.4 and 10 g) and energy (1641 KJ to 1725 KJ) are varied between groups. The oat powder meal resulted in a slightly increased iAUC while the sugar beet fiber and rye bran meals showed a slightly reduced glucose iAUC compared to control group, while the mixed meal was more similar to control. The incremental peak glucose responses carried out by rye bran and sugar beet fiber meals are significantly lower compared to oat powder (31 and 28%, respectively). The oat powder influenced the mixed meal to diminish the glucose-lowering effects. The postprandial

incremental triglyceride concentrations only significant higher for oat powder and the mixed meal at 60 min compared to the control meal. The possibilities that oat powder did not show any elimination in postprandial glycemic and lipidimic response may due to the critically low amount soluble fiber (2.7 g) and insoluble fiber (3.3 g total fiber) in the oat powder group. Secondly, high level of carbohydrates in the liquid matrix of the oat powder group is another possibility that did not lower the postprandial glucose response. Present results for oat powder that tended to give a high postprandial triglyceride simultaneously with high insulin and glucose responses contrast with suggested mechanism that viscous dietary fiber in oat can decrease digestion and absorption of macronutrients then followed by lowered insulin secretion and slower clearance of glucose and triglycerides from blood, maybe due to the low fiber content as suggested by the authors.

Oat powder in 20- 50 μm is the smallest one compare to rye bran< 800 μm and sugar beet fiber< 125 μm. The smaller particle size of oat powder after heat treated may also be an explanation for the process sensitive by high temperature or extreme pH for soluble fiber in oat. As concluded by Granfeldt and collages, the dependent correlation between oat flake thickness or particle size on metabolic responses and food structure after process: less disrupted outer layer of the endosperm and/or the cell walls may induce slower digestion of thick flakes, lower glycemic response and with relative lower glycemic index (Granfeldt et al., 2000). The structure degrading of soluble fiber in oats could be related to the physiological activity after consumption.

Three hundred gram sour milk with 50 g cereal bran flakes or oat flakes or the 50 g corn flakes as a reference meal were served to twelve healthy subjects with normal body mass index (Hlebowicz et al., 2007). The postprandial incremantal blood glucose level was statistically significantly lower at 40 min ($p= 0.045$) and 120 min ($p = 0.023$) after the cereal bran flakes meal compared to the cornflakes reference meal and oat flakes test meal, respectively. There was no statistical significance between the areas under the curve (AUCs) of the cereals of blood glucose was found. It was emphasized that the whole meal oat flakes meal contained only 0.5 g β-glucan was too small to affect the blood glucose response.

2.5.2 Experimental of oat bran on postprandial glycemia and lipidemia

Oat bran is the outer husk of the oat grain. It is also a good source of dietary fiber, in particular soluble fiber β-glucan. A number of studies have examined the relationship between oat bran intake and postprandial glycemic and lipedemic response.

Cara et al. (1992) reported the trial on 6 males with normal lipid profile tested with a low-fiber test meal (2.8 g insoluble dietary fiber and 70 g fat and 756 mg cholesterol, 121.3 g carbohydrate) enriched with 10 g total dietary fiber come from oat bran, rice bran, or wheat fiber or 4.2 g total dietary fiber as wheat germ. Postprandial glucose only gradually increased and reached peaked between 3 and 6 h after the ingestion of the control low-fiber test meal while the maximum rise was observed after 3.5 h. Adding oat bran fiber to the test meal induced not markedly increase in plasma glucose responses. Meanwhile, highly different individual responses were observed so the areas under the curve of postprandial glycemia did not significantly change, neither. The triglyceridemia nearly doubled 2 h after the ingestion of the control test meal. Oat bran addition significantly decreased *($P< 0.05$*) the postprandial triglyceride rise occurring before the maximal value was reached 2.5 h after the meal intake. In this study a complex, mixed, solid-liquid test meal was designed include white bread, pasta, eggs, butter, yogurt, sunflower oil and tomato sauce. Energy of the control meal consists of 12.7% protein, 37.9% carbohydrate and 49.4% fat. Since adding oat bran fiber supplied an additional 18 g carbohydrate mainly in the form of starch. This likely explains the tendency to slightly elevated glucose areas.

Redard et al. (1990) found that high-fiber (15 g) meal for 6 males and low fiber (12 g) for 6 females consumed with a test meal (42% total calories as carbohydrate, 16% as protein, and 42% as fat). Results suggest postprandial glucose and triglycerides concentrations depend on both of the fiber supplementation and genders of the subjects: males showed significant *($P< 0.05$)* glycemic elevations at 1, 2, and 3 h after the fiber-supplemented meal from baseline while glycemic response for females was significantly higher than baseline values at 2 h after high-fiber test meals and 3 h after the low-fiber test meals. Postprandial triglyceridemia in male subjects was unaffected by fiber supplementation while in females, postprandial triglycerides was greater for the high

fiber supplementation meal than for the low-fiber meal. The unchangeable postprandial glucose concentration after either high fiber or low fiber meal was explained similar with Cara et al. (1991) may be contributed by the starch in the mixed meal not the simple sugar also unpredictable interactions among the physical properties, macronutrients, and non-nutrients of a meal suggested by the authors. However, interventions tested in this experimental in the combination of oat bran and guar gum, since the modification of the glycaemia and lipedemia between oat soluble fiber and guar gum is not identified to each other suggested by Braaten et al. (1991), so the mixed results from both oat bran and guar gum should be further identified.

Fifteen subjects (8 men and 7 women) ingested meals contain 82 g oat bran meal (12.6 g dietary fiber and 5 g of soluble fiber) or a control meal without dietary fiber (Ulmius et al., 2011). Both of the meals contain oat bran, black currant beverage with pulp, white bread and the 75 g available carbohydrate was balanced by dextrose power while lipid was balanced by rapeseed oil in the control meal. Postprandial glucose after oat bran meal consumption was slightly lower than the control meal. The postprandial triglycerides showed slightly but not significantly higher after the oat bran meal compared with the control meal. All the results demonstrated without gender difference. Even the dose of oat bran is high with seemed large amount of soluble fiber induced low postprandial blood glucose; however, the reduction is not significantly different compared with control group. Also the slightly increased blood triglycerides level is not consistent with the expected effect of oat bran.

The inclusion of oat bran into the snack products with a 15% replacement to flour on postprandial glycaemia and in vitro digestibility were evaluated by Brennan et al. (2011). Twelve subjects were requested to intake a standard 25 g glucose drink also a recipe without added dietary fiber as control compared with the dietary fiber treatment. Oat bran inclusion reduced in vitro starch digestibility but not in vivo glycaemic response. In vivo postprandial blood glucose levels of the control and oat bran snacks were similar to the glucose drink at 20 min and 60 min time points. However, a shallower gradient induced by the oat bran treatment between 20 and 60 min while not observed in the other samples in the study. It was suggested that the oat bran may slow the overall digestion

rate of starch then modify the glucose release rate by observation which blood glucose concentration in the oat bran treatment did not fall back to the fasting blood glucose within the 2 h. Oat bran was incorporated into the snack products seems to extend the glycaemic response of individuals compared to the control snack, suggesting a possibility of prolonging glucose release. Authors also suggested that in vivo results cannot correlate with the results of in vitro experimental concerned of glucose response.

An effective dose dependence of extruded muesli product based on β-glucan-rich oat bran on postprandial glycaemia was found by Granfeldt and collages (Granfeldt et al., 2008). Muesli with 3 g or 4 g of β-glucans served with yoghurt, white wheat bread, cheese and butter containing 50 g available carbohydrates were given to nineteen and thirteen healthy volunteers with normal body mass index, respectively. Results indicated that muesli with 3 g of β-glucans with a mixed bread meal did not induce significant differences in glycaemic response compared to the reference meal without muesli and β-glucan, except for at one time point, 15 min, where the blood glucose level was significantly lower after the test meal with the oat bran flakes compared to the reference meal ($P< 0.05$). A reduction of the incremental area under curve (0–95 min) by 17.6% was detected compared to the reference meal, however, it did not reach the significant level. Muesli with 4 g of β-glucan significantly ($P< 0.05$) lowered the glucose responses during the first 70 min period compared to the reference breakfast based on high glycaemic index products. It was observed in the late postprandial period from 95 min to 120 min, the glucose response after the reference meal fell faster compared with the 4 g β- glucan muesli treatment meal and at 95 min the glucose response was significantly higher after the 4 g β- glucan muesli test meal than the reference meal ($P< 0.05$). The area under the glucose curve after the 4 g β- glucan muesli test meal was significantly lower than after the reference meal ($P< 0.05$). One more gram of the β-glucan in the 4 g β-glucan containing muesli induced a doubled reduction in postprandial area from 0–90 min compared to the 3 g β-glucan containing muesli test meal (17.6 and 35.5%, respectively). Although a tendency was seen to lower responses with 3 g lower β-glucans level in the musli, a total of 4 g of β-glucans from oats seems to be a critical level for a significant decrease in glycemic responses in healthy people. It also confirms the results

found by Hlebowicz et al. (2008): 24.5 g muesli includes 4 g oat β-glucan decreased the postprandial glucose response significantly compared to the cornflakes meal ($p = 0.045$) at 30 min time point but not 60 min could be possible owing to a reduced glycemic load compared to the muesli with cornflakes group.

In the analysis of effects of wheat and oat brans alone and as combination in semisolid food matrix on postprandial glycemic responses, Juvonen et al. (2011) identified semisolid meal enriched in 30 g oat bran decreased postprandial plasma glucose level. The isoenergetic and isovolumic (1250 kJ, 300 g) puddings with different insoluble and soluble dietary fiber content were tested with twenty healthy, normal-weight subjects with crossover design. Plasma glucose ($P = 0.001$) response was reported in the lowest after the pudding with the greatest amount of β-glucan (10.6 g total dietary fiber, 5.5g insoluble fiber, 5.1 g β-glucan) compared with the combination group(10.1 g total dietary fiber, 7.6 g insoluble fiber, 2.5 g β-glucan) wheat bran group and pudding with no added fiber. However, when take consideration into the time points after post-hoc analysis, the glucose concentration after the oat bran pudding was non-significantly lower at 30 min and slightly higher at 180 min compare with wheat bran treatment ($P = 0.010$) and the combination ($P= 0.018$) puddings. No significant differences in the postprandial AUC for glucose among the meals were detected. The results in this study confirm findings of previous studies demonstrating that glucose lowering effects depend on the amount of soluble fiber β-glucan in the test meal: 2.5 g of β-glucan did not decrease postprandial glycaemia in the combination group with oat bran and wheat bran is consistent with the conclusion in the study of Granfeldt et al. (2008) that 4 g oat β-glucan appears to be the minimum effective amount for a significant reduction in glucose responses.

