ASSOCIATION BETWEEN LIFESTYLE FACTORS AND METABOLIC SYNDROME AMONG POPULATION OF BANPHAEO, SAMUTSAKORN THAILAND, 2008

NITIKORN PHOOSUWAN

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N. Phoosuwan
.....
Mr. Nitikorn Phoosuwan
Candidate

Kulaya Narksawat
.....
Assoc. Prof. Kulaya Narksawat,
D.D.S., M.P.H., M.Sc.
Major advisor

M. Pradipasen
.....
Assoc. Prof. Mandhana Pradipasen,
Dr.P.H. (Epidemiology)
Co-advisor

Rewadee Chongsuwat
.....
Asst. Prof. Rewadee Chongsuwat,
Ph.D. (Food, Nutrition, and Dietetics)
Co-advisor

B. Mahaisavariya
.....
Prof. Banchong Mahaisavariya,
M.D., Dip Thai Board of Orthopedics
Dean
Faculty of Graduate Studies
Mahidol University

Dusit Sij
.....
Assoc. Prof. Dusit Sujirarat,
M.Sc. (Biostatistics)
Program Director
Master of Science (Public Health)
Major in Infectious Diseases and
Epidemiology
Faculty of Public Health
Mahidol University

Thesis
entitled

ASSOCIATION BETWEEN LIFESTYLE FACTORS AND METABOLIC SYNDROME AMONG POPULATION OF BANPHAEO, SAMUTSAKORN THAILAND, 2008

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Major in Infectious Diseases and Epidemiology
on
April 11, 2011

N. Phoosuwan
.....
Mr. Nitikorn Phoosuwan
Candidate

P. Panmat
.....
Mr.Panuwat Panket,
M.D., Ph.D. (Public Administration)
Chair

Kulaya Narksawat
.....
Assoc. Prof. Kulaya Narksawat,
D.D.S., M.P.H., M.Sc.
Member

M. Pradya
.....
Assoc. Prof. Mandhana Pradipasen,
Dr.P.H. (Epidemiology)
Member

Rewadee Chongsuwat
.....
Asst. Prof. Rewadee Chongsuwat,
Ph.D. (Food, Nutrition, and Dietetics)
Member

B. Maha
.....
Prof. Banchong Mahaisavariya,
M.D., Dip Thai Board of Orthopedics
Dean
Faculty of Graduate Studies
Mahidol University

.....
Assoc. Prof. Phitaya Charupoonphol,
M.D., Dip.Thai Board of Epidemiology
Dean
Faculty of Public Health
Mahidol University

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Nitikorn Phoosuwan

ASSOCIATION BETWEEN LIFESTYLE FACTORS AND METABOLIC SYNDROME AMONG POPULATION OF BANPHAEO DISTRICT, SAMUTSAKORN THAILAND, 2008

NITIKORN PHOOSUWAN 5037152 PHPH/M

M.Sc. (PUBLIC HEALTH) MAJOR IN INFECTIOUS DISEASES AND EPIDEMIOLOGY

THESIS ADVISORY COMMITTEE: KULAYA NARKSAWAT D.D.S., M.P.H., M.Sc., MANDHANA PRADIPASEN, Dr.P.H., REWADEE CHONGSUWAT, Ph.D.(FOOD, NUTRITION, AND DIETETICS)

ABSTRACT

A population based case-control study was conducted to assess the association between lifestyle and metabolic syndrome in Banphaeo District, Samutsakorn Province, Thailand. The study population consisted of cases who had metabolic syndrome and the controls were those who did not have metabolic syndrome; using the definition of the third report of National Cholesterol Education Program (NCEP). Both cases and controls were participants of the Banphaeo Routine Hypertension and Diabetes Screening Program (BRHDSP) between November 2007 and June 2008. Data were collected by home visits to conduct interviews regarding lifestyles over the past 1-2 years, including physical activities from occupation and exercise, eating habits, smoking and alcohol drinking habits, and mental health.

Results from multivariate analysis, stratified by gender, found significant lifestyle differences between male participants who undertook physical activity and those who had sedentary activity occupations (OR 2.82, 95% CI 1.22-6.55), together with a high intake of seafood (squid, shrimp, shells, excluding fish) (OR 4.82, 95% CI 1.36-17.15). Among female participants, there were no significant lifestyle differences but when their age increased by one year, they were more likely to have at least three out of the five criteria for metabolic syndrome (OR 1.05, 95%CI 1.01-1.10).

Results from the study suggest the risk factors are found in both men and women. Strengthening the existing health promotion programs for the modification of lifestyles can help to reduce the risks of developing metabolic syndrome and future cerebrovascular and heart diseases.

KEY WORDS : METABOLIC SYNDROME / RISK FACTOR / LIFESTYLE

89 pages

ปัจจัยทางวิถีชีวิตที่มีผลต่อการเกิดโรคเมตาบอลิกซินโดรมในประชาชนอำเภอบ้านแพ้ว จังหวัดสมุทรสาคร

ASSOCIATION BETWEEN LIFESTYLE FACTORS AND METABOLIC SYNDROME AMONG POPULATION OF BANPHAEO DISTRICT, SAMUTSAKORN THAILAND, 2008

นิติกร ภู่สุวรรณ 5037152 PHPH/M

วท.ม. (สาธารณสุขศาสตร์) สาขาวิชาเอกโรคติดเชื้อและวิทยาการระบาด

คณะกรรมการที่ปรึกษาวิทยานิพนธ์ : กุลยา นาคสวัสดิ์, M.Sc.(MEDICAL EPIDEMIOLOGY), มันทนา ประทีปะเสน, Dr.P.H.(EPIDEMIOLOGY), เรวดี จงสุวัฒน์, Ph.D. (FOOD, NUTRITION, AND DIETETICS)

บทคัดย่อ

การศึกษาเชิงสังเกตแบบเคสคอนโทลในชุมชน มีวัตถุประสงค์เพื่อศึกษาปัจจัยทางวิถีชีวิตและปัจจัยอื่นที่มีผลต่อโรคเมตาบอลิกซินโดรมในประชาชนอำเภอบ้านแพ้ว จังหวัดสมุทรสาคร การศึกษาใช้เกณฑ์โรคเมตาบอลิกซินโดรมจากรายงานฉบับที่ 3 ของ NCEP โดยกลุ่มประชากรศึกษาประกอบด้วยกลุ่มผู้ป่วยด้วยโรคเมตาบอลิกซินโดรมจำนวน 196 ราย และกลุ่มเปรียบเทียบที่ไม่มีโรคเมตาบอลิกซินโดรมจำนวน 212 ราย ที่มีอายุ 35 ถึง 60 ปี ที่เข้าร่วมโครงการคัดกรองโรคความดันโลหิตสูงและโรคเบาหวานที่ดำเนินการโดยกระทรวงสาธารณสุขในเดือนพฤศจิกายน 2550 ถึงเดือนมิถุนายน 2551 เก็บข้อมูลด้วยการเยี่ยมบ้านและโดยสัมภาษณ์ปัจจัยทางวิถีชีวิตในช่วง 1-2 ปีที่ผ่านมา ซึ่งได้แก่ ลักษณะของกิจกรรมทางกายจากการประกอบอาชีพ และการออกกำลังกาย พฤติกรรมบริโภคอาหาร การดื่มแอลกอฮอล์ การสูบบุหรี่ และสุขภาพจิต

ผลการวิเคราะห์ความสัมพันธ์เชิงซ้อนโดยการแยกเพศ พบว่าในเพศชายปัจจัยทางวิถีชีวิตหรือพฤติกรรมที่มีนัยสำคัญทางสถิติ ได้แก่ การมีอาชีพที่ส่วนใหญ่ของเวลาการทำงานไม่ได้เคลื่อนไหวหรือใช้แรงงาน (OR 2.82, 95%CI 1.22-6.55) และการบริโภคอาหารทะเลได้แก่ ปลาหมึก กุ้ง และหอย ไม่รวมปลา (OR 4.82, 95%CI 1.36-17.15) ในเพศหญิงไม่พบปัจจัยทางวิถีชีวิตที่มีนัยสำคัญทางสถิติ แต่พบว่าโอกาสที่เพศหญิงจะมีอาการ 3 ใน 5 ของเมตาบอลิกซินโดรมจะเพิ่มขึ้นทุกปีที่อายุเพิ่มขึ้นหนึ่งปี (OR 1.05, 95%CI 1.01-1.10)

ผลการศึกษาชี้ให้เห็นถึงปัจจัยเสี่ยงต่อการเกิดโรคอ้วนลงพุงในเพศชาย การพัฒนาและปรับปรุงกิจกรรมสร้างเสริมสุขภาพที่มีอยู่ เช่น การออกกำลังกาย จะสามารถลดอุบัติการณ์การเกิดโรคอ้วนลงพุงที่สามารถนำไปสู่การเกิดโรคหลอดเลือดสมองและโรคหลอดเลือดหัวใจได้

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CHAPTER I
INTRODUCTION

1. Rational and Background

The Metabolic syndrome (MetS) is comprised of dyslipidemia, elevated blood pressure, elevated plasma glucose, abdominal obesity and associated prothrombotic and proinflammatory states. It is accompanied by a life long twofold increased risk of cardiovascular disease and a fivefold increase risk for diabetes [1]. **The metabolic syndrome**, a collection of unhealthy body measurements and abnormal laboratory test results, may identify persons at high risk for developing cardiovascular disease. Aggressive lifestyle modification and possible use of medications to treat the conditions that make up the metabolic syndrome may reduce a person's chances of developing heart disease or stroke [2]. The metabolic syndrome has also been called syndrome X or insulin resistance syndrome [3].

The National Cholesterol Education Program's (NCEP) Adult Treatment Panel (ATP) III provided a working definition and drew the attention to metabolic syndrome [4] and proposed a working criterion for the metabolic syndrome, which addresses obesity, dyslipidemia, hyperglycemia and hypertension[5]. The metabolic syndrome was defined as the presence of at least three out of five of the following components [6]: and adapt for Asian; waist circumference ≥ 90 cm in men or ≥ 80 cm in women, serum triglyceride concentration ≥ 150 mg/dl (or on drug treatment for elevated triglycerides), serum high density lipoprotein (HDL) concentration < 40 mg/dl in men and <50 mg/dl in women or taking drug treatment for controlling low density lipoprotein (LDL-C), systolic blood pressure ≥ 130 mmHg and/or diastolic blood pressure ≥ 85 mmHg (or on antihypertensive drug) and fasting plasma glucose ≥ 100 mg/dL or taking drug treatment for controlling blood glucose).