2.5.3 Experimental of concentrate oat β-glucan and related physicochemical property concentration on postprandial glycemia

Several attempts have been made to investigate the effect of oat β-glucan concentrate on postprandial glycemia since the first experimental identified the purified oat gum lowed the postprandial blood glucose made by Braaten et al. (1991). In the study, Braaten and collages evaluated 14.5 g of oat gum consumed with 50 g glucose load were

reported significant lower plasma glucose between 20 and 60 mm *(P‹ 0.01*), mean AUC of plasma glucose decreased to 57.2%, compared to glucose load alone in nine healthy, fasting subjects. Even response of oat gum meal was nearly identical to guar gum meal, the oat gum showed more palatable than the latter intervention. Compared with the glucose drink (3.0 ± 0.3 mmol/L), peak above baseline was also lower after the oat gum (1.9 ± 0.3 mmol/L) *(P‹ 0.0 1*).

The whole-meal rye bread fortified with oat β-glucan concentrate was explored as one of the interventions to evaluate factors can modify the postprandial glycemic response (Juntunen et al., 2002). Each product provided 50 g available carbohydrate in all of the treatments: whole-kernel rye bread, dark durum wheat pasta, and wheat bread made from white wheat flour, whole meal rye bread containing 5.4 g oat β-glucan, the results indicate that even the differences in glucose responses during the first postprandial 90 min were not significant, glucose responses to β-glucan fortified rye bread at postprandial 120 min (*P = 0.012*) were greater than the response to white wheat bread. The author concluded the glucose response of oat β-glycan containing rye bread may come from the detected reduced average molecular weight prior to bake (› 1 000 000) and after bake(‹ 250 000) probably induced by endogenous β-glucanases of rye flour during baking. The starch hydrolysis rate of β-glucan fortified rye bread also was analyzed, it was not significantly different from that of white wheat bread, even with 5.4 g high content of soluble fiber in the rye bread. The author concluded that the different glycemic index of the starchy food may induce by the altered structure of those foods despite the diets are in identical nutrients compositions as well as type and amount of dietary fiber.

The relationship between concentration and molecular weight of β-glucan and glycaemic response to an oral glucose load from established clinical studies was analyzed by Wood and collages (Wood, Beer and Butler, 1999). They revealed that significant relationship between changes in peak blood glucose and a combination of logarithm of the concentration and logarithm of Molecular weight of β-glucan: $\Delta G = 7.93 - 0.68 \log10(c) - 1.01 \log10(Mw)$. The authors also emphasized that real foods will have various differences from the theoretical drink model induced glycaemic response and related logarithmically to concentration and molecular weight established. The factors may

interfere the effective correlation above in reality may come from following: unfixed liquid volume of intake as well as interactions and microstructure from other food not just in the glucose drink load may lower solubility or depolymerisation of the polysaccharides with reduced physiological effectiveness. Lastly, particulate matter in the gastrointestinal tract is not as simple as measurements of the treatments consumed in the experimental can remarkable modify the rheological behavior of soluble fiber. In 2009, Regand and collages verified the relationship exists between physicochemical properties of β-Glucan in differently processed oat foods and postprandial glycemic response (Regand et al., 2009). Isocaleric crisp bread, granola, porridge, and pasta include 4 g of β-glucan and control products with low β-glucan content were induced to the in vitro as well as in vivo experimental. The source of β-glucan in oat bread and pasta was only Oatwell 22 which contains 22% β-glucan while oat granola and porridge were prepared with a mix of either whole oat flakes (containing 4.3% β-glucan) or oat bran (containing 7.4% β-glucan), in addition to Oatwell. The physicochemical properties of β-glucan include viscosity, peak molecular weight (Mp), and concentrations (C) were evaluated in vitro extract. Fasting and postprandial blood glucose concentrations were evaluated in human subjects. Porridge and granola were found with the highest efficacy in attenuating the peak blood glucose response (PBGR) owing to their high Mp and viscosity when compared to the wheat muffin as control group ($p< 0.05$). Pastas, known to have low glycemic responses, showed the lowest peak blood glucose response. Beta-Glucan depolymerization in bread and pasta reduced β-glucan bioactivity. The analyses of these products demonstrated that there was a significant inverse linear relationship between (Mp ×C) and mean PBGR. 73% of the bioactivity in decreasing peak blood glucose response can be explained by Mp × C when the wheat muffin control was used as a reference for five oat products above are compared to their own wheat controls. The authors also explain the when prepared the oat porridge, the ingredients were boiled at 100 ℃ and then simmered lasted for 5 min in wet heat. Short preparation times which resulted in high β-glucan Mp values. The glycosidic linkages of β-glucan appear to be stable during the heat treatment. Meanwhile, the authors suggested that β-glucan bioactivity should be considered with critical factors such as the effect of microstructure and/or starch digestibility. Gradient in the reduction

of glycemic response is correlated to the MW ×concentration of β-glucan. The intrinsic physicochemical properties of β-glucan include concentration mainly depend on the raw materials used and their processing history.

Two studies demonstrate that 5.1 g of β-glucan as the oat bran beverage form had more effective postprandial effect on glucose than what was observed here after an equal amount of β-glucan in semisolid meal in (Juvonen et al., 2009; Juvonen et al., 2011) . This may indicate smaller differences in rheological property in the stomach and gut lumen after the semisolid test meals than after the liquid meals. So, not only the importance of food structure, but also food matrix in postprandial metabolism should be emphasized. However, the concentration of β-glucan from the oat bran in the beverage treatments did not been further evaluated in this experimental and no control group was designed to evaluate how different physicochemical properties of β-glucan containing beverages to modify the postprandial glucemic and lipidemic responses.

CHAPTER III

MATERIALS and METHODS

3.1 Subjects

Inclusion criteria for selecting the subjects were as follows: adult age between 18-45 years old; BMI between 18.5-22.9 kg/ m²; fasting total cholesterol‹ 200mg/dl; fasting triglycerides‹ 150 mg/dl; no chronic disease. If any participant were in the following criteria, they were excluded from the study: fasting blood glucose › 100 mg/dL; chronic diseases includes metabolic disease, cardiovascular disease, liver or kidney disease; gastrointestinal disease; intake of any medication or dietary supplementation two weeks before the study; smoking and alcohol drinking. The study was approved by the office of Ethics Review Committee for Research Involving Human Research Subjects, Human Science Group, Chulalongkorn University (Appendix C). Every participant signed the informed consent before the study.

The night before every test breakfast, subjects were requested to be fasting 8 to 10 hours and allowed to drink only water. Participants were recommended to keep their physical activity and lifestyle unchanged throughout the study.

3.2 Oatmeal preparation

The Quick cooking oatmeal (The Quaker Oats Company, Made in Malaysia) were obtained from supermarket on one batch. One serving of oat (35 g) was prepared with three content of water (169 ml, 210 ml and 335 ml) to induce different concentration by cooking 4 minutes over 80℃ (Table 3.2). All the oatmeal was served with the container in the same size after cooking and with the same time intervals (7 minutes) prior to serving to each participant.

3.3 High carbohydrate and high fat meal

Three test meals: high carbohydrate and high fat breakfast and three different concentration of oatmeal 1 serving oat flakes 35 g and three amount of water (169 ml, 210 ml and 335 ml) were served to the subjects in randomized fashion. High carbohydrate and high fat consists of one serving (two slice, 48 g) of white bread (Farm House company, Thailand), two servings (30 g) unsalted butter (Anchor, New Zealand), glucose solution made of 75 g glucose diluted with 125 ml drinking water, 100 ml UHT milk (Thai-Danish, Thailand) and 2 tablespoon (30 ml) of whipping cream (Anchor, New

Zealand), 25 ml drinking water (Figure 3.1). The nutritional information was shown in Table 3.1. The interventional breakfast meals were randomly served. Each clinic visit will be separated by at least one week wash-out periods.

Table 3.1 A high carbohydrate and high fat meal (HCHF) nutrition composition.

Ingredients / **Nutrituion composition**	**Glucose**	**White bread** (2 slices, 48 g , 1 serving)	**Butter** (30 g, 2 serving)	**Milk** (100 ml)	**Whipping Cream** (30 g)	**Total weight** (g)	**Total energy** (kcal)
CHO	75 g	22 g	0.18g	3.6 g	0.93 g	101.71	406.84
Fat	-	2.5g	24.8g	4 g	10.6 g	41.9	377.1
Protein	-	5 g	0.18 g	4 g	0.8g	9.98	39.92
Total energy	823.86 kcal						
CHO: fat: protein	49:46:5						

Table 3.2 The different concentration of oat 1 serving (35 g) prepared by various amount of water.

Different amount of water (mL) with oat 1 serving (35 g)	**Concentration (g/ mL)**
169	0.207
210	0.176
335	0.104

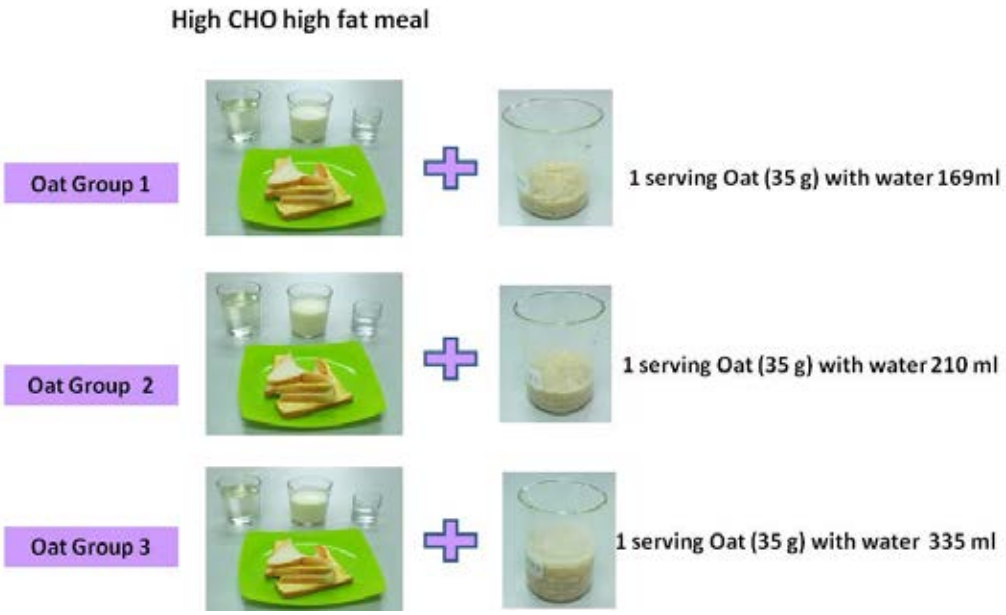

Figure 3.1 Components of three test meals. Participants were induced hyperglycemia and hypertriglyceridemia by a high carbohydrate and high fat meal (HCHF) which composed of 101.7g carbohydrate (49% of total energy) and 41.9g fat (46 % of total energy). One serving oat (35 g) contains 139 kcal of energy with 23 g carbohydrate, 3 g fat and 5 g protein.