The dominant underlying risk factors for this syndrome appear to be abdominal obesity and insulin resistance. Insulin resistance is a generalized metabolic disorder, in which the body can't use insulin efficiently. This is why the metabolic

syndrome is also called the insulin resistance syndrome. Other conditions associated with the syndrome include physical inactivity factor, aging factor, hormonal imbalance and genetic predisposition. Some people are genetically predisposed to insulin resistance. Acquired factors, such as excess body fat and physical inactivity, can elicit insulin resistance and the metabolic syndrome in these people. Most people with insulin resistance have abdominal obesity. The biologic mechanisms at the molecular level between insulin resistance and metabolic risk factors aren't fully understood and appear to be complex[7].

Lifestyle, modifiable risk factors, is always mentioned as the important risk factors of hypertension. Life style is a set of habits and customs that is influenced, modified encouraged, or constrained by the life long process of socialization. These habits and customs included use of substance such as alcohol, tobacco, tea, coffee, dietary habits, exercise, which have important implications for health and are often the subject of epidemiologic investigation[8]. Most components of metabolic syndrome are related separately to lifestyle factors such as weight control [9], dietary habit [9-14], and physical inactivity[15, 16] .

In Thailand, prevalence of hypertension, diabetes, overweight and obesity were 22.0% (95% (CI) = 20.5-23.6), 6.7% (6.0% in men and 7.4% in women), 28.3% and 6.8% respectively. While the prevalence of metabolic syndrome in rural Thai people, in north-eastern of Thailand (Khonkaen) were 23%, 15% (15.3% Men, 14.6% women) and in Bangkok was 12.8% (15.7% men, 11.7% women) respectively [17-22].

Not only prevalence of metabolic syndrome is high, but the prevalence of component of metabolic syndrome as hypertriglyceridemia (> 150 mg/dL), large waist circumference and low HDL-C (< 40 mg/dL) were also high in 40 %, 43.5% and 14 %, respectively.[23, 24]

Metabolic syndrome becomes an important problem in rural Thai populations who even lives with basic lifestyle in the non-urbanized and non-industrialized areas.[20]

For the lifestyle factors, prevalence of unhealthy dietary habit was 30.5%, physical inactivity was 49.6% (47.9% male and 55.6% female). In Thailand, prevalence of smoking was 19.5% (37.2% in male and 2.1% in female), prevalence of alcohol consumption was 29.3% and prevalence of having stress was 47.4% [25-28]

Samutsakorn province located in central region of Thailand with 36 kilometers from Bangkok. Banphaeo district is one among 3 districts of Samutsakorn province. Total population was 91,363 (2008) with population density of 373 per square meters. Banphaeo district consists of 12 sub districts with 97 villages. Common occupations among population are agriculture and fisheries.

In 2007, Samutsakorn had morbidity of cardiovascular diseases in 315.78 per 1000 population, while prevalence of hypertension and diabetes in Banphaeo district were 6.78 % and 2.73% respectively[29].

The aim of this study was to identify the association between MetS and via lifestyle factors of population in Banphaeo district, Samutsakorn province, Thailand. The results from this study will be used for providing specific health promotion activities for this group of population in an attempt to reduce incidence of metabolic syndrome which can lead to some important chronic diseases such as stroke, diabetes mellitus.

2. Objectives of the study

2.1 General Objectives

To assess the associations between metabolic syndrome and lifestyle factors among the community people of Banphaeo district, Samutsakorn province, Thailand.

2.2 Specific objectives

2.2.1 To assess the association between dietary habits and metabolic syndrome among the study population in Banphaeo district, Samutsakorn province, Thailand.

2.2.2 To assess the association between physical activity and metabolic syndrome among the study population in Banphaeo district, Samutsakorn province, Thailand.

2.2.3 To assess the association between smoking and metabolic syndrome among the study population in Banphaeo district, Samutsakorn province, Thailand.

2.2.4 To assess the association between alcohol consumption, and metabolic syndrome among the study population in Banphaeo district, Samutsakorn province, Thailand.

2.2.5 To assess the association between type A behavioral and metabolic syndrome among the study population in Banphaeo district, Samutsakorn province, Thailand.

2.2.6 To assess the association between mental health and metabolic syndrome among the study population in Banphaeo district, Samutsakorn province, Thailand.

3. Operational definitions

Metabolic syndrome referred to a person who met 3 or more of 5 components of metabolic syndrome; there were waist circumference ≥ 90 cm in men or ≥ 80 cm in women, serum triglyceride concentration ≥ 150 mg/dl (or on drug treatment for elevated triglycerides), serum HDL concentration < 40 mg/dl in men and <50 mg/dl in women (or on drug treatment for reduced HDL-C), systolic blood pressure ≥ 130 mmHg and/or diastolic blood pressure ≥ 85 mmHg (or on antihypertensive drug) and Fasting Plasma Glucose ≥ 100 mg/dL (or on drug treatment for elevated glucose).[6]

High waist circumference referred to a person who had waist circumference ≥ 90 cms in male or female ≥ 80 cms. Waist circumference was measured by a non elastic tape measure at umbilicus point with the subject standing.

High serum triglyceride referred to a person who had blood plasma triglyceride level in fasting ≥ 150 mg/dl (≥ 1.7 mmol/l**).** Triglyceride was collected from blood samples which drawn from antecubital vein into tube containing EDTA and determined by enzymatic method.[30]

Low serum HDL-C referred to a person who had blood plasma high density lipoprotein after 12 hours fasting <40 mg/dl (<1.03 mmol/l) in male or 50 mg/dl (<1.29 mmol/l) in female. HDL-C was collected from blood samples drawn from antecubital vein into tube containing EDTA and measured after dextran-magnesium precipitation.[31]

Hypertension referred to a person who had blood pressure ≥ 130/85 mmHg or taking antihypertensive drugs. Blood pressure measured by using an automatic (arm cuff) blood pressure monitor (Omron, Vernon Hills, IL) after 5 minutes resting.

High fasting plasma glucose referred to a person who had blood plasma after 12 hours fasting ≥ 100 mg/dl or taking antidiabetic drugs. Blood samples were drawn from antecubital vein into tube containing a serum separator gel and assessed with a hexokinase2glucose-6-phosphate dehydrogenase method.

Dietary habits referred to dietary pattern of participant during previous year collected by using questionnaire based on a validated quantitative food frequency questionnaire (FFQ) which were reported by types of food intake such as fruit and vegetable, meat, seafood, fish, milk, desserts, drinks, starch and carbohydrate. There were two levels of intake [32];

- low intake referred to frequency of eating was less than or equal to 3 times per week.

- high intake referred to frequency of eating more than 3 times per week.

Physical activity referred to any bodily movement produced by skeletal muscles that result in an expenditure of energy and divided to 3 types of physical activity [33] ;

Occupational physical activity: is completed regularly as part of one's job. It includes activities such as walking, hauling, lifting, pushing, carpentry, shoveling, and packing boxes.

Leisure-time physical activity or exercise: is the physical activity that is planned or structured. It involves repetitive bodily movement done to improve or maintain one or more of the components of physical fitness—cardiorespiratory endurance (aerobic fitness), muscular strength, muscular endurance, flexibility, and body composition[33].

Cigarette Smoking referred to pattern of smoking cigarette by types of cigarette, frequency of smoking, amount of cigarette smoking per day and duration of smoking.

Alcohol consumption referred to pattern of alcohol drinking by types of alcohol used, frequency of drinking, amount of drinking and duration of drinking.

Non drinkers: person who never drank alcoholic beverages in their lives.

Ex-smoker: person who drank previously but had already stopped drinking for certain period prior to the study.

Current drinker: person who drank alcohol beverages when they participated in social activity and/or who drank alcoholic beverage regularly.

Type A Behavior referred to behavior that satisfies score of standard questionnaire for type A behavior.

Mental health referred to mental status defined on stress and measured using Suanprung stress test (SPST-20) which classified as mild, moderate high and severe stress[34].

Age referred to age in years of study population at the time of measurement.

Having history of family members with some diseases referred to 1st generation of family members have diabetes mellitus, hypertension or hypertriglyceridemia.

Level of education referred to : highest educational level of participant classified as[35];

No education for a person who had not attended any formal school.

Primary school for a person who had finished 6 year elementary school.

Pre high school for a person who had finished 3 years in high school.

High school for a person who had finished grade 12 or finished vocational college.

Undergraduate school for a person who had finished a bachelor degree from university or college.

Graduate school for a person who had finished a master degree and higher.

Income referred to: total income of participant in each month reported by participant without no expenses exclusion. It was divided into 4 categories; no income, ≤5,000 baht, 5,001-10,000 baht and ≥10,001 baht.

Menopausal status was defined as amenorrhea for 12 months following the final menstrual period. It was defined as a woman with natural menopause for more than 1 year of her age at menopause and did not receive HRT (Hormone replacement therapy)[36].