3.4 Blood sample analysis

In each clinic visit, venous blood samples were obtained by the intravenous catheter inserted into the forearm vein. The blood samples were obtained at fasting state before the intervention, then the postprandial blood was collected at 1 hour, 3 hour and 5 hour after intervention assigned in each clinic visit. Blood sample were kept in blood sample collect tubes with Sodium Fluoride (EDTA, 3ml) and BD vacutainer with clotting activator to separate blood plasma and serum (6 ml) after centrifuge immediately at 3000 rpm for 10 min at 20℃. All the plasma and serum were sent to the Health center of Allied Health Science Faculty, Chulalongkorn University.

Venous blood glucose was analyzed by the automatic analyzer-Flexor (Netherlands). Blood glucose was determined after enzymatic oxidation in the presence of glucose oxidase. The hydrogen peroxide formed reacts, under catalysis of peroxidase,

with phenol and 4-aminophenazone to form a red –violet quinoneimine dye as indicator. Venous blood triglycerides were determined after enzymatic hydrolysis with lipases. The indicator is a quinoneimine formed from hydrogen-peroxide, 4-aminophenazone and 4-chlorophenol under the catalytic influence of peroxidase. Blood total cholesterol was determined after enzymatic hydrolysis and oxidation. The indicator quinoneimine is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxidase. Direct HDL-c was determinated by the assay consists of 2 reaction steps which are elimination of chylomicron, Very low-density lipoprotein-cholesterol (VLDL-c) and LDL-c by cholesterol esterase, cholesterol oxidase and subsequently catalase then followed by measurement of HDL-c after release of HDL-c by detergents. LDL-c was calculated by the Friedewald equation**.**

3.5 Statistical analysis

The characteristics of the subjects in screening and results in three visits were given as means ± Standard Error of the Mean (SEMs). Area Under Curve (AUC) and incremental Area Under Curve (iAUC) of blood glucose and triglyceride from 0 to 5 hours was calculated by Trapetzoidal method. The effect of treatment for postprandial glucose and triglycerides, incremental (Δ) of postprandial glucose and triglycerides, AUC and iAUC of postprandial glucose and triglycerides were submitted to one way repeated ANOVA. Bonferroni test was used to control multiple comparisons to assess the difference between individual means. Statistical significance defined as $p < 0.05$ by SPSS version 17.0 (SPSS, Chicago, IL, USA).

CHAPTER IV

RESULTS

Eleven healthy participants (10 female and 1 male) were included to the study after screening with normal fasting lipid profile and body mass index. Table 4.1 presents baseline characteristics of the 11 participants. Included participants were in average age 21.03 years old with normal BMI 20.47 and normal serum total cholesterol 172.55 mg/ dL.

Table 4.1 Baseline characteristics of 11 participants.

Characteristics	**Results** (Mean ±SEM)
Age (year)	21.03 ±0.50
BMI (kg/ m²)	20.47 ±0.37
Serum Total Cholesterol （mg/ dL)	172.55 ±3.66
Serum HDL-c （mg/ dL)	51.18 ±2.00
Serum LDL-c （mg/ dL)	110.55 ±3.59
Serum Triglycerides （mg/ dL)	56.86 ±2.31
Plasma glucose （mg/ dL)	83.45 ±0.86

Each clinic visit after 8-10 hours fasting, subjects were induced hyperglycemia and hypertriglyceridemia by high carbohydrate and high fat meal (HCHF). HCHF meal included two slice of white bread (48g), two servings (30 g) unsalted butter, glucose solution made of 75 g glucose diluted with 125 ml drinking water, 100 ml UHT milk and 2 tablespoon (30 ml) of whipping cream, 25 ml drinking water. Test meals were 1 serving size of oats boiled in different amount of water: 169, 210, 335 ml.

The peaks of postprandial glucose were at 1 hour after all test meals as shown in figure 4.1 and Table 4.2. Plasma glucose of oat 1 serving with 169, 210, 335 ml water and HCHF meal were 127.09, 140.82 and 133.27 mg/ dL, respectively. At hour 3, postprandial blood glucose for corresponding test meals were 95.00, 103.73 and 107.55 mg/ dL. At hour 5, postprandial blood glucose for the three tested meals dropped to 78.00, 74.73 and 78.18 mg/ dL (Table 4.2). Effect of ingestion of oatmeal in different concentration induced by distinctive amount of water (169, 210 and 335 ml) with HCHF meal on postprandial blood glucose analyzed by one way repeated ANOVA with non-significant different: oat + 169 ml water vs. oat + 210 ml water p= 0.429; oat + 169 ml

water vs. oat + 335 ml water p= 0.436; oat + 210 ml water vs. oat + 335 ml water p= 1.000 when compare between each group after adjusted by multiple comparison.

Table 4.2 Effect of different concentration oatmeal with HCHF on postprandial plasma glucose level (mg/ dL).

Time point / Test meal	Fasting (Mean±SEM)	Hour 1 (Mean ±SEM)	Hour 3 (Mean ±SEM)	Hour 5 (Mean±SEM)
Oat 35g + 169 ml water + HCHF meal	82.82 ±1.89	127.09 ±11.22	95.00 ±5.15	78.00 ±2.32
Oat 35g + 210 ml water + HCHF meal	83.00 ±1.33	140.82 ±12.22	103.73 ± 7.63	74.73 ± 2.63
Oat 35g + 335 ml water + HCHF meal	84.55 ±1.26	133.27 ±12.72	107.55 ±11.95	78.18 ±3.19

The peaks of postprandial glucose were at 1hr after all test meals. The plasma glucose of oat 1 serving with 169, 210 and 335 ml water and HCHF meal were not significantly different with each other (oat + 169 ml water vs. oat + 210 ml water p= 0.429; oat + 169 ml water vs. oat + 335 ml water p= 0.436; oat + 210 ml water vs. oat + 335 ml water p= 1.000).

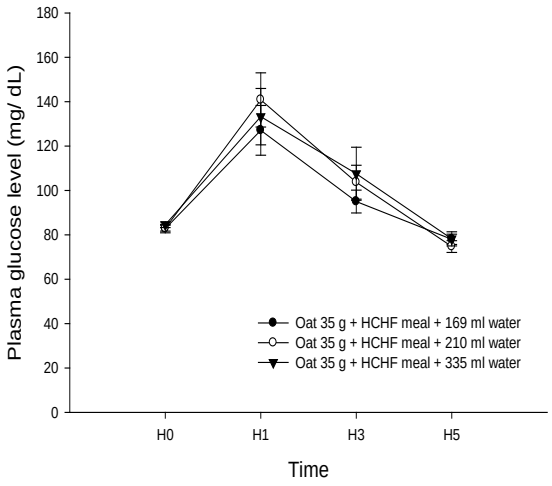

Figure 4.1 Effect of different concentration oatmeal with HCHF on postprandial plasma glucose level (mg/ dL). The peaks of postprandial glucose were at 1hr after all test meals. The plasma glucose of oat 1 serving with 169, 210 and 335 ml water and HCHF meal were not significantly different with each other (oat + 169 ml water vs. oat + 210 ml water p= 0.429; oat + 169 ml water vs. oat + 335 ml water p= 0.436; oat + 210 ml water vs. oat + 335 ml water p= 1.000)

Hypertriglyceridemia were induced by the 1 serving oat flakes with HCHF meal in 5 hours after consumption of three tests groups. Figure 4.2 and Table 4.3 compare the results induced by HCHF meal and oat 1 serving (35 g) with 169, 210 and 335 ml water on postprandial triglyceride levels. At Hour 1, postprandial triglyceride for oat 1 serving (35 g) with 169, 210 and 335 ml water plus HCHF meal were 60.27, 63.11and 62.89 mg / dL. At Hour 3, postprandial triglyceride after three tested meals intake were 70.55, 65.00, and 71.20 mg/ dL. At Hour 5, postprandial triglyceride for the corresponding meals gradual rose to 82.45, 80.00 and 81.40 mg/ dL (Table 4.3). After assessed by one way repeated ANOVA, no significant effect on postprandial triglycerides level was found between three tested meals after multiple comparisons: oat + 169 ml water vs. oat + 210 ml water p= 0.285; oat + 169 ml water vs. oat + 335 ml water p= 0.840; oat + 210 ml water vs. oat + 335 ml water p= 1.000**.**

Table 4.3 Effect of different concentration oatmeal with HCHF on postprandial serum triglycerides level (mg/ dL).

Time point / **Test meal**	**Fasting** (Mean ±SEM)	**Hour 1** (Mean ±SEM)	**Hour 3** (Mean ±SEM)	**Hour 5** (Mean ±SEM)
Oat 35g + 169 ml water + HCHF meal	59.27 ±4.60	60.27 ±7.58	70.55 ±9.51	82.45 ±10.18
Oat 35g + 210 ml water + HCHF meal	56.78 ±2.80	63.11 ±4.78	65.00 ±7.91	80.00 ±5.07
Oat 35g + 335 ml water + HCHF meal	54.00 ±4.23	62.89 ±6.25	71.20 ±10.77	81.40 ±6.14

The serum triglycerides of oat 1 serving with 169, 210, 335 ml water and HCHF were not significantly different after 5 hours of consumptions(oat + 169 ml water vs. oat + 210 ml water p= 0.285; oat + 169 ml water vs. oat + 335 ml water p= 0.840; oat + 210 ml water vs. oat + 335 ml water p= 1.000)**.**