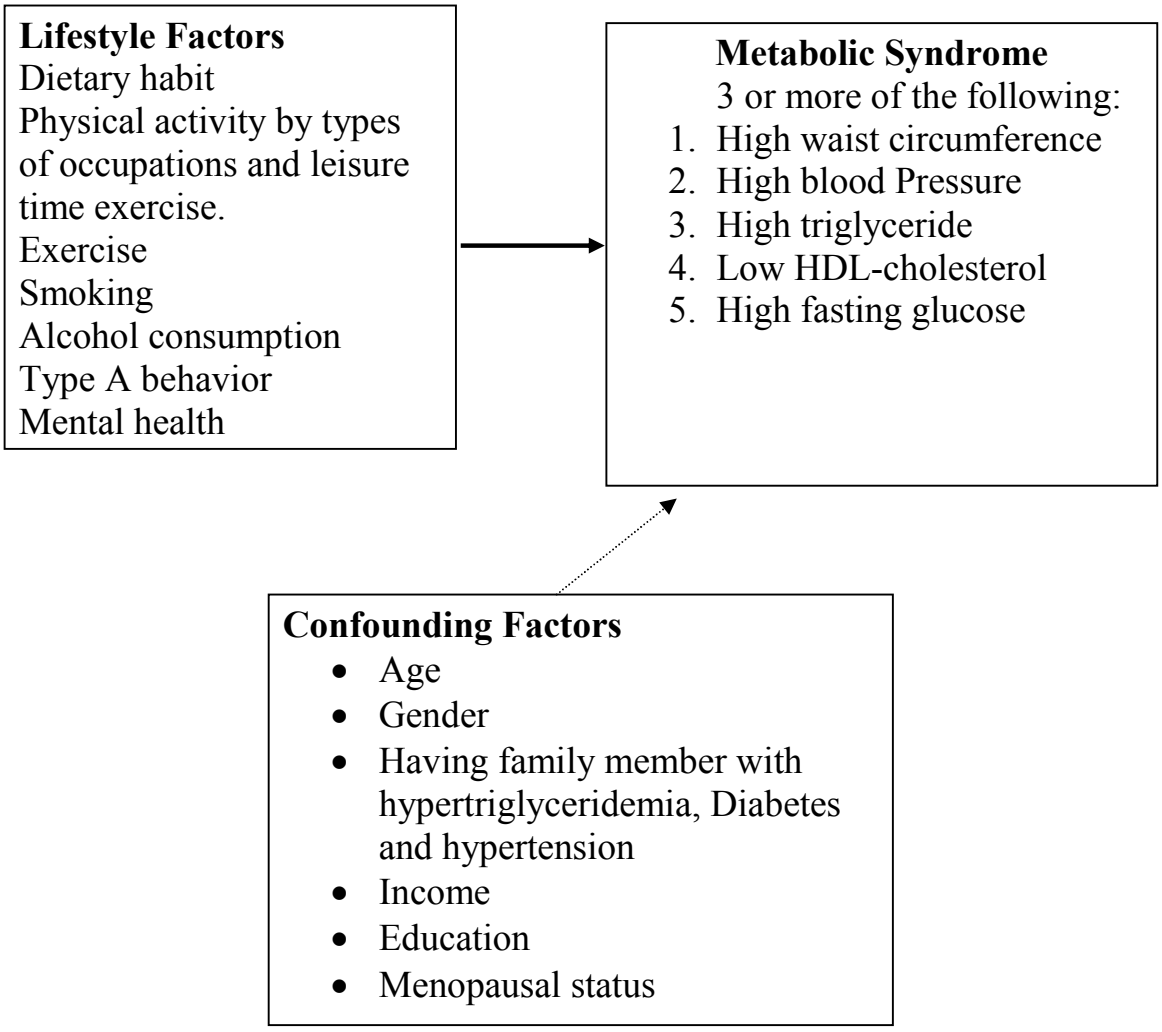

Figure 1.1 Conceptual Framework

CHAPTER II
LITERATURE REVIEW

Literature was reviewed as the following;

1. Definition and criteria for clinical diagnosis of metabolic syndrome.
2. Components of metabolic syndrome.
3. Pathogenesis of metabolic syndrome.
4. Risk factors of metabolic syndrome.
5. Related studies in association of lifestyle risk factors and metabolic syndrome.

The metabolic syndrome, the clustering of abdominal obesity, dyslipidemia, hyperglycemia and hypertension is a major public health challenge worldwide [1, 2]. The metabolic syndrome is not benign; it is associated with a substaintially elevated risk of type 2 diabetes and of cardiovascular disease (CVD) (2-3-fold)[2], and its increasing prevalence could possibly reverse the gains made through recent declining CVD mortality.

The metabolic syndrome is not a new condition. It was first described in the 1920s by Kylin, a Swedish physician, as the association of hypertension, hyperglycaemia and gout. In the 1940s, attention was drawn to upper body adiposity (android or male-type obesity) as the obesity phenotype commonly associated with type 2 diabetes and CVD.

Just as the metabolic syndrome has borne a variety of different names, numerous definitions have also surfaced. The World Health Organization definition[3], and the two others, developed by European Group for the study in Insulin resistance[4] and the National Cholesterol Education Program Third Adult Treatment Panel (ATP III)[5], have been the main ones in use. Each of these agreed on the core components of obesity, hyperglycemia, dyslipidaemia and hypertention.

1. Definitions and Criteria for Clinical Diagnosis of Metabolic Syndrome

At least 3 organizations have recommended clinical criteria for the diagnosis of the metabolic syndrome[5, 6]. Their criteria are similar in many aspects, but they also reveal fundamental differences in positioning of the predominant causes of the syndrome. Now there are currently two major definitions for metabolic syndrome provided by the International Diabetes Federation and the revised National Cholesterol Education Program, respectively[7, 8]. The revised NCEP and IDF definitions of metabolic syndrome are very similar and it can be expected that they will identify many of the same individuals as having metabolic syndrome. The two differences are that IDF excludes any subject without increased waist circumference, while in the NCEP definition metabolic syndrome can be diagnosed based on other criteria and the IDF uses geography-specific cut points for waist circumference, while NCEP uses only one set of cut points for waist circumference regardless of geography. These two definitions are much closer to each other than the original NCEP and WHO definitions.

WHO[9]

The World Health Organization criteria (1999) require presence of diabetes mellitus, impaired glucose tolerance, impaired fasting glucose or insulin resistance, AND two of the following:

- blood pressure: ≥ 140/90 mmHg
- dyslipidaemia: triglycerides (TG): ≥ 1.695 mmol/L and high-density lipoprotein cholesterol (HDL-C) ≤ 0.9 mmol/L (male), ≤ 1.0 mmol/L (female)
- central obesity: waist:hip ratio > 0.90 (male); > 0.85 (female), and/or body mass index > 30 kg/m^2
- microalbuminuria: urinary albumin excretion ratio ≥ 20 mg/min or albumin:creatinine ratio ≥ 30 mg/g

EGIR [9]

The European Group for the Study of Insulin Resistance (1999) requires insulin resistance defined as the top 25% of the fasting insulin values among non-diabetic individuals AND two or more of the following:

Central obesity: waist circumference ≥ 94 cm (male), ≥ 80 cm (female)

Dyslipidaemia: TG ≥ 2.0 mmol/L and/or HDL-C < 1.0 mmol/L or treated for dyslipidaemia

Hypertension: blood pressure ≥ 140/90 mmHg or antihypertensive medication

Fasting plasma glucose ≥ 6.1 mmol/L

NCEP [9]

The US National Cholesterol Education Program Adult Treatment Panel III (2001) requires at least three of the following.

Central obesity: waist circumference ≥ 102 cm or 40 inches (male), ≥ 88 cm or 36 inches(female)

Dyslipidaemia: TG ≥ 1.695 mmol/L (150 mg/dl)

Dyslipidaemia: HDL-C < 40 mg/dL (male), < 50 mg/dL (female)

Blood pressure ≥ 130/85 mmHg

Fasting plasma glucose ≥ 6.1 mmol/L (110 mg/dl)

American Heart Association/Updated NCEP [9]

There is confusion as to whether AHA/NHLBI intended to create another set of guidelines or simply update the NCEP ATP III definition. According to Scott Grundy, University of Texas Southwestern Medical School, Dallas, Texas, the intent was just to update the NCEP ATP III definition and not create a new definition:

Elevated waist circumference:

Men — Equal to or greater than 40 inches (102 cm)

Women — Equal to or greater than 35 inches (88 cm)

Elevated triglycerides: Equal to or greater than 150 mg/dL

Reduced HDL (“good”) cholesterol:

Men — Less than 40 mg/dL

Women — Less than 50 mg/dL

Elevated blood pressure: Equal to or greater than 130/85 mm Hg or use of medication for hypertension

Elevated fasting glucose: Equal to or greater than 100 mg/dL (5.6 mmol/L) or use of medication for hyperglycemia

Note : adapted for Asian by American Heart Association[10].

Central obesity define on Waist circumference

Men	≥ 90 cms (> 36 inches)
Women	≥ 80 cms (> 32 inches)

2. Components of Metabolic syndrome.

ATP III identified 6 components of the metabolic syndrome that related to CVD [11] :

Abdominal obesity

Atherogenic dyslipidemia

Raised Blood Pressure

Insulin resistance ± glucose intolerance

Proinflammatory state

Prothrombotic state

Abdominal obesity is the form of obesity most strongly associated with the metabolic syndrome. It presents clinically as increased waist circumference[11].

Atherogenic dyslipidemia manifests in routine lipoprotein analysis by raised triglycerides and low concentrations of HDL cholesterol. A more detailed analysis usually reveals other lipoprotein abnormalities, eg, increased remnant lipoproteins, elevated apolipoprotein B, small LDL particles, and small HDL particles. All of these abnormalities have been implicated as being independently atherogenic[11].

Elevated blood pressure strongly associates with obesity and commonly occurs in insulin-resistant persons. Hypertension thus commonly is listed among metabolic risk factors. However, some investigators believe that hypertension is less

"metabolic" than other metabolic-syndrome components. Certainly, hypertension is multifactorial in origin. For example, increasing arterial stiffness contributes significantly to systolic hypertension in the elderly. Even so, most conference participants favored inclusion of elevated blood pressure as one component of the metabolic syndrome[11].

Insulin resistance is present in the majority of people with the metabolic syndrome. It strongly associates with other metabolic risk factors and correlates univariately with CVD risk. These associations, combined with belief in its priority, account for the term insulin resistance syndrome. Even so, mechanisms underlying the link to CVD risk factors are uncertain, hence the ATP III's classification of insulin resistance as an emerging risk factor. Patients with longstanding insulin resistance frequently manifest glucose intolerance, another emerging risk factor. When glucose intolerance evolves into diabetes-level hyperglycemia, elevated glucose constitutes a major, independent risk factor for CVD[11].

A proinflammatory state, recognized clinically by elevations of C-reactive protein (CRP), is commonly present in persons with metabolic syndrome. Multiple mechanisms seemingly underlie elevations of CRP. One cause is obesity, because excess adipose tissue releases inflammatory cytokines that may elicit higher CRP levels[11].