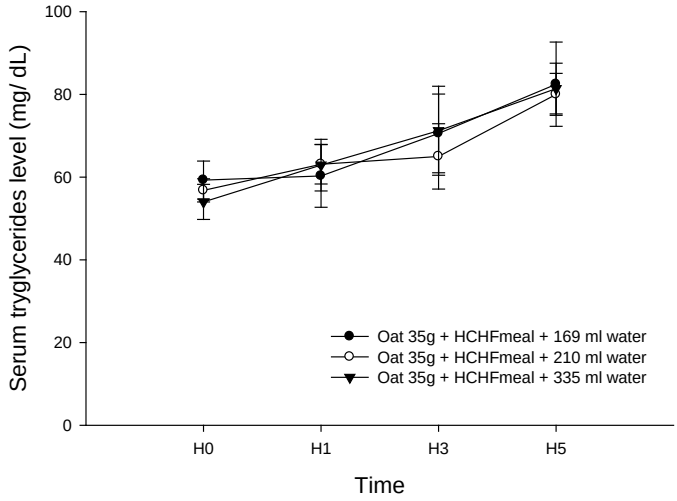

Figure 4.2 Effect of different concentration oatmeal with HCHF on postprandial serum triglycerides level (mg/ dL). The serum triglycerides of oat 1 serving with 169, 210, 335 ml water and HCHF meal were not significantly different after 5 hours of consumptions (oat + 169 ml water vs. oat + 210 ml water p= 0.285; oat + 169 ml water vs. oat + 335 ml water p= 0.840; oat + 210 ml water vs. oat + 335 ml water p= 1.000)**.**

The Area Under curve(AUC) of blood glucose for 1 serving oat in 169, 210 and 335 ml water with HCHF meal as provided in Table 4.4 from fasting to Hour 1 , Hour 3 and Hour 5, respectively. AUC of blood glucose in 5 hour experimental time period are 500.09, 535.09 mg/ dL and 535.73 for blood glucose present in Figure 4.3. The Area Under curve (AUC) of blood triglycerides for 1 serving oat in 169, 210 and 335 ml water with HCHF meal as provided in Table 4.5 from fasting to Hour 1 , Hour 5 and Hour 5, respectively. AUC of blood triglycerides in 5 hour experimental time period are 330.83, 343.50 and 366.83 mg/ dL for blood triglyceride as shown in Figure 4.4. No significant effect on blood glucose (oat + 169 ml water vs. oat + 210 ml water p= 1.000; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000) and triglycerides level (oat + 169 ml water vs. oat + 210 ml water p= 1.000; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000) were found among the three test oat meals between each other after multiple comparisons in one way repeated ANOVA.

Table 4.4 Area Under curve (AUC) of blood glucose response of three test meals (mg/dL).

Time period / Test meal	Fasting to Hour 1 (Mean ±SEM)	Fasting to Hour 3 (Mean ±SEM)	Fasting to Hour 5 (Mean ±SEM)
Oat 35g + 169 ml water + HCHF	222.00±15.64	172.91±4.36	500.09±23.81
Oat 35g + 210 ml water + HCHF	244.55±19.02	178.46±7.06	535.09±31.56
Oat 35g + 335 ml water + HCHF	240.82±23.32	185.73±13.18	535.73±41.72

AUC of blood glucose response of oat 1 serving with 169, 210 and 335 ml water and HCHF meal were not significantly different compare to each group. (oat + 169 ml water vs. oat + 210 ml water p= 1.000; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000)

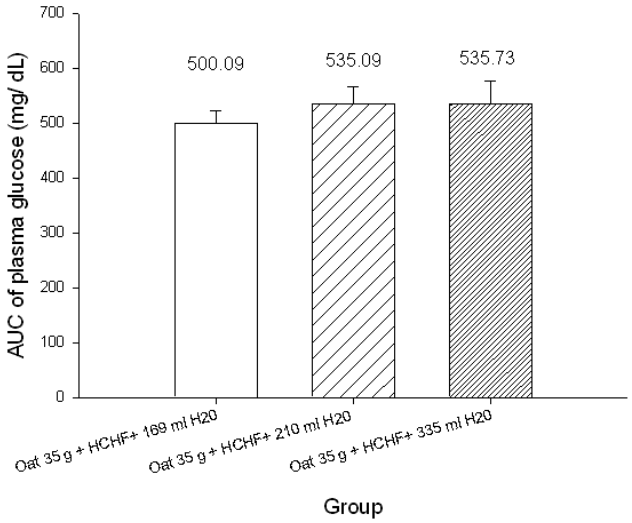

Figure 4.3 Area Under curve (AUC) of blood glucose response of three test meals in 5 hour experimental period (mg/ dL). AUC of blood glucose response of oat 1 serving with 169, 210 and 335 ml water and HCHF meal were not significantly different compared to each group (oat + 169 ml water vs. oat + 210 ml water p= 1.000; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000)**.**

Table 4.5 Area Under curve (AUC) of blood triglycerides response of three test meal (mg/ dL).

Time period / Test meal	Fasting to Hour 1 (Mean ±SEM)	Fasting to Hour 3 (Mean ±SEM)	Fasting to Hour 5 (Mean ±SEM)
Oat 35g + 169 ml water + HCHF	126.33±14.58	145.00±16.02	330.83±33.87
Oat 35g + 210 ml water + HCHF	130.67±17.54	151.33±16.16	343.50±36.92
Oat 35g + 335 ml water + HCHF	143.00±26.34	163.17±21.88	366.83±54.87

AUC of blood triglycerides response of oat 1 serving with 169, 210 and 335 ml water and HCHF meal were not significantly different compare to each group (oat + 169 ml water vs. oat + 210 ml water p= 1.000; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000)**.**

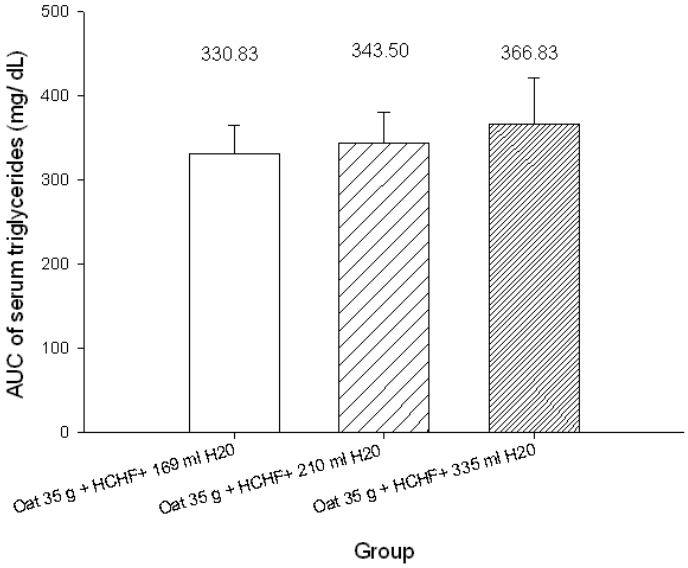

Figure 4.4 Area Under curve (AUC) of blood triglycerides response of three test meals in 5 hour experimental period (mg/ dL). AUC of blood triglycerides response of oat 1 serving with 169, 210 and 335 ml water and HCHF meal were not significantly different compare to each group (oat + 169 ml water vs. oat + 210 ml water p= 1.000; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000)**.**

The incremental glucose concentrations were demonstrated in Table 4.6 and Figure 4.5. Incremental Plasma glucose of oat 1 serving with 169, 210, 335 ml water and HCHF meal in Hour 1 were 44.27, 57.82 and 48.73 mg/ dL, respectively. At hour 3, incremental blood glucose concentration for corresponding test meals were 12.18, 20.73, and 23.00 mg/ dL. At hour 5, incremental blood glucose concentration for the tested meals decreased to -4.82, -8.27 and -6.36 mg/ dL (Table 4.6; Figure 4.5). Effect of ingestion of oatmeal in different concentration induced by distinctive amount of water (169, 210 and 335 ml) with high carbohydrate and high fat meal on incremental blood glucose concentration analyzed by one way repeated ANOVA, no significant difference was detected after adjusted for multiple comparison: oat + 169 ml water vs. oat + 210 ml water p= 0.479; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000**.**

Table 4.6 Effect of different concentration oatmeal with HCHF on incremental plasma glucose concentrations (mg/ dL).

Time point / **Test meal**	**Hour 1** (Mean ±SEM)	**Hour 3** (Mean ±SEM)	**Hour 5** (Mean ±SEM)
Oat 35g + 169 ml water + HCHF meal	44.27±12.46	12.18±6.44	-4.82±2.09
Oat 35g + 210 ml water + HCHF meal	57.82±12.29	20.73±7.58	-8.27±3.09
Oat 35g + 335 ml water + HCHF meal	48.73±13.09	23.00±11.90	-6.36±2.67

The incremental plasma glucose concentration of oat 1 serving with 169, 210 and 335 ml water and HCHF meal were not significantly different with each other. (oat + 169 ml water vs. oat + 210 ml water p= 0.479; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000)**.**

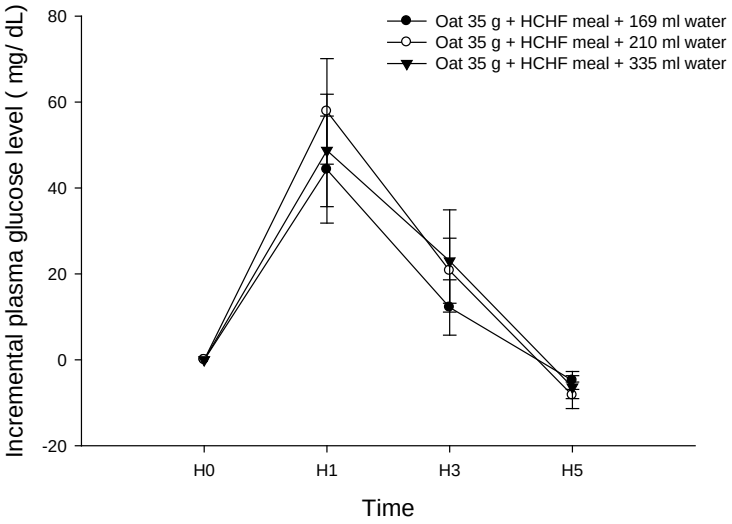

Figure 4.5 Effect of different concentration oatmeal with HCHF on incremental plasma glucose concentration (mg/ dL). The incremental plasma glucose concentration of oat 1 serving with 169, 210 and 335 ml water and HCHF meal were not significantly different with each other. (oat + 169 ml water vs. oat + 210 ml water p= 0.479; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000)**.**

Figure 4.6 and Table 4.7 compare the incremental triglyceride concentration induced by high carbohydrate and high fat meal with oat 1 serving (35 g) in 169, 210 and 335 ml water. At Hour 1, incremental triglyceride for HCHF meal plus oat 1 serving (35 g) with 169, 210 and 335 ml water were 1.00, 6.33 and 9.50 mg / dL. At Hour 3, incremental triglyceride concentration after three tested meals intake were 11.27, 8.22 and 19.33 mg/ dL. At Hour 5, incremental triglyceride concentrations for the corresponding meals grow to 23.18, 23.22 and 31.00 mg/ dL (Table 4.7; Figure 4.6). After assessed by one way repeated ANOVA, no significant effect on postprandial incremental triglycerides concentration was detected among the three tested meals after multiple comparisons by Bonferroni test: oat + 169 ml water vs. oat + 210 ml water p=1.000; oat + 169 ml water vs. oat + 335 ml water p= 0.716; oat + 210 ml water vs. oat + 335 ml water p= 0.089.

Table 4.7 Effect of different concentration oatmeal with HCHF on incremental serum triglycerides concentration (mg/ dL).

Time point / Test meal	Hour 1 (Mean ±SEM)	Hour 3 (Mean ±SEM)	Hour 5 (Mean ±SEM)
Oat 35g + 169 ml water + HCHF meal	1.00±5.35	11.27±6.79	23.18±8.16
Oat 35g + 210 ml water + HCHF meal	6.33±4.52	8.22±7.66	23.22±5.73
Oat 35g + 335 ml water + HCHF meal	9.50±3.59	19.33±8.67	31.00±6.07

incremental serum triglycerides concentration (mg/ dL) for oat 1 serving with 169, 210, 335 ml water and HCHF meal were not significantly different after 5 hours of consumption (oat + 169 ml water vs. oat + 210 ml water p=1.000; oat + 169 ml water vs. oat + 335 ml water p= 0.716; oat + 210 ml water vs. oat + 335 ml water p= 0.089)

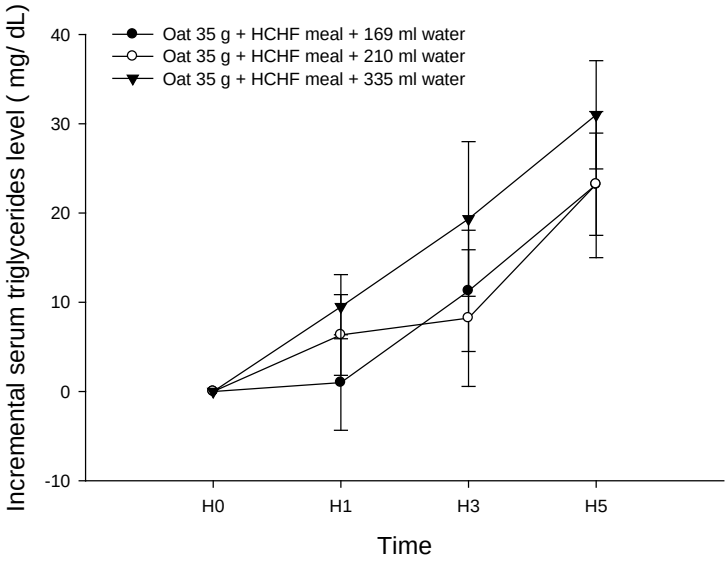

Figure 4.6 Effect of different concentration oatmeal with HCHF on incremental serum triglycerides concentration (mg/ dL). incremental serum triglycerides concentration (mg/ dL) for oat 1 serving with 169, 210, 335 ml water and HCHF meal were not significantly different after 5 hours of consumption (oat + 169 ml water vs. oat + 210 ml water p=1.000; oat + 169 ml water vs. oat + 335 ml water p= 0.716; oat + 210 ml water vs. oat + 335 ml water p= 0.089).

The incremental Area Under curve (iAUC) of blood glucose of 1 serving oat in 169, 210 and 335 ml water and HCHF meal as provided in Table 4.8 from fasting to Hour 1 , Hour 3 and Hour 5, respectively. iAUC of blood glucose in 5 hour experimental time period are 86.00, 120.09 mg/ dL and 112.91 for blood glucose present in Figure 4.7. The iAUC of blood triglycerides for 1 serving oat in 169, 210 and 335 ml water with HCHF meal as provided in Table 4.9 from fasting to Hour 1, Hour 3 and Hour 5, respectively. iAUC of blood triglycerides in 5 hour experimental time period are 47.50, 48.33 and 85.83 mg/ dL for blood triglyceride as shown in Figure 4.8. No significant effect on blood glucose and triglycerides level were found among the three test oat meals between each other after multiple comparisons by Bonferroni test.

Table 4.8 Incremental Area Under curve (iAUC) of blood glucose response of three test meals (mg/dL).

Time period / **Test meal**	**Fasting to Hour 1** (Mean±SEM)	**Fasting to Hour 3** (Mean±SEM)	**Fasting to Hour 5** (Mean ±SEM)
Oat 35g + 169 ml water + HCHF	22.36± 6.24	78.82± 24.40	86.00± 30.41
Oat 35g + 210 ml water + HCHF	29.09± 6.17	107.64± 25.12	120.09± 31.80
Oat 35g + 335 ml water + HCHF	24.55± 6.56	96.27± 29.98	112.91± 42.24

iAUC of blood glucose response of oat 1 serving with 169, 210 and 335 ml water and HCHF meal were not significantly different compare to each group. (oat + 169 ml water vs. oat + 210 ml water p= 1.000; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000)**.**

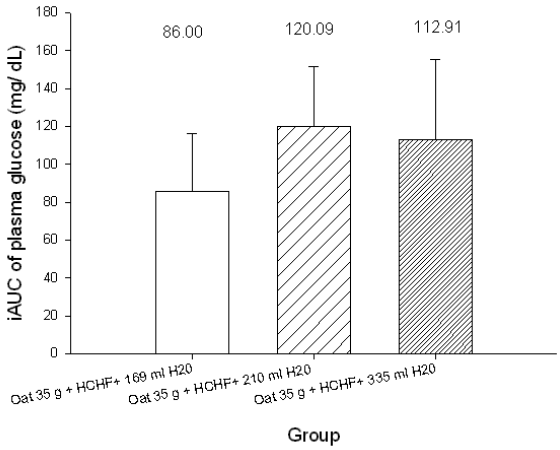

Figure 4.7 Incremental Area Under curve (iAUC) of blood glucose response of three test meals in 5 hour experimental period (mg/ dL). iAUC of blood glucose response of three test meal were not significantly different compared to each group (oat + 169 ml water vs. oat + 210 ml water p= 1.000; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000)**.**

Table 4.9 Incremental Area Under curve (iAUC) of blood triglycerides response of three test meal (mg/ dL).

Time period / **Test meal**	**Fasting to Hour 1** (Mean ±SEM)	**Fasting to Hour 3** (Mean ±SEM)	**Fasting to Hour 5** (Mean ±SEM)
Oat 35g + 169 ml water + HCHF	2.83± 3.34	15.67± 16.74	47.50± 32.81
Oat 35g + 210 ml water + HCHF	2.17± 3.49	14.83± 20.05	48.33± 36.08
Oat 35g + 335 ml water + HCHF	4.50± 1.86	35.17± 17.83	85.83± 31.57

iAUC of blood glucose response of oat 1 serving in 169, 210 and 335 ml water with HCHF meal were not significantly different compare to each group. (oat + 169 ml water vs. oat + 210 ml water p= 1.000; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000) from fasting to H1 and H5. (oat + 169 ml water vs. oat + 210 ml water p= 1.000; oat + 169 ml water vs. oat + 335 ml water p= 0.906; oat + 210 ml water vs. oat + 335 ml water p= 1.000) from fasting to H3.

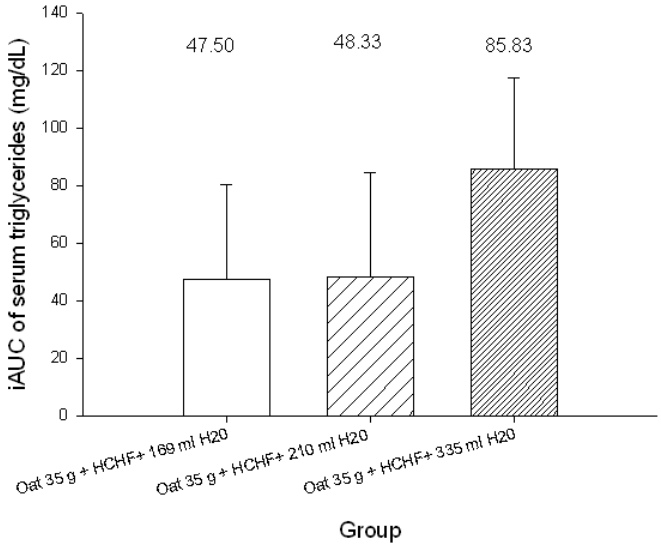

Figure 4.8 Incremental Area Under curve (iAUC) of blood triglycerides response of three test meals in 5 hour experimental period (mg/ dL). iAUC of blood triglycerides response of oat 1 serving with 169, 210 and 335 ml water and HCHF meal were not significantly different compare to each group (oat + 169 ml water vs. oat + 210 ml water p= 1.000; oat + 169 ml water vs. oat + 335 ml water p= 1.000; oat + 210 ml water vs. oat + 335 ml water p= 1.000) .

The individually incremental level from fasting to Hour 1, Hour 3 and Hour 5 and percentage difference (% Difference) when compare the incremental level in Hour 1, Hour 3, Hour 5 to fasting for plasma glucose and serum triglycerides concentration of oat 1 serving with 169, 210 and 335 ml water and HCHF meal were shown in Appendix A and Appendix B, respectively.

The time duration (minutes) of each participant consumed the three test groups were presented in Table 4.10.

Table 4.10 Time duration (minutes) of each participant consumed the three test groups.

Test meal / Subject	Oat 35g + HCHF + 169 ml H_20	Oat 35g + HCHF + 210 ml H_20	Oat 35g + HCHF + 335 ml H_20
Subject No.1	50	60	44
Subject No.2	36	45	45
Subject No.3	20	27	20
Subject No.4	20	20	35
Subject No.5	20	20	37
Subject No.6	40	35	55
Subject No.7	20	15	25
Subject No.8	30	27	48
Subject No.9	20	20	38
Subject No.10	35	27	35
Subject No.11	40	43	25

CHAPTER V
DISCUSSION

A potent anti-hypercholesterolemic effect of β-glucan has been found in oat (*Avena sativa*). However, the effect of oat flakes on plasma glucose and serum triglycerides is not yet well established. The purpose of the current study was to evaluate the effect of 1 serving of oat flakes (35 g) in different concentration (prepared with 169, 210 and 335 ml water) with high carbohydrate, high fat meal on postprandial plasma glucose and serum triglyceride levels in healthy individuals.

Results of this study indicate that different concentrations of oat flakes with high carbohydrate and high fat meal did not induce significant effect on postprandial plasma glucose and serum triglycerides levels of the 11 healthy participants.