A prothrombotic state, characterized by increased plasma plasminogen activator inhibitor (PAI)-1 and fibrinogen, also associates with the metabolic syndrome. Fibrinogen, an acute-phase reactant like CRP, rises in response to a high-cytokine state. Thus, prothrombotic and proinflammatory states may be metabolically interconnected[11].

3. Pathogenesis of Metabolic syndrome.

Obesity and Abnormal Body Fat distribution

ATP III considered the “obesity epidemic” as mainly responsible for the rising prevalence of metabolic syndrome. Obesity contributes to hypertension, high serum cholesterol, low HDL cholesterol, and hyperglycemia, and it otherwise associates with higher CVD risk. Abdominal obesity especially correlates with metabolic risk factors. Excess adipose tissue releases several products that apparently exacerbate these risk factors. They include nonesterified fatty acids (NEFA), cytokines, PAI-1, and adiponectin. A high plasma NEFA level overloads muscle and liver with lipid, which enhances insulin resistance. High CRP levels accompanying obesity may signify cytokine excess and a proinflammatory state. An elevated PAI-1 contributes to a prothrombotic state. Whereas low adiponectin levels that accompany obesity correlate with worsening of metabolic risk factors. The strong connection between obesity (especially abdominal obesity) and risk factors led ATP III to define the metabolic syndrome essentially as a clustering of metabolic complications of obesity.

Insulin Resistance

A second category of causation is insulin resistance. Many investigators place a greater priority on insulin resistance than on obesity in pathogenesis[12, 13]. The argue that insulin resistance, or its accomplice, hyperinsulinemia, directly causes other metabolic risk factors. Identifying a unique role for insulin resistance is complicated by the fact that it is linked to obesity. Insulin resistance generally rises with increasing body fat content, yet abroad range of insulin sensitivities exists at any given level of body fat[14]. Most people with categorical obesity (body mass index [BMI] >= 30 kg/m2) have postprandial hyperinsulinemia and relatively low insulin sensitivity, but variation in insulin sensitivities exist even within the obese population[14]. Overweight persons (BMI 25 to 29.9 kg/m2) likewise exhibit a spectrum of insulin sensitivities, suggesting an inherited component to insulin resistance. In some population, (e.g. South Asians), insulin resistance occurs commonly even with BMI < 25 kg/m2 and apparenntly contributes to a high prevalence of type 2 diabetes and premature CVD. South Asians and others who

manifest insulin resistance with only mild-to-moderate overweight can be said to have primary insulin resistance. Even with primary insulin resistance, however, weight gain seems to enhances insulin resistance and metabolic syndrome. Thus, dissociation of obesity and primary insulin resistance in patients with metabolic syndrome is difficult.

Independent Factors That Mediate Specific Components of the Metabolic Syndrome

Beyond obesity and insulin resistance, each risk factors of the metabolic syndrome is subject to its own regulation through both genetic and acquired factors. This leads to variability in expression of risk factors. Lipoprotein metabolism, for instance, is richly modulated by genetic variation; hence, expression of dyslipidaemias in response to obesity and/or insulin resistance varies considerably. The same holds for blood pressure regulation. Moreover, glucose levels depend on insulin-secretary capacity as well as insulin sensibility. This variation in distal regulation cannot be ignored as an important factor in causation of metabolic syndrome.

Dyslipidaemia

In general, the increasing in free fatty acid fluxs to the liver, increased production of apo B-containing triglyceride-rich very low-density lipoproteins (VLDL) occurs[15]. The effect of insulin on this process in somewhat complex. In the setting of insulin resistance, increased flux of free fatty acids to the liver increases hepatic triglyceride synthesis; however, under physiological conditions, insulin inhibits rather than increases the secretion of VLDL into the systemic circulation[16]. This response in part is an effect of insulin on the degradation of apo B[17]. Yet, insulin is also lipogenic, increasing the transcription and enzyme activity of many genes that relate to triglyceride biosynthesis[18]. Whether or not this pathway remains operational in the setting of systemic insulin resistance has not been completely addressed. Additionally, insulin resistance could also reduce the concentrations of lipoprotein lipase in peripheral tissues (i.e. in adipose tissue more than muscle)[19]. This alteration in lipoprotein lipase, however, seems to contribute less to the hypertriglyceridaemia excellent reflection of the insulin resistance condition and is one of the important criteria for diagnosis of the metabolic syndrome.

The other major lipoprotein disturbance in the metabolic syndrome is a reduction in HDL cholesterol. This reduction is a consequence of changes in HDL composition and metabolism. In the presence of hyperglyceridaemia, a decrease in the cholesterol content of HDL results from decreases in the cholesteryl ester content of the lipoprotein core with variable increases in triglyceride making the particle small and dense, a function in part of cholesteryl ester transfer protein[20]. This change in lipoprotein composition also results in an increased clearance of HDL from the circulation[21]. The relation of these changes in HDL to insulin resistance are probably indirect, arising in concert with the changes in triglyceride-rich lipoprotein metabolism.

In addition to HDL, the composition of LDL is also modified in a similar way. In fact, with fasting serum triglycerides > 2.0 mmol/l, almost all patients have a predominance of small dense LDL[22, 23]. This change LDL composition is attributable to relative depletion of unspecified cholesterol, esterifies cholesterol, and phospholipids with either no change or an increase in LDL triglyceride[24, 25]. Small dense LDL might be more atheogenic than buoyant LDL because (1) it is more toxic to the endothelium; (2) it is more able to transit through the endothelial basement membrane; (3) it adheres well to glycosaminoglycans; (4) it has increased susceptibility to oxidation, and/or (5) it is more selectively bound to scavenger receptors on monocyte derived macrophages[26, 27]; however, this contention is not entirely accepted[28]. In some studies, this alteration in LDL composition is an independent risk factor for cardiovascular disease[29]. However, more often this association is not independent, bur related to the concomitant changes in other lipoproteins and other risk factors[30].

Glucose Intolerance

The defects in insulin action in glucose metabolism include deficiencies in the ability of the hormone to suppress glucose production by the liver and kidney, and to mediate glucose uptake and metabolism in insulin sensitive tissues (i.e. muscle and adipose tissue). The relation between impaired fasting glucose or impaired glucose tolerance and insulin resistance is well supported by human, non-human primate, and rodent studies. To compensate for defects in insulin action, insulin secretion and/or

clearance must be modified to sustain euglycaemia. If this compensation fails, defects in insulin secretion predominate.

Insulin resistance in pancreatic islet beta cells implies that signals that generate glucose-dependent insulin secretion have been adversely modified, and fatty acids are prime candidates. Although free fatty acids can stimulate insulin secretion, increasing and prolonged exposure to excessive concentrations results in fall in insulin secretion[31]. The mechanism for this alteration has been attributed to lipotoxicity through several potential different mechanisms[32-34].

Insulin also can feedback on its own secretion. The importance of this system comes from experiments in rodents in which the insulin receptor is tissue specifically deleted. When the insulin receptor is deleted in skeletal muscle, hyperglycemia does not result[35]; however, the beta-cell specific knockout of the insulin receptor produces progressive glucose intolerance and diabetes[36]. In people with genetic predispositions to development of diabetes, the presumed stress of the insulin resistant environment of beta cell function causes glucose intolerance and ultimately higher risk of diabetes.

Hypertension

The relation between insulin resistance and hypertension is well established[37], and relates to several different mechanisms. First, it is important to note that insulin is vasodilator when given intravenously to people of normal weight[38], with secondary effects on sodium reabsorption in the kidney[39]. Evidence indicates that sodium reabsorption is increased in white people but not Africans or Asians with the metabolic syndrome. In the setting of insulin resistance, the vasodilatory effect in insulin can be lost, but the renal effect on sodium reabsorption preserved. Fatty acids themselves can mediate relative vasoconstriction. Insulin also increases the activity of the sympathetic nervous system[40], an effect that might also be preserved in the setting of the insulin resistance[41]. However, when assessed by concentrations of fasting insulin, HOMA or the HOMA insulin resistance index (HOM-IR), insulin resistance contributes only modestly to the increased prevalence of hypertension in the metabolic syndrome[42].

4. Risk factors of metabolic syndrome.

The cause of the metabolic syndrome is unknown. The pathophysiology is extremely complex and has been only partially elucidated. Most patients are older, obese, sedentary, and have a degree of insulin resistance. The most important factors in order are aging, genetics and lifestyle, i.e., low physical activity and excess caloric intake.

There is debate regarding whether obesity or insulin resistance is the *cause* of the metabolic syndrome or if they are consequences of a more far-reaching metabolic derangement. However, metabolic syndrome is not observed in the absence of insulin resistance, while obesity is not present in many individuals who present with metabolic syndrome. A number of markers of systemic inflammation, including C-reactive protein, are often increased, as are fibrinogen, interleukin 6 (IL–6), Tumor necrosis factor-alpha (TNFα) and others. Some have pointed to oxidative stress due to a variety of causes including increased uric acid levels caused by dietary fructose [9, 43-45].

Age. The prevalence of metabolic syndrome increases with age, affecting less than 10 percent of people in their 20s and 40 percent of people in their 60s. However, some research shows that about one in eight schoolchildren has three or more components of metabolic syndrome. And, other research has identified an association between childhood metabolic syndrome and adult cardiovascular disease decades later[46].

Obesity. A body mass index (BMI) — a measure of percentage of body fat based on height and weight — greater than 25 increases risk of metabolic syndrome. So does abdominal obesity — having an apple shape rather than a pear shape[46].

History of diabetes. Those are more likely to have metabolic syndrome if they have a family history of type 2 diabetes or a history of diabetes during pregnancy (gestational diabetes) [46].

Other diseases. A diagnosis of high blood pressure, cardiovascular disease or polycystic ovary syndrome — a similar type of metabolic problem that affects a woman's hormones and reproductive system — also increases risk of metabolic syndrome[46].

Lifestyle is defined a set of habits and customs that is influenced, modified encouraged, or constrained by the life long process of socialization. These habits and customs included use of substance such as alcohol, tobacco, tea, coffee, dietary habits, exercise, which have important implications for health and are often the subject of epidemiologic investigation[47].