There are several possible explanations for the results. These insignificant modification on postprandial plasma glucose and serum triglycerides level with 1 serving oat (35 g) induced by 169, 210 and 335 ml water in the intervention meals may be the lack of adequate dietary fiber. One serving oat contains 3 g of dietary fiber in the oat flakes of our study. Juvonen et al. (2011) identified plasma glucose response in the lowest level after the isoenergetic and isovolumic pudding with the greatest amount dietary fiber from oat source (10.6 g total dietary fiber) compared with the combination group (10.1 g total dietary fiber) with wheat bran, wheat bran group and pudding with no added fiber. The results in this study confirmed that glucose lowering effect depend on the amount of oat source of dietary fiber in the test meal.

The serum triglycerides level of oat 1 serving oats were not significantly different after 5 hours of consumptions. Earlier finding suggested a low-fiber control meal contains 2.8 g insoluble dietary fiber, 70 g fat and 756 mg cholesterol, 121.3 g carbohydrate enriched with 10 g total oat source dietary fiber significantly decreased the postprandial triglyceride rise occurring before the maximal value (Cara et al., 1992). However, in our present study, the amount of total dietary fiber is 3 g in the 1 serving (35 g) oat flakes in the three test meals, this critical factor may be one reason to explain the insignificant amelioration of oatmeal on postprandial hyperglycemia and hypertriglyceridemia.

The oat flakes in the present study is the commercial Quick cooking oatmeal, some studies (Granfeldt et al., 2000; Ulmius et al., 2009) have speculated that postprandial glycemic response is related to the size of oat flakes. Thin flakes processed under conditions imitating commercial processing was demonstrated with high glucose response with glycemic index (GI) 88–118 while it was not significantly different from white wheat bread; whereas all thick oat flakes showed significantly lower metabolic responses with GI 70–78 than the reference bread (Granfeldt et al., 2000). Oat powder in spray dried oat drink as liquid matrix after heat treated with low amount total dietary fiber (3.3g) and slightly increased postprandial glucose iAUC compared to control group with same amount of available carbohydrate (75 g) and influenced the effects of the mixed meal, diminishing the glucose-lowering effects while postprandial incremental triglyceride concentrations was only significant higher for oat powder compared to control meal. It can therefore assume that the dependent correlation between oat flake thickness on metabolic responses and food structure after process: less disrupted oat outer layer of the cell walls may induce slower digestion of oat thick flakes, lower glycemic response and with relative lower glycemic index.

The hypoglycemia and hypo-triglycerides effect has been found in previous studies with equal or similar carbohydrate available in the oat containing test meals. It was reported by Granfeldt et al. (2002) in an experimental to determine the postprandial glycemic response with different degree improvement induced by test meals consisted of the oat flake products (82.6 –94.8 g) in distinctive flakes thickness compared to the reference bread meal (116.4 g). All meals providing 50 g of starch, 12.0 g of protein based on similar energy. In another experimental, four groups isoenergetic and isovolumic pudding were intervened by Juvonen et al. (2011): oat fiber group, oat fiber combined with wheat bran group, wheat bran group or no fiber added group. The four test groups contained similar amount of carbohydrate vary from 52.9 g to 56.8 g. The results in this study showed that overall plasma glucose concentration was in the lowest level after the oat source fiber containing group compares combining groups with less oat fiber added or group without oat fiber. In the present study, the available carbohydrate in the three oat containing test meals is 124.71 g which includes 23 g (3 g dietary fiber, 20 g

starch) from 1 serving oat flakes. The possible explanation for the barely insignificant effect on postprandial plasma glucose and serum triglycerides concentration in the three oat flakes containing test meal should be further evaluated.

The different concentration of oat 1 serving (35 g) prepared by various amount of water was shown in Table 3.2. The medium amount of water (210ml) in the current study is recommended as the standard to prepare the oatmeal from the intervention commercial oat flake. In our preliminary study, except the standard water (210 ml) group, the oatmeal prepared by 169 ml and 335 ml water and 1 serving of oat flakes were in the most degree that can be accepted and palatable feed backed by the subjects. It was suggested that the concentration of the soluble fiber β-glucan in oat is one of the factors to modulate the glycemic and lipidemic control (Khoury et al., 2012). The amount of soluble fiber ranges from 2.3% to 8.5 % in oat groat (Butt et al., 2008). Even the different concentration of oat were induced by various amount of water in our study, however, it limited by the unknown amount and concentration of soluble fiber that we did not evaluate, future trails should assess insight of the dietary fiber also the related concentration of soluble fiber.

The constitution of high carbohydrate high fat meal in the three test meal was presented in Table 3.1. Forty nine percent of energy (823.68 kcal) came from the 101.71 carbohydrate, 46% of the energy contributed by the 41.9 g fat. This high carbohydrate and high fat characteristic in one meal was similar with the HCHF meal in a previous study (Ghanim et al., 2009) which contained 88g carbohydrate(41% of total energy) and 51 g fat (41% of total energy) from 910 kcal total energy compared to the intervention meal in their study riches in fruit and fiber from oatmeal. However, after add 139 kcal of energy from 1 serving oat (35 g) (contains 23 g carbohydrate, 3 g fat and 5 g protein) to the HCHF meal in our present study, total energy the participants consumed was 962.86 kcal, this energy content might be not proportionate enough as the breakfast for the young and healthy participants consumed in the current study.

The three intervention groups in current study were composed by HCHF meal, oat 1 serving with different amount of water to induce distinctive concentration of oatmeal. The three test groups were in the same nutrient profiles in energy, carbohydrate, protein, fat as well as dietary fiber. One source of weakness in this study which could affected the

observation was that the lack of control meal which should almost exactly in the same nutrient profile except the dietary fiber content from oat in the study design to interpret the results.

The glucose levels are likely to drop gradually in all three test meal in Hour 3, therefore the time interval of the blood collection in the first 2 hours in the present study might be relatively infrequent to be observed any marginally significant difference between the three groups.

The postprandial triglycerides concentration were still in the sharp rising trend in Hour 5 as observed in postprandial triglycerides level (Table 4.3; Figure 4.2) and incremental triglycerides concentration (Table 4.7; Figure 4.6), especially for the group with oat in 335 ml of water and HCHF meal which was in the highest incremental triglycerides concentration in Hour 5. Thereby the time points for blood collection might be necessary to last longer than 5 hours to warrant the improvement of high fiber oat flakes on postprandial triglycerides level with HCHF meal.

Some authors suggested that healthy characteristics of the participants can contribute to the insignificant effect of intervention on glucose response (Hallfrisch and Behall, 2000). It was speculated the glycemic lowering effects are less significant if subjects are young, fit and have normal glucose responses whereas effects on glycemic improvement are more likely to be observed in subjects whom are hypercholesterolemic, older, less slim and in noninsulin-dependent diabetes mellitus (NIDDM). The subjects in the present study are young participants with normal BMI, fasting lipid profile and blood glucose level, these healthy characteristics of the participants may be a contributor to the null hypothesis in the current study.

CHAPTER VI

SUMMARY and SUGGESTION

This current study was designed to evaluate 1 serving oat flakes (35 g) in various concentrations (prepared with 169, 210 and 335 ml water) on postprandial plasma glucose and serum triglycerides level induced hyperglycemia and hypertriglyceridemia by a high carbohydrate and high fat meal in healthy individuals. The following conclusion can be drawn from the present study: 1 serving (35 g) oatmeal in various concentration prepared by 3 amount of water did not induce significant effect on postprandial hyperglycemia and hypertriglyceridemia which induced by a high carbohydrate and high fat meal in all healthy subjects. The current research was limited by the low dose of dietary fiber in the intervention meals, various duration participants consumed the test meal, lack of control meal. It was not specifically designed to evaluate the size of the oat flakes which should be followed insight for future research. In addition, more frequent time intervals and longer experimental period for the blood sample collection are needed to warrant the results.

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APPENDICES

Appendix A

Individually difference and % difference of plasma glucose (mg/ dL) between fasting (H0) and Hour 1, Hour 3, Hour 5 with three test meal.

Subject	Test meal	Difference (H1to H0)	% Difference	Difference (H3to H0)	% Difference	Difference (H5to H0)	% Difference
Subject No.1	Oat 35g + HCHF + 169 ml H_20	9	10.47 %	-6	-6.98 %	-7	-8.14 %
	Oat 35g + HCHF + 210 ml H_20	42	49.41 %	50	58.82 %	-19	-22.35 %
	Oat 35g + HCHF + 335 ml H_20	43	51.81 %	3	3.61 %	-4	-4.82 %
Subject No.2	Oat 35g + HCHF + 169 ml H_20	46	58.23 %	14	17.72 %	9	11.39%
	Oat 35g + HCHF + 210 ml H_20	119	150.63 %	55	69.62 %	2	2.53 %
	Oat 35g + HCHF + 335 ml H_20	51	62.96 %	14	17.28 %	-19	-23.46 %
Subject No.3	Oat 35g + HCHF + 169 ml H_20	42	54.55 %	24	31.17%	4	5.19 %
	Oat 35g + HCHF + 210 ml H_20	40	47.62 %	11	13.10 %	-20	-23.81 %
	Oat 35g + HCHF + 335 ml H_20	26	31.70 %	-3	-3.66 %	-23	-28.05 %
Subject No.4	Oat 35g + HCHF + 169 ml H_20	54	66.67 %	16	19.75 %	-3	-3.70 %
	Oat 35g + HCHF + 210 ml H_20	85	98.84 %	17	19.77 %	-6	-6.98 %
	Oat 35g + HCHF + 335 ml H_20	24	27.27 %	25	28.41 %	-6	-6.82 %
Subject No.5	Oat 35g + HCHF + 169 ml H_20	44	55.00 %	1	1.25 %	-5	-6.25 %
	Oat 35g + HCHF + 210 ml H_20	59	78.67 %	12	16.00 %	5	6.67 %
	Oat 35g + HCHF + 335 ml H_20	104	128.40 %	14	17.28 %	9	11.11 %

Subject No.6	Oat 35g + HCHF + 169 ml H_20	11	12.64 %	-4	-4.60 %	-13	-14.94 %
	Oat 35g + HCHF + 210 ml H_20	48	53.33 %	11	12.22 %	-4	4.44 %
	Oat 35g + HCHF + 335 ml H_20	19	21.84 %	17	19.54 %	-6	-6.90 %
Subject No.7	Oat 35g + HCHF + 169 ml H_20	9	10.71 %	23	27.38 %	-8	-9.52%
	Oat 35g + HCHF + 210 ml H_20	-10	-12.05 %	-17	-20.48 %	-1	-1.20 %
	Oat 35g + HCHF + 335 ml H_20	-5	-5.10 %	4	4.49 %	-10	-11.24 %
Subject No.8	Oat 35g + HCHF + 169 ml H_20	127	178.87 %	52	73.24 %	-5	-7.04 %
	Oat 35g + HCHF + 210 ml H_20	98	125.64 %	45	57.69 %	-5	-6.41 %
	Oat 35g + HCHF + 335 ml H_20	81	106.58 %	58	76.32 %	-8	- 10.53 %
Subject No.9	Oat 35g + HCHF + 169 ml H_20	101	118.82 %	36	42.35 %	-14	-16.47 %
	Oat 35g + HCHF + 210 ml H_20	110	125.00 %	50	56.82 %	-26	-29.55 %
	Oat 35g + HCHF + 335 ml H_20	142	161.36 %	129	146.59 %	1	1.14 %
Subject No.10	Oat 35g + HCHF + 169 ml H_20	56	65.12 %	7	8.14 %	-10	-11.63 %
	Oat 35g + HCHF + 210 ml H_20	27	32.93 %	-1	-1.22 %	-16	19.51 %
	Oat 35g + HCHF + 335 ml H_20	30	34.09 %	-2	-2.27 %	-2	-2.27 %
Subject No.11	Oat 35g + HCHF + 169 ml H_20	-12	-12.63 %	-19	- 20 %	-1	-1.05 %
	Oat 35g + HCHF + 210 ml H_20	18	21.69 %	-5	-6.02 %	-1	-1.20 %
	Oat 35g + HCHF + 335 ml H_20	21	24.14 %	-6	-6.90 %	-2	- 2.30 %

Appendix B

Individually difference and % difference of serum triglycerides (mg/ dL) between fasting (H0) and Hour 1, Hour 3, Hour 5 with three test meal.

Subject	Test meal	Difference (H1to H0)	% Difference	Difference (H3to H0)	% Difference	Difference (H5to H0)	% Difference
Subject No.1	Oat 35g + HCHF + 169 ml H_20	-6	-9.23 %	3	4.62 %	10	15.38 %
	Oat 35g + HCHF + 210 ml H_20	-	-	-	-	-	-
	Oat 35g + HCHF + 335 ml H_20	0	0 %	12	22.64 %	13	24.53 %
Subject No.2	Oat 35g + HCHF + 169 ml H_20	1	1.64 %	2	3.28 %	23	37.70 %
	Oat 35g + HCHF + 210 ml H_20	-6	-9.68 %	23	37.10 %	-6	-9.68 %
	Oat 35g + HCHF + 335 ml H_20	5	7.58 %	-8	-12.12 %	4	6.06 %
Subject No.3	Oat 35g + HCHF + 169 ml H_20	4	4.88 %	-5	-6.10 %	-5	-6.10 %
	Oat 35g + HCHF + 210 ml H_20	9	15.00 %	-11	-18.33 %	5	8.33 %
	Oat 35g + HCHF + 335 ml H_20	12	16.67 %	46	63.89 %	23	31.94 %
Subject No.4	Oat 35g + HCHF + 169 ml H_20	-24	-38.71 %	-2	-3.23 %	-6	-9.68 %
	Oat 35g + HCHF + 210 ml H_20	-11	-18.97 %	-7	-12.07 %	23	39.66 %
	Oat 35g + HCHF + 335 ml H_20	0	0 %	18	42.86 %	29	69.05 %
Subject No.5	Oat 35g + HCHF + 169 ml H_20	-22	-41.51 %	12	22.64 %	22	41.51 %
	Oat 35g + HCHF + 210 ml H_20	11	21.15 %	34	65.38 %	31	59.62 %
	Oat 35g + HCHF + 335 ml H_20	-	-	19	38.00 %	63	126.00 %
Subject No.6	Oat 35g + HCHF + 169 ml H_20	-20	-39.22 %	19	37.25 %	10	19.61 %

	Oat 35g + HCHF + 210 ml H_20	-6	-11.76 %	-1	-1.96 %	30	58.82 %
	Oat 35g + HCHF + 335 ml H_20	11	25.00 %	-3	-6.82%	53	120.45 %
Subject No.7	Oat 35g + HCHF + 169 ml H_20	21	53.85 %	13	33.33 %	16	41.03 %
	Oat 35g + HCHF + 210 ml H_20	-1	-1.41 %	6	8.45 %	20	28.17 %
	Oat 35g + HCHF + 335 ml H_20	-	-	-	-	-	-
Subject No.8	Oat 35g + HCHF + 169 ml H_20	13	24.07 %	25	46.30 %	57	105.56 %
	Oat 35g + HCHF + 210 ml H_20	9	20.00 %	3	6.67 %	21	46.67 %
	Oat 35g + HCHF + 335 ml H_20	0	0 %	4	9.76 %	35	85.37 %
Subject No.9	Oat 35g + HCHF + 169 ml H_20	19	38.78 %	30	61.22 %	55	112.24 %
	Oat 35g + HCHF + 210 ml H_20	26	54.17 %	25	52.08 %	55	114.58 %
	Oat 35g + HCHF + 335 ml H_20	24	52.17 %	11	23.91 %	30	65.22 %
Subject No.10	Oat 35g + HCHF + 169 ml H_20	-1	-2.17 %	0	0 %	-1	-2.17 %
	Oat 35g + HCHF + 210 ml H_20	-	-	-	-	-	-
	Oat 35g + HCHF + 335 ml H_20	-	-	-	-	-	-
Subject No.11	Oat 35g + HCHF + 169 ml H_20	26	28.89 %	65	72.22 %	74	82.22 %
	Oat 35g + HCHF + 210 ml H_20	26	40.63 %	48	75.00 %	30	46.88
	Oat 35g + HCHF + 335 ml H_20	24	33.33 %	75	104.17 %	29	40.28 %

-: Excluded data with abnormal triglycerides.

Appendix C

Approval ethic review of the current experimental.

AF 01-12

คณะกรรมการพิจารณาจริยธรรมการวิจัยในคน กลุ่มสหสถาบัน ชุดที่ 1 จุฬาลงกรณ์มหาวิทยาลัย
อาคารสถาบัน 2 ชั้น 4 ซอยจุฬาลงกรณ์ 62 ถนนพญาไท เขตปทุมวัน กรุงเทพฯ 10330
โทรศัพท์: 0-2218-8147 โทรสาร: 0-2218-8147 E-mail: eccu@chula.ac.th

COA No. 176/2555

ใบรับรองโครงการวิจัย

โครงการวิจัยที่ 134.1/55 : การศึกษาผลของ OAT β – glucan ที่ความหนืดต่างกันต่อระดับน้ำตาลและคลอเลสเตอรอลในอาสาสมัครที่มีสุขภาพดี

ผู้วิจัยหลัก : อาจารย์ ดร.สุวิมล ทรัพย์วโรบล

หน่วยงาน : คณะสหเวชศาสตร์ จุฬาลงกรณ์มหาวิทยาลัย

คณะกรรมการพิจารณาจริยธรรมการวิจัยในคน กลุ่มสหสถาบัน ชุดที่ 1 จุฬาลงกรณ์มหาวิทยาลัย ได้พิจารณา โดยใช้หลัก ของ The International Conference on Harmonization – Good Clinical Practice (ICH-GCP) อนุมัติให้ดำเนินการศึกษาวิจัยเรื่องดังกล่าวได้

ลงนาม..... ลงนาม.....
(รองศาสตราจารย์ นายแพทย์ปรีดา ทัศนประดิษฐ) (ผู้ช่วยศาสตราจารย์ ดร.นันทรี ชัยชนะวงศาโรจน์)
ประธาน กรรมการและเลขานุการ

วันที่รับรอง : 6 ธันวาคม 2555 **วันหมดอายุ** : 5 ธันวาคม 2556

เอกสารที่คณะกรรมการรับรอง
1) โครงการวิจัย
2) ข้อมูลสำหรับกลุ่มประชากรหรือผู้มีส่วนร่วมในการวิจัยและใบยินยอมของกลุ่มประชากรหรือผู้มีส่วนร่วมในการวิจัย
3) ผู้วิจัย
4) ใบประชาสัมพันธ์

เลขที่โครงการวิจัย 134.1/55
วันที่รับรอง -6 ธ.ค. 2555
วันหมดอายุ -5 ธ.ค. 2556

เงื่อนไข
1. ขั้นตอนการรับรองโครงการวิจัยนี้เป็นการพิจารณาจริยธรรม หากต้องมีการเก็บข้อมูลการวิจัยก่อนได้รับการอนุมัติจากคณะกรรมการพิจารณาจริยธรรมการวิจัย
2. หากใบรับรองโครงการวิจัยหมดอายุ การดำเนินการวิจัยต้องหยุด เว้นแต่ได้ยื่นขอต่ออายุและได้รับอนุมัติก่อนล่วงหน้าไม่ต่ำกว่า 1 เดือน พร้อมส่งรายงานความก้าวหน้าการวิจัย
3. ต้องดำเนินการวิจัยตามที่ระบุไว้ในโครงการวิจัยอย่างเคร่งครัด
4. ใช้เอกสารข้อมูลสำหรับกลุ่มประชากรหรือผู้มีส่วนร่วมในการวิจัย ใบยินยอมของกลุ่มประชากรหรือผู้มีส่วนร่วมในการวิจัย และเอกสารอื่นๆ ที่เกี่ยวข้อง (ถ้ามี) เฉพาะที่ประทับตรารับรองโดยคณะกรรมการฯ เท่านั้น
5. หากเกิดเหตุการณ์ไม่พึงประสงค์ที่รุนแรงในมนุษย์ที่เกี่ยวข้องกับการวิจัย ต้องรายงานต่อคณะกรรมการฯ ภายใน 5 วันทำการ
6. หากมีการเปลี่ยนแปลงการดำเนินการวิจัย ให้ส่งคณะกรรมการพิจารณาจริยธรรมการวิจัยรับรองก่อนดำเนินการ
7. โครงการวิจัยไม่เกิน 1 ปี ส่งแบบรายงานสิ้นสุดโครงการวิจัย (AF 03-12) และรายงานความก้าวหน้าการวิจัยภายใน 30 วัน เมื่อโครงการวิจัยเสร็จสิ้น สำหรับโครงการวิจัยที่เป็นวิทยานิพนธ์ให้ส่งบทคัดย่อการวิจัย ภายใน 30 วัน เมื่อโครงการวิจัยเสร็จสิ้น

AF 05-07

หนังสือแสดงความยินยอมเข้าร่วมการวิจัย

ทำที่.....

วันที่.....เดือน.....พ.ศ. .....