5. Related studies in association of lifestyle risk factors and MetS.

Dietary habits

Dietary habits had been associated with the prevalence of metabolic syndrome and produced OR 1.13 (95% CI 1.05-1.21) on component with potatoes, red or white meat, and meat products while percent of carbohydrate is not significant[48, 49]. One study reported that subjects in the highest quartile of dairy consumption had lower odds of having metabolic syndrome (OR by quartile 1-4 : 1, 0.83, 0.74, 0.69 respectively)[50] but higher intake of dietary carbohydrates and lower intake of crude fiber were each associated with an increased risk for metabolic syndrome (RR ranging from 1.3 to 1.9)[51].

Physical activity by job

One study found that physical activity associated with the prevalence of metabolic syndrome. Participants who did not engage in any moderate or vigorous physical activity during leisure time had almost twice the odds of having metabolic syndrome (OR, 1.90; 95% CI, 1.22 to 2.97) as those who reportedly engaged in $\geq$ 150 min/wk of such activity[52]. One study showed that physical activity was protective against metabolic syndrome (RR 0.84 [95%CI 0.76-0.92][51] and the one was an independent protective factor(OR 0.70 [0.41-1.19]) [53].While compare physically active with sedentary was OR 0.63 (0.51-0.78)[54]. Regarding to gender, Moderate exercise (2-3 sessions/week) in men produced protective effect to the metabolic syndrome (OR 0.6; 95% CI 0.5-0.9)[49].

Cigarette smoking

For cigarette smoking, one study find that OR was 1.51 compared between Ex-smoker and nonsmoker and another compared between 0-5 cigarettes/day and ≥ 6 cigarettes/day was 1.78 (95% CI 0.61-5.22) but both not significant (p = 0.423)[51, 55]. One study find that current smoking were associated factor for the metabolic syndrome regardless gender with OR 1.4 (95% CI 1.1-1.8) and 1.6 (95% CI 1.2-2.1) in men and women respectively[49] or 1.12 (95% CI 1.01-1.24) after adjusted potential confounder[54].

Alcohol consumption

The adjusted odds Ratio for the metabolic syndrome in the group consuming 1-14.9 g alcohol/day was 0.71 (95% CI 0.53-0.95)[56] whereas non drinks/day compared with 1-3 drinks/day produced protective effect to MetS was 1.36 (95% CI 1.11-1.66)[51]. One study find that light alcohol drinking (<15 g/day) in woman demonstrated protective effect of odds of the metabolic syndrome (OR 0.8 [95% CI 0.7-0.9])[49] and alcohol beverages has an odds ratio of 1.26 (95% CI 1.21-1.33) in light dose[54].

Type A Bahavior

After adjustment for age, blood pressure, cigarette smoking, serum cholesterol, consumption of alcohol, and educational attainment yielded relative risks of 0.99 in usual care, 0.81 in special intervention, and 0.87 overall (95% confidence interval = 0.59-1.28) [57]

Mental health or stress

Chronic work stress have been associated with the metabolic syndrome with OR adjusted for age was 2.25 (95% CI 1.31 to 3.85)[58].

Menopause

For menopause status, postmenopausal status was associated with an increased risk of the metabolic syndrome independent of normal aging in Korean women (OR 1.60 95%CI 1.04-2.46)[55].

CHAPTER III
MATERIALS AND METHODS

1. Study design

A population based case-control study was used to demonstrate associations between lifestyle factors and metabolic syndrome (MetS) among population of Banphaeo district, Samutsakorn Province. Adult participants aged 35 and above who enrolled in Banphaeo Routine Hypertension and Diabetes Screening Program (BRHDSP) during November 2007 to June 2008 were invited to participate for interviewing past history of their lifestyle and other factors during the past 2-3 years which related to metabolic syndrome.

2. Population and the study population

Population were adult population aged 35 years and above in Banphaeo district, Samutsakorn Province.

Sampling frame was list of the names of participants who enrolled in Banphaeo Routine Hypertension and Diabetes Screening Program (BRHDSP) during November 2007 to June 2008 BRHDSP.

Study population were adult population aged 35 years to 60 years in Banphaeo district, Samutsakorn Province who enrolled and participated in Banphaeo Routine Hypertension and Diabetes Screening Program (BRHDSP) during November 2007 to June 2008. The criteria of case and control were :

Case referred to those who satisfied 3 or more of the 5 criteria of metabolic syndrome from NCEP ATP III. The 5 criteria included;

Waist circumference ≥ 90 cm in men or ≥ 80 cm in women.

Serum triglyceride concentration ≥ 150 mg/dl (or on drug treatment for elevated triglycerides)

Serum HDL concentration < 40 mg/dl in men and <50 mg/dl in women (or on drug treatment for reduced HDL-C)

Systolic blood pressure ≥ 130 mmHg and/or diastolic blood pressure ≥ 85 mmHg (or on antihypertensive drug)

Fasting Plasma Glucose ≥ 100 mg/dL (or on drug treatment for elevated glucose)

Controls referred to those who were not satisfied any criteria or possessed only 1 or 2 criteria of metabolic syndrome from NCEP ATP III.

The study population : The results from (BRHDSP) were used for selection of cases and controls.

3. Inclusion and exclusion criteria

Inclusion criteria

1. Subject who had complete information on 5 components of metabolic syndrome according to NCEP ATP III.
2. Subject who agreed to participate in this study under informed consent.

Exclusion criteria

1. Subject who had physical and mental health problem at the time of data collection and was not able to provide the answer for lifestyle questions by verbal or writing.
2. Subjects who had severe complication of any diseases

4. Scope of study

This study was conducted among adults of Banphaeo district, Samutsakhorn Province who participated in Routine Hypertension and Diabetes Screening Program (BRHDSP) during November 2007 to June 2008 in order to identify the associations of the lifestyle factors and MetS.

5. Benefits of the study

The results from this study demonstrated potential lifestyle factors significantly associated with metabolic syndrome that will be used for improving primary prevention program in order to prevent and to reduce the prevalence of metabolic syndrome and the incidence of cardiovascular disease (CVD) among adults of Banphaeo district, Samutsakhorn Province in the future.

6. Sample Size calculation

Sample Size calculation for this study was based on the design for case - control study with the ratio of cases per controls in 1:1 with the following formula:

$$n = \frac{[Z_{\alpha/2}\sqrt{(c+1)P(1-P)} + Z_{\beta}\sqrt{cP_1(1-P_1)+P_0(1-P_0)}]^2}{c(P_1 - P_0)^2}$$

$$P_1 = \frac{OR \times P_0}{OR \times P_0 + (1 - P_0)}$$

$$P = \frac{P_1 - cP_0}{(c+1)}$$

When :

n = minimum sample size of case

α = type I error (at 95% confidence interval) = 0.05 so $Z_{\alpha/2} = 1.96$

β = type II error

OR = Odds Ratio of low intake of fruit and vegetable =1.9 [85]

P_0 = rate of low intake of fruit and vegetable among population in central region of Thailand. = 75.5% among male, 72.1% among female**

c = Ratio between control and case

*** Ministry of public health, Third national health survey among Thai population by examination in 2003-2004.*

The minimum sample size for 178 for cases and 178 for controls when relative precision was 50%.

7. Sampling technique

Cases and controls were selected from the sampling frame of participants who enrolled in BRHDSP. Sampling technique by list of the name of participants who enrolled during November 2007 to June 2008 with the number of 23,691 with screening rate 73.28% of total population of Banphaeo district, Samutsakorn Province. There were 2 types of screening program according to budget, with and without blood test. Total number of participants had blood test were 2,283. There were 753 populations were also excluded. Then 1,530 participants were eligible for the study and categorized into cases and controls by the criteria of MetS of NCEP ATP III definition; cases had at least 3 or more of 5 criteria, controls had normal or 1-2 components. Stratified random sampling with proportional to size of participants by sub-district were used to select cases and controls. Finally, there were 196 cases and 212 controls included in the analysis.

8. Materials

Data collection was performed by using questionnaires for lifestyle factors and other factors which contained the following contents:

Part 1 General characteristics of participants on age, gender, marital status, religion, address, education level, occupation, income and family history of chronic diseases.

Part 2 History of lifestyle behavior in the past 3 years (smoking, alcohol consumption, physical activity, dietary habit),

Part 3 Standard questionnaire for Type A behavior test to define behavior type.

Part 4 Standard questionnaire for Suanprung Stress Test to define stress status.

Part 5 Data in components of the metabolic syndrome from medical record retrieved from Banphaeo Routine Hypertension and Diabetes Screening Program (BRHDSP) by research.

9. Methods of data collection

Preparing Stage

1. Proposal was submitted and reviewed by the director of Banphaeo hospital and the head of Banphaeo public health office for permission to use data of participants according to criterion of metabolic syndrome from BRHDSP.

2. Developing questionnaire and pretest for validity and reliability in part II of the questionnaire.

3. Submission of the proposal to the Ethical Committee of Mahidol University for reviewing.

Action Stage

1. Selection of case and control from the sample frame.

2. Data collection for MetS criteria in BRHDSP.

In BRHDSP; data on criteria of metabolic syndrome was mainly performed by trained health officers from district hospital at local health centers and collected data from Banphaeo hospital which summarized from all local health center by monthly report.

2.1 Waist circumference was measured by using a non elastic tape at umbilicus point as recommended by the Ministry of Public Health with the subject standing and performed by trained public health personnel in local health center.

2.2 Blood pressure was measured by using an automatic (arm cuff) blood pressure monitor (Omron, Vernon Hills, IL) after 5 minutes resting and performed by public health personals.

2.3 Blood test

After 12 hours fasting, blood sample of participants was drawn from antecubital vein into tube containing EDTA which performed by public health personnel at local health centers. Blood sample tubes were transported to Banphaeo Hospital laboratory within at noon in the same day for determining triglyceride by enzymatic method, HDL-C by dextran-magnesium precipitation, and fasting blood glucose by hexokinase2glucose-6-phosphate dehydrogenase method.

3. Collecting data for lifestyle factors and genetic factors by interviewing with questionnaires was performed in both cases and controls by the researcher and trained health personnel by home visit.

Data analysis

1. Descriptive statistics

Description of characteristics of cases and controls by using frequency, percentage, mean and standard deviation. Chi square test and students' t test will be used for identifying significant differences

2. Analytical statistics

Univariate analysis: crude Odds Ratio and 95% confidence interval was used to demonstrate association of lifestyle factors and MetS, and non-parametric test for non normal distribution data.

Multivariate analysis: the adjusted Odds Ratio and 95% confidence interval by Binary logistic regression analysis will be used to examine the association between lifestyle factors and metabolic syndrome simultaneously through entering method and stratification by gender.

CHAPTER IV
RESULTS

This population based case control study was conducted to determine the associations between lifestyle factors and metabolic syndrome(MetS) among population of Banphaeo, Samutsakorn province. Datas were collected among 196 cases and 212 controls who lived in Banphaeo district and attended hypertension and diabetes screening projects (BRHDSP) in year 2008 (November 2007 to June 2008).

The results of the study were presented as follow:

1. Descriptive characteristics of study population by components of metabolic syndrome, demographic factors, and other important factors among cases with metabolic syndrome and control without metabolic syndrome.

1.1 Components of metabolic syndrome

1.2 General characteristics and other factors

1.2.1 Age

1.2.2 Sex

1.2.3 Marital Status

1.2.4 Religion

1.2.5 Education level

1.2.6 Occupation

1.2.7 Income

1.2.8 Family history of hypertension

1.2.9 Family history of DM

1.2.10 Family history of hypercholesterolemia

1.2.11 Menopausal status (among female)

2. Prevalence and crude association (Crude Odds Ratio : OR) with 95% confidence interval (95% CI) of behavioral lifestyle factors of metabolic syndrome which were.

2.1 Smoking

2.2 Alcohol drinking

2.3 Physical activity

2.4 Dietary habit

2.5 Behavioral Type A

2.6 Level of stress

3. Adjusted associations (Adjusted Odds Ratio : OR) with 95% confidence interval (95% CI) between each lifestyle factor and metabolic syndrome by multiple logistic regression analysis controlling for the effects of others lifestyle factors including confounding variables which were age, gender, having history of hypercholesterolemia, DM, hypertension in family member, income, education and menopausal status. Interaction term was identified, and stratification by gender for male and female participants.

1. Characteristics of the study population

1.1 Components of metabolic syndrome

When considered having the 5 components metabolic syndrome among cases and controls which were waist circumference (WC), Triglyceride (TG), high density lipoprotein (HDL), blood pressure(BP) and fasting plasma Glucose (FPG). Participants, with waist circumference bigger than normal, were 81.12% in cases and 1.42% in controls. Participants, with triglyceride higher than normal, were 83.16% in cases and 0.94% in controls. Participants, with HDL components lower than normal, were 56.69%% in cases and none in controls. Participants, with high BP, were 80.61% in cases and 13.21% in controls. And fasting blood sugar higher than normal were 57.14% in cases and none in controls (Table 4.1).

Table 4.1 Prevalence (%) of each components of metabolic syndrome among cases and controls.

Components	Cases		Controls	
	n	%	n	%
WC ≥ normal*	159	81.12	3	1.42
TG ≥ 150 mg/dl	163	83.16	2	0.94
HDL ≤ normal**	117	59.69	0	0.00
BP > 130/85 mmHg	158	80.61	28	13.21
FPG ≥ 100 mg/dL	112	57.14	0	0.00

*WC (waist circumference) : ≥ 90 cm in men or ≥ 80 cm in women by Asian definition

**HDL (serum HDL) : concentration < 40 mg/dl in men and <50 mg/dl in women (or on drug treatment for reduced HDL-C)

1.2. General characteristics of the study population.

1.2.1 Age

The mean of age were 50.96±6.30 years for cases and 49.14±7.09 years for controls group. There was statistically significant difference (p<0.001) between means of age among cases and controls. In crude association, those who aged 45-54 years were more likely to develop MetS 2.23 times compared to aged 55-60 years (OR=2.23 95%CI 1.31-3.80). (Table 4.2)

1.2.2 Gender

Among cases and controls, proportions of male participants were 37.24% and 34.91% and female participants were 62.76% and 65.09%, respectively. There was statistical difference in proportions of gender between cases and controls (p<0.001). There was no significant crude association between gender and MetS. (Table 4.2)

1.2.3 Marital status

Most of cases in 84.69% and controls in 73.58% were married .The participants, who were single group, were 9.18% among cases and 18.87% among controls, widowed group were 5.10% among cases and 4.72% among controls and divorce were less group of 1.02% in cases and 2.83% in controls. There was statistically significant differences in proportions of marital status between cases and controls (p<0.001) (Table 4.1). In crude association, who were widowed were more likely to develop MetS 2.36 times compared to who were couple (OR=2.36 95%CI 1.30-4.30). (Table 4.2)

1.2.4 Religion

The majority of this group of study population were Buddhism in 100% in cases and 99.53% in controls. There was 1 who was Christian in control. (Table 4.2).

1.2.5 Level of Education

The large proportions of cases and control finished primary school in 83.16% and 79.25% respectively. Illiterate rate found in 7.65% and 3.77% in cases and controls respectively. The less group in cases finished pre-high school and high school which 4.59% while in controls group finished high school in 3.30%, and finished pre-high school in 8.49% and finished undergraduate in 5.19% in controls. There was statistically significant differences in proportions of education level between cases and controls (p<0.001) (Table 4.2). In crude association, who finished pre high school level were more likely to develop MetS 3.75 times compared to who were illiterate (OR=3.75 95%CI 1.16-12.12).

1.2.6 Occupation

The majority of cases in 54.08% and controls in 54.25% were agriculturist. Proportion of employee (labor work) was 17.86% in cases and 19.31% in controls. Proportion of participants who did not work was 10.20% in cases and 10.38% in controls. Being merchant found 9.18% in cases and 6.13% in controls. Occupation in office employee were 2.55% among cases and 2.36% among controls,

being servant found 2.04% among cases and 3.77% among controls and housework were 1.02% among cases and 1.89% among controls while others was 3.06% in case and 1.89% on controls. There was statistically significant differences in proportions of types of occupations between cases and controls ($p< 0.001$) (Table 4.2). There was no significant crude association between occupation and MetS.

1.2.7 Personal monthly income

Most of cases had personal income less than or equal to 5,000 bahts per month and of controls was 5,001-10,000 bahts in 40.31% and 39.62% respectively. Proportions of those with personal income between 5,001 – 10,000 bahts per month in cases was 32.14% and less than or equal to 5,000 bahts in controls was 35.38% in controls, there were participants with no income in cases and controls in 22.96% and 17.92% respectively. Proportions of cases with personal income more than 10,001 were 4.59% and 7.08% respectively. Average personal income in case was 7,013$\pm$7247.73 and in controls was 8,201$\pm$7,338.98. There was statistically significant difference in proportions of family income between cases and controls ($p< 0.001$) (Table 4.2). There was no significant crude association between personal monthly income and MetS.

1.2.8 Having family history of hypertension

Most of cases and controls had no family history of hypertension with 64.62% and 71.23% respectively. There was statistically significant difference in proportions of family history of hypertension between cases and controls ($p< 0.001$) (Table 4.2). There was no significant crude association between having family history of hypertension and MetS.

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1.2.9 Having family history with diabetes mellitus

Most of cases and controls had no family history of DM with 70.26% and 83.49% respectively. There was statistically significant difference in proportions of family history of DM between cases and controls ($p< 0.001$) (Table 4.2). In crude association, who had family history of DM were more likely to develop

MetS 2.14 times compared to who had no family history of DM (OR=2.14 95%CI 1.33-3.44).

1.2.10 Having family history with hypercholesterolemia

Most of cases and controls had no family history of hypercholesterolemia with 81.63% and 84.43% respectively. There was statistically significant difference in proportions of family history of hypercholesterolemia between cases and controls ($p< 0.001$) (Table 4.2). There was no significant crude association between family history of hypercholesterolemia and MetS.

1.2.11 Menopausal status (only in female)

Cases were in menopausal status in 40.98% and control were in menopausal status in 29.71%. There was no statistically significant difference in proportions of menstruation status between cases and controls ($p = 0.237$) (There was no significant crude association but borderline significant association between menopausal status and MetS.(Table 4.2).

Table 4.2 Demographic characteristics of study population and crude association with Metabolic syndrome.

Characteristics	**Case (n=196)**		**Control (n=212)**		**Crude OR**	***95% CI***	***p* – value***
	n	**%**	**n**	**%**			
Age (Year)							
35-44**	36	18.37	65	30.66	1.00		
45-54	87	44.39	88	41.51	1.78	1.08-2.95	0.02
55-60	73	37.24	59	27.83	2.23	1.31-3.80	<0.01
Mean	50.96		49.14				
SD	6.30		7.09				

* Chi-square test

** Reference group

Table 4.2 Demographic characteristics of study population and crude association with Metabolic syndrome (continued).

Characteristics	**Case (n=196)**		**Control (n=212)**		**Crude OR**	***95% CI***	***p* – value***
	n	**%**	**n**	**%**			
Gender							
Male**	73	37.24	74	34.91	1.00		
Female	123	62.76	138	65.09	1.11	0.74-1.66	0.62
Marital status							
Couple**	166	84.69	156	73.58	1.00		
Widowed	10	5.10	10	4.72	2.36	1.30-4.30	< 0.01
Divorce	2	1.02	6	2.83	3.19	0.64-16.05	0.16
Single	18	9.18	40	18.87	1.06	0.43-2.63	0.89
Religion							
Buddhist	196	100.00	211	99.53	**N/A**		
Christian	0	0.00	1	0.47			
Education level							
Illiterate**	15	7.65	8	3.77	1.00		
Primary	163	83.16	168	79.25	1.93	0.80-4.68	0.14
Pre-high school	9	4.59	18	8.49	3.75	1.16-12.12	0.03
High school and over	9	4.59	18	8.49	1.46	0.39-5.40	0.57

* Chi-square test

** Reference group

Table 4.2 Demographic characteristics of study population and crude association with Metabolic syndrome (continued).