เรียน ประชากรตัวอย่างหรือผู้มีส่วนร่วมในการวิจัย.....

ข้าพเจ้า ซึ่งได้ลงนามท้ายหนังสือนี้ ขอแสดงความยินยอมเข้าร่วมโครงการวิจัย

ชื่อโครงการวิจัย การศึกษาผลของ oat β – glucan ที่ความหนืดต่างกันต่อระดับน้ำตาลและคลอเลสเตอรอลในอาสาสมัครที่มีสุขภาพดี

ชื่อผู้วิจัย อ.ดร.สุวิมล ทรัพย์วโรบล ตำแหน่ง หัวหน้าภาควิชาโภชนาการและการกำหนดอาหาร
ภาควิชาโภชนาการและการกำหนดอาหาร คณะสหเวชศาสตร์ จุฬาลงกรณ์มหาวิทยาลัย
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ข้าพเจ้า **ได้รับทราบรายละเอียดเกี่ยวกับที่มาและวัตถุประสงค์ในการทำวิจัย** รายละเอียดขั้นตอนต่างๆ ที่จะต้องปฏิบัติหรือได้รับการปฏิบัติ ความเสี่ยง/อันตราย และประโยชน์ซึ่งจะเกิดขึ้นจากการวิจัยเรื่องนี้ โดยได้อ่านรายละเอียดในเอกสารชี้แจงผู้เข้าร่วมการวิจัย โดยตลอด และได้รับคำอธิบายจากผู้วิจัย **จนเข้าใจเป็นอย่างดีแล้ว**

ข้าพเจ้าจึงสมัครใจเข้าร่วมในโครงการวิจัยนี้ ตามที่ระบุไว้ในเอกสารชี้แจงผู้เข้าร่วมการวิจัย โดยข้าพเจ้ายินยอมให้เจาะเลือดตามรายละเอียด ดังนี้ ในการศึกษาที่ 1 เวลาก่อนและหลังดื่มเครื่องดื่มกลูโคส 15, 30, 60, 90, 120 นาทีผ่านทางสายที่ต่อค้างไว้ ภายใต้การดูแลของพยาบาลวิชาชีพ เก็บเลือดครั้งละ 1 ช้อนชา และข้าพเจ้าจะเข้าร่วมจำนวนทั้งสิ้น 3 ครั้ง และการศึกษาที่ 2 ก่อนและหลังรับประทานอาหารไขมันสูง 1, 2, 3, 4, 5 ชั่วโมงผ่านทางสายที่ต่อค้างไว้ เก็บเลือดครั้งละ 1 ช้อนชา และข้าพเจ้าจะเข้าร่วมจำนวนทั้งสิ้น 3 ครั้ง *(รวมเป็นทั้งสิ้น 6 ครั้งตลอดระยะเวลาการศึกษา)* เมื่อเสร็จสิ้นการวิจัยแล้วข้อมูลที่เกี่ยวข้องกับผู้มีส่วนร่วมในการวิจัยจะถูกทำลาย เลือดที่นำไปใช้แล้ว จะถูกทำลายทิ้งตามกระบวนการทางวิทยาศาสตร์

ข้าพเจ้ามีสิทธิถอนตัวออกจากการวิจัยเมื่อใดก็ได้ตามความประสงค์ **โดยไม่ต้องแจ้งเหตุผล** ซึ่งการถอนตัวออกจากการวิจัยนั้นจะไม่มีผลกระทบผลต่อการเรียนหรือผลกระทบอื่นใดต่อข้าพเจ้าทั้งสิ้น

ข้าพเจ้าได้รับคำรับรองว่า ผู้วิจัยจะปฏิบัติต่อข้าพเจ้าตามข้อมูลที่ระบุไว้ในเอกสารชี้แจงผู้เข้าร่วมการวิจัย และข้อมูลใดๆ ที่เกี่ยวข้องกับข้าพเจ้า ผู้วิจัยจะเก็บรักษาเป็นความลับ โดยจะนำเสนอข้อมูลการวิจัยเป็นภาพรวมเท่านั้น ไม่มีข้อมูลใดในการรายงานที่จะนำไปสู่การระบุตัวข้าพเจ้า

หากข้าพเจ้าไม่ได้รับการปฏิบัติตรงตามที่ได้ระบุไว้ในเอกสารชี้แจงผู้เข้าร่วมการวิจัย ข้าพเจ้าสามารถร้องเรียนได้ที่ คณะกรรมการพิจารณาจริยธรรมการวิจัยในคน กลุ่มสหสถาบัน ชุดที่ 1 จุฬาลงกรณ์มหาวิทยาลัย ชั้น 4 อาคารสถาบัน 2 ซอย จุฬาลงกรณ์ 62 ถนนพญาไท เขตปทุมวัน กรุงเทพฯ 10330 โทรศัพท์ 0-2218-8147, 0-2218-8141 โทรสาร 0-2218-8147 E-mail: eccu@chula.ac.th

ข้าพเจ้าได้ลงลายมือชื่อไว้เป็นสำคัญต่อหน้าพยาน ทั้งนี้ข้าพเจ้าได้รับสำเนาเอกสารชี้แจงผู้เข้าร่วมการวิจัย และสำเนาหนังสือแสดงความยินยอมไว้แล้ว

ลงชื่อ.....
(.....)
ผู้วิจัยหลัก

ลงชื่อ.....
(.....)
ผู้มีส่วนร่วมในการวิจัย

ลงชื่อ.....
(.....)
พยาน

เลขที่โครงการวิจัย 134.1/55
วันที่รับรอง -6 S.A. 2555
วันหมดอายุ -5 S.A. 2556

ประชาสัมพันธ์

ภาควิชาโภชนาการและการกำหนดอาหาร คณะสหเวชศาสตร์

รับสมัคร อาสาสมัครเข้าร่วมงานวิจัย

เรื่อง การศึกษาผลของ oat β – glucan ที่ความหนืดต่างกันต่อระดับน้ำตาล และคลอเลสเตอรอลในอาสาสมัครที่มีสุขภาพดี

หากท่านมีคุณสมบัติ ดังต่อไปนี้

1. มีอายุระหว่าง 18 - 50 ปี สุขภาพดี
2. ดัชนีมวลกายอยู่ในช่วงปกติ (18.5 – 22.9)
3. ไม่มีโรคประจำตัวไม่มีประวัติการใช้ยาที่มีผลต่อน้ำตาลและระดับคลอเลสเตอรอลในเลือดอย่างน้อย 3 เดือนก่อนการศึกษา
4. ไม่สูบบุหรี่และไม่ดื่มเครื่องดื่มแอลกอฮอล์
5. ไม่มีประวัติการเจ็บป่วยใดๆ ก่อนเข้าร่วมการวิจัยอย่างน้อย 3 เดือน

สนใจสอบถามรายละเอียดเพิ่มเติมได้ที่

อ.ดร.สุวิมล ทรัพย์วโรบล ภาควิชาโภชนาการและการกำหนดอาหาร คณะสหเวชศาสตร์ โทร. 02 218 1116 ต่อ 24

หมายเหตุ การศึกษาครั้งนี้จะรับเฉพาะ อาสาสมัครที่อ่านออก เขียนได้ และสามารถสื่อสารเป็นภาษาไทยได้เท่านั้น เพื่อประโยชน์ในการสื่อสาร ไม่รับอาสาสมัครที่เป็นกลุ่มผู้ด้อยโอกาส/เปราะบาง ผู้ป่วยโรคจิต ผู้ต้องขัง เด็กอายุต่ำกว่า 18 ปี สตรีมีครรภ์ ผู้สูงอายุที่มีความจำบกพร่อง ผู้ป่วยสมองเสื่อม คนพิการ ชนกลุ่มน้อยผู้ย้ายถิ่น/อพยพ ปัญญาอ่อน ทหารเกณฑ์ และผู้ป่วยอาการหนักไม่สามารถให้คำยินยอมด้วยตนเองได้

เลขที่โครงการวิจัย 134.1/55

วันที่รับรอง - 6 S.A. 2555

วันหมดอายุ - 5 S.A. 2556

บันทึกข้อความ

ส่วนงาน คณะกรรมการพิจารณาจริยธรรมการวิจัยในคน กลุ่มสหสถาบัน ชุดที่ 1 ไทร.0-2218-8147

ที่ จว 748/55 **วันที่** 11 ธันวาคม 2555

เรื่อง แจ้งผลผ่านการพิจารณาจริยธรรมการวิจัย

เรียน คณบดีคณะสหเวชศาสตร์

สิ่งที่ส่งมาด้วย 1. ใบรับรองผลการพิจารณา
2. เอกสารข้อมูลสำหรับผู้มีส่วนร่วมในการวิจัย
3. หนังสือแสดงความยินยอม
4. ใบประชาสัมพันธ์

ตามที่ อาจารย์ ดร.สุวิมล ทรัพย์วโรบล อาจารย์ คณะสหเวชศาสตร์ จุฬาลงกรณ์มหาวิทยาลัย ได้เสนอโครงการวิจัยที่ 134.1/55 เรื่อง การศึกษาผลของ OAT β – glucan ที่ความหนืดต่างกันต่อระดับน้ำตาลและคลอเลสเตอรอลในอาสาสมัครที่มีสุขภาพดี (EFFECTS OF DIFFERENT VISCOSITY OF OAT β – glucan ON BLOOD GLUCOSE AND CHOLESTEROL IN HEALTHY INDIVIDUALS) เพื่อให้กรรมการผู้ทบทวนหลักพิจารณาจริยธรรมการวิจัยความละเอียดแจ้งแล้วนั้น

การนี้ กรรมการผู้ทบทวนหลัก ได้เห็นสมควรให้ผ่านการพิจารณาจริยธรรมการวิจัยได้ รับรองวันที่ 6 ธันวาคม 2555

จึงเรียนมาเพื่อโปรดทราบ

(ผู้ช่วยศาสตราจารย์ ดร.บันทริ ชัยชนะวงศ์โรจน์)
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กลุ่มสหสถาบัน ชุดที่ 1 จุฬาลงกรณ์มหาวิทยาลัย

เรียน คณบดีคณะสหเวชศาสตร์
เพื่อโปรดทราบผลการพิจารณาของ
คณะกรรมการพิจารณาจริยธรรมการวิจัยในคน
ขอแสดงความนับถือ
ผศ.ดร.สุวิมล ทรัพย์วโรบล
18 ธ.ค. 55

BIOGRAPHY

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Bachelor Degree of Traditional Chinese Medicine (2011), Faculty of Traditional Chinese Medicine, Macau University of Science and Technology, Macau, The People's Republic of China.

Scholarships received:

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