Characteristics	**Case (n=196)**		**Control (n=212)**		**Crude OR**	***95% CI***	***p* – value***
	n	**%**	**n**	**%**			
Occupation							
Agriculture	106	54.08	115	54.25	1.65	0.41-6.71	0.48
Servant	4	2.04	8	3.77	1.52	0.60-3.88	0.38
Employee(labor)	35	17.86	41	19.34	1.10	0.28-4.37	0.89
Housework	2	1.02	4	1.89	0.55	0.09-3.33	0.52
Office employee	5	2.55	5	2.36	0.94	0.44-2.00	0.87
Merchant	18	9.18	13	6.13	0.55	0.14-2.11	0.38
Others	6	3.06	4	1.89	1.01	0.52-1.96	0.97
No work**	20	10.20	22	10.38	1.00		
Personal Income per month (Baht)							
No income	45	22.96	38	17.92	1.76	0.72-4.25	0.21
≤ 5,000**	79	40.31	75	35.38	1.00		
5,001 – 10,000	63	32.14	84	39.62	1.40	0.89-2.21	0.14
≥10,001	9	4.59	15	7.08	0.89	0.52-1.52	0.67
Mean	7,013.38		8,201.39				
S.D.	7,247.73		7,338.98				

* Chi-square test

** Reference group

Table 4.2 Demographic characteristics of study population and crude association with Metabolic syndrome (continued)**.**

Characteristics	Case (n=196)		Control (n=212)		Crude OR	*95% CI*	*p* – value*
	n	%	n	%			
Family history of hypertension							
Yes	69	35.38	61	28.77	1.36	0.89-2.06	0.15
No**	126	64.62	151	71.23	1.00		
Family history of DM							
Yes	58	29.74	35	16.51	2.14	1.33-3.44	0.002
No**	137	70.26	177	83.49	1.00		
Family history of hypercholesterolemia							
Yes	36	18.37	33	15.57	1.22	0.73-2.05	0.45
No**	160	81.63	179	84.43	1.00		
Note: 1 data missing							
Menopause Status (In female)							
N	122		138				
No **	72	59.02	97	70.29	1		0.058
Yes	50	40.98	41	29.71	1.64	0.98-2.74	

* Chi-square test

** Reference group

2. Prevalence and crude association of behavioral lifestyle factors for metabolic syndrome.

2.1 Smoking

Most of cases and control was non-smoker which were 80.10% and 79.72% respectively. Current smoker was 12.76% in case and 9.43% in controls while ex-smoker was 7.14% and 10.85% in cases and controls group respectively. The crude association did not demonstrate significant association of smoking with MetS (Table 4.3).

Table 4.3 Prevalence of smoking among total study populations and crude association with metabolic syndrome.

Characteristics	**Case (n=196)**		**Control (n=212)**		***Crude OR***	***95% CI***	***p* – value***
	n	**%**	**n**	**%**			
Smoking							
Current smoker	25	12.76	20	9.43	0.74	0.40-1.39	0.35
Ex-smoker	14	7.14	23	10.85	1.53	0.76-3.07	0.24
Non-smoker**	157	80.10	169	79.72	1.00		

* Chi-square test

** Reference group

Most of cases and controls were non-smoker which were 84.95% in case and 86.67% in control while 1-10 cigarettes per day was 10.75% in case and 10.26% in controls. There was statistical difference in proportions of smoking in cases and controls ($p<0.01$). There was no statistical association in dose of smoking to MetS in crude analysis (Table 4.4).

Table 4.4 Prevalence of smoking quantity among total study populations and crude association with metabolic syndrome

Characteristics	Case (n=186)		Control (n=195)		*Crude OR*	*95% CI*	*p* – value*
	n	%	n	%			
Smoking quantity							
Non-smoker **	158	84.95	169	86.67	1.00		
1-10 cigarettes a day	20	10.75	20	10.26	0.94	0.48-1.80	0.84
11s and more a day	8	4.30	6	3.08	0.70	0.24-2.07	0.52

* Chi-square test

** Reference group

2.2 Alcohol drinking

Most of cases and controls group were non-drinker which were 72.45% and 79.33% respectively. The less group were ex-drinker in cases and controls which is 6.12% and 4.81% respectively while current drinker were 21.43% and 15.87% in cases and controls group. There was statistically difference in proportions of alcohol drinking in cases and controls ($p<0.01$), but no significant association in crude analysis between alcohol drinking and MetS. (Table 4.5).

Table 4.5 Prevalence of alcohol drinking among total study populations and crude association with metabolic syndrome.

Characteristics	Case (n=196)		Control (n=208)		*Crude OR*	*95% CI*	*p* – value*
	n	%	n	%			
Alcohol							
Current drinker	42	21.43	33	15.87	0.68	0.41-1.12	0.13
Ex-drinker	12	6.12	10	4.81	0.72	0.30-1.71	0.45
Non-drinker**	142	72.45	165	79.33	1.00		

* Chi-square test

** Reference group

2.3 Physical activity

2.3.1 By daily transportation

There were 4 types of daily traveling to and from work among study population by car, by motorcycle, by bicycle and by walking. Most of cases and controls usually traveled to work by walking which were 59.78% and 55.68% respectively. Traveling to work by motorcycle were 22.75% and 29.19% in cases and controls respectively. Some of them traveled by car which were 11.38% and 7.03% in cases and controls group and by bike were 1.80% and 4.32% respectively. There was significant difference in proportions by traveling to work between cases and controls group.

Grouping daily traveling to work was performed into 3 groups according to light heavy and others which was not clear in physical activity by daily traveling to work. Traveling by car and motorcycle were light, and walking and biking were heavy physical activity.

It was found that strong physical activity by walking in daily traveling to work was the major group which were 61.68% in cases and 60.00% in controls, mild group was 34.13% in case and 36.22% in controls and others transport physical activity was 4.19% in cases and 1.09% in controls. The crude analysis did not demonstrate significant association between daily transportation and MetS (Table 4.6).

Table 4.6 Prevalence of physical activity by daily traveling to work among study populations and crude association with metabolic syndrome.

Characteristics	**Case (n=167)**		**Control (n=185)**		***Crude OR***	***95% CI***	***p* – value***
	n	**%**	**n**	**%**			
Physical activity by daily traveling to work							
Light	57	34.13	67	36.22	0.93	0.32-2.74	0.89
Heavy**	103	61.68	111	60.00	1.00		
Others	7	4.19	7	3.78	1.09	0.70-1.70	0.70

* Chi-square test

** Reference group

2.3.2 By type of occupations

There were many kinds of occupations among study population. Cases and controls were divided into 2 types of job by heavy, light and moderate works. Heavy works were standing, walking continuously including construction work and carrying heavy things all working time, while light and moderate works were in house working, selling and working in office. Heavy work found in 60.10% in cases and 69.19% in controls. The crude analysis did not demonstrate significant association between types of job and MetS (Table 4.7).

Table 4.7 Prevalence of physical activity by type of occupations among total study populations and crude association with metabolic syndrome.

Characteristics	Case (n=193)		Control (n=211)		*Crude OR*	*95% CI*	*p* – value*
	n	%	n	%			
Type of job							
Heavy**	116	60.10	145	69.19	**1.00**		
Light and moderate	77	39.90	65	30.81	**0.67**	**0.44-1.01**	**0.06**

* Chi-square test

** Reference group

2.3.3 By leisure time activity (Exercise)

Physical activity by leisure time activity or exercise in cases and controls were grouped into 4 types; no exercise, irregular exercise (less than 3 times per week and 30 minutes or more per time) and regular exercise (more than or equal to 3 times per week and 30 minutes or more per time). Most of cases and controls had no exercise which was 85.20% and 81.60% respectively. The least small group were irregular exercise which was 4.08% in cases and 4.72% in controls respectively. The crude analysis did not demonstrate significant associations between exercise and MetS (Table 4.8).

Table 4.8 Prevalence of exercise habits among total study population and crude association with metabolic syndrome.

Characteristics	Case (n=186)		Control (n=188)		*Crude OR*	*95% CI*	*p* – value*
	n	%	n	%			
Leisure time activity							
Regular exercise**	21	10.71	29	13.68	1.00		
Irregular exercise	8	4.08	10	4.72	0.75	0.41-1.37	0.35
No exercise	167	85.20	173	81.60	0.90	0.31-2.68	0.86

* Chi-square test

** Reference group

Cases and controls were sleep after main meal in in sometimes especially of their lunch which were 63.98% and 66.49% respectively, while usually sleep after meal in cases and mostly after meal in controls were 4.84% and 5.85% respectively. The crude analysis did not demonstrate significant association between sleep after meal and MetS (Table 4.9).

Table 4.9 Prevalence of sleeping after right main meals among total study populations and crude association with metabolic syndrome.

Characteristics	Case (n=186)		Control (n=188)		*Crude OR*	*95% CI*	*p* – value*
	n	%	n	%			
Sleep right after main meals							
Usually	9	4.84	12	6.38	1.31	0.76-2.25	0.32
Mostly	29	15.59	11	5.85	3.64	1.57-8.45	0.003
Sometimes	119	63.98	125	66.49	1.03	0.38-2.78	0.95
Never**	29	15.59	40	21.28	1.00		

* Chi-square test

** Reference group

2.4 Dietary habits

2.4.1 Food taste preference and pattern of eating

Most of cases and controls defined main taste was salty 29.38% and 24.76% respectively, while who favored sweety main taste were 23.20% and 23.33% in cases and controls. Who favored sour taste were 21.13% and 20.95% in cases and controls. Who favored normal taste in 13.40% and 18.57% in cases and controls. Who favored spicy were 12.89% and 12.38% in cases and controls respectively. The crude analysis did not demonstrate significant associations between favorite taste and MetS (Table 4.10).

Table 4.10 Prevalence of preferrent main taste among total study population and crude association with metabolic syndrome.

Characteristics	**Case (n=194)**		**Control (n=210)**		***Crude OR***	***95% CI***	***p* – value***
	n	**%**	**n**	**%**			
Main taste							
Normal**	26	13.40	39	18.57	1.00		
Sweety	45	23.20	49	23.33	**1.44**	**0.69-3.02**	**0.33**
Sour	41	21.13	44	20.95	**1.64**	**0.88-3.06**	**0.12**
Salty	57	29.38	52	24.76	**1.40**	**0.73-2.69**	**0.32**
Spicy	25	12.89	26	12.38	**1.38**	**0.73-2.61**	**0.33**

* Chi square test ** Reference group

Most of cases and controls had main meals 3 times a day which were 79.79% and 83.02% respectively while less than 3 times a day were 15.43% and 13.21% in cases and controls and more than 3 times a day were 4.79% and 3.77% respectively. The crude analysis did not demonstrate significant association between eating pattern and MetS (Table 4.11).

Table 4.11 Prevalence of having main meals among total study populations and crude association with metabolic syndrome.

Characteristics	Case (n=188)		Control (n=212)		*Crude OR*	*95% CI*	*p* – value*
	n	%	n	%			
Frequency of eating pattern per day							
Less than 3 times**	29	15.43	28	13.21	1.00		
3 times a day	150	79.79	176	83.02	0.82	0.47-1.44	0.50
More than 3 times	9	4.79	8	3.77	1.09	0.37-3.21	0.88

* Chi square test

** Reference group

Most of cases and controls usually ate while watching T.V. less than 3 times a week which 93.85% and 96.40% respectively. The crude analysis did not demonstrate significant association between eat while watching T.V. and MetS (Table 4.12).

Table 4.12 Prevalence of eating while watching T.V. among the total study populations and crude association with metabolic syndrome.

Characteristics	Case (n=130)		Control (n=135)		*Crude OR*	*95% CI*	*p* – value*
	n	%	n	%			
Eating while watching T.V. per week							
0-7 times**	122	93.85	134	96.40	**1.00**		
More than 7 times	8	6.15	5	3.60	**0.57**	**0.18-1.79**	**0.33**

* Chi square test

** Reference group

2.4.2 Frequency of food intake for each types of food

Frequency of intake foods was grouped into low and high intake by types of foods. Low intake meant frequency of eating was less than or equal to 3 times per week and high intake was more than 3 times per week.

Most of cases and controls had low intake all meat foods which were 75.90% and 75.36% respectively. The crude analysis did not demonstrate significant association between all meat foods and MetS (Table 4.13).

Seafood referred to squid, shrimp, shells, excluding fish. Most of cases and control had low intake seafood which were 83.67% and 90.57% respectively. But high intake of seafood among cases was higher than among controls (16.33% and 9.43%). The crude analysis demonstrate significant association between seafoods and MetS by who usually had high intake of seafood were more likely to develop MetS (OR 1.87 95% CI 1.03-3.40) when compared high intake to low intake (Table 4.13).

Most of cases had low intake fish which was 50.51% while most of controls had eating high intake fish which was 56.13%. The crude analysis did not demonstrate significant association between fish and MetS (Table 4.13).

Most of cases and control had low intake brown rice which were 88.27% and 89.15% respectively. The crude analysis did not demonstrate significant association between having brown rice and MetS (Table 4.13).

Most of cases and control had high intake fruits and vegetables which were 55.61% and 59.91% respectively. The crude analysis did not demonstrate significant association between fruits and vegetables intake and MetS (Table 4.13).

Table 4.13 Frequency of eating by types of food among the total study populations and crude association with metabolic syndrome.

Types of food	Case		Control		*Crude OR*	*95% CI*	*p* – value*
	n	%	n	%			
All meat							
Low intake**	148	75.90	159	75.36	1.00		
High intake	47	24.10	52	24.64	0.97	0.62-1.53	0.90
Seafood							
Low intake**	164	83.67	192	90.57	1.00		
High intake	32	16.33	20	9.43	1.87	1.03-3.40	0.04
Fish							
Low intake	99	50.51	93	43.87	1.31	0.88-1.93	0.18
High intake**	97	49.49	119	56.13	1.00		
Brown Rice							
Low intake	173	88.27	189	89.15	0.92	0.50-1.69	0.78
High intake**	23	11.73	23	10.85	1.00		
Fruit and vegetable							
Low intake	87	44.39	85	40.09	1.19	0.80-1.77	0.38
High intake**	109	55.61	127	59.91	1.00		

* Chi square test

** Reference group

2.5 Behavioral Type A

Most of cases and control had both type A and B trait at he same time which were 63.08% and 58.77%, while having type behavior B only were 25.64% and 35.55% in cases and controls respectively. Having type A behavior only in cases was 7.69% and in controls was 3.79% and definite type B behavior only were 3.59% in cases and 1.90% in controls. The crude analysis did not demonstrate significant association between having type A behavior and MetS (Table 4.14).

Table 4.14 Prevalence of behaviors among the total study populations and crude association with metabolic syndrome.

Behaviors	**Case (n=196)**		**Control (n=212)**		***Crude OR***	***95% CI***	***p* – value***
	n	**%**	**n**	**%**			
Definite type B**	7	3.59	4	1.90	1.00		
Type B	50	25.64	75	35.55	0.38	0.11-1.37	0.14
Both A and B trait	123	63.08	124	58.77	0.57	0.16-1.98	0.38
Type A	15	7.69	8	3.79	1.07	0.24-4.79	0.93
Mean	44.11		41.82				
S.D.	14.17		13.10				

* Chi square test

** Reference group

2.6 Stress score

Most of cases and controls had moderate stress which were 46.43% and 52.83% respectively, while who had high stress in cases was 37.24% and controls was 32.08%. In cases who had severe stress was 11.73% and 4.59% was mild stress. For controls who had mild stress and severe stress were 7.55% equally. The crude analysis did not demonstrate significant association between stress and MetS (Table 4.15).

Table 4.15 Prevalence of stress among total study populations and crude association with metabolic syndrome.

Characteristics	Case (n=196)		Control (n=212)		*Crude OR*	*95% CI*	*p* – value*
	n	%	n	%			
Stress score							
Mild Stress**	9	4.59	16	7.55	1.00		
Moderate stress	91	46.43	112	52.83	1.44	0.61-3.42	0.40
high stress	73	37.24	68	32.08	1.91	0.79-4.60	0.15
Severe stress	23	11.73	16	7.55	2.56	0.91-7.20	0.08

* Chi square test

** Reference group

3. The multivariate analysis

After detection for collinearly of variables and interaction of variables, unconditional logistic regression analysis was used to demonstrate adjusted association among lifestyle variables and other variables with enter method. Stratification of analysis was performed in two models by gender which were model for male participants and model for female participants because of the differences in lifestyle factors.

Model for male participants.

Model consisted of the following variables:

Dependent variables: metabolic syndrome status (yes/no)

Independent variables: which were lifestyle factors, which were eating habit by frequency of intake of fruits and vegetable, and seafood (low intake / high intake), (low intake / high intake), exercise (regular/irregular), physical activity by types of job (labor/non labor), physical activity by types of daily traveling to work (mild/strong), smoking habit (non smoker/ex or current smoker), alcohol drinking (non or ex drinker/current drinker), Type A Behavior (yes/no), stress (mild and moderate stress/high and severe stress).

Confounding variables: which were having history of hypertension in family (yes/no), having history of DM in family (yes/no) and age(years).

Model for female participants.

Model consisted of the following variables:

Dependent variables: metabolic syndrome status (yes/no)

Independent variables: was lifestyle factors, which were frequency of intake of fruits and vegetable, and seafood (low intake / high intake), exercise (regular/irregular), physical activity by types of job (labor/non labor), physical activity by types of daily traveling to work (mild/strong), alcohol drinking (non or ex drinker/current drinker), Type A Behavior (yes/no), stress (mild and moderate stress/high and severe stress).

Confounding variables: which were having history of hypertension in family (yes/no), having history of DM in family (yes/no), menstruation status (yes/no) and age (years).

The dependent variable, independent variables and confounding factors were put into the same final model by enter method for simultaneously analysis. The results demonstrated the adjusted association between metabolic syndrome status with lifestyle factors by adjusted Odds ratios with 95%CI and p-value, and presented in the following tables Table 4.16 and 4.17.

3.1 Association between lifestyle factors and metabolic syndrome in male participants.

Among male participants, it was found that male participants who had occupation with light and moderate work were more likely to develop Metabolic syndrome 2.82 times (OR 2.82 95%CI 1.22-6.55) compared to male participants who had occupation in heavy work. While male participants who ate high intake of seafood were more likely to develop Metabolic syndrome 4.83 times (OR 4.83 95% CI 1.36-17.15) compared to male participants who ate low intake of seafood (Table 4.16).

Table 4.16 Adjusted association between lifestyle factors and other factor with metabolic syndrome in male participants.

Variable	Crude OR	95%CI	Adjusted OR	95%CI	p-value
Fruit and vegetable					
Low intake	1.15	0.60-2.19	1.22	0.57-2.58	0.61
High intake*	1.00		1.00		
Exercise					
Regular*	1.00		1.00		
Irregular	1.52	0.41-5.63	2.69	0.51-14.29	0.25
Physical activity by types of job					
Heavy*	1.00		1.00		
Light and moderate	2.31	1.10-4.84	2.85	1.24-6.56	0.01**
Smoking					
Non smoker*	1.00		1.00		
Ex or current smoker	0.87	0.46-1.67	0.57	0.25-1.27	0.17
Alcohol drinking					
Non or ex drinker*	1.00		1.00		
Current drinker	1.36	0.69-2.66	1.80	0.80-4.09	0.16
Type A Behavior					
Non type A*	1.00		1.00		
Type A	1.30	0.67-2.52	1.53	0.68-3.41	0.30
Stress					
Mild and moderate*	1.00		1.00		
High and severe	1.60	0.83-3.07	1.74	0.81-3.72	0.16
Seafood					
Low intake*	1.00		1.00		
High intake	4.91	1.56-15.52	4.83	1.36-17.15	0.02*
Age (year)	1..004	0.96-1.06	1.004	0.94-1.07	0.90

** Reference group* *** Significant risk*

*** adjusting for all variables in table including having history of hypertension in family, having history of DM in family.

3.2 Association between lifestyle factors and metabolic syndrome in female participants.

Among female participants, results from the analysis did not demonstrate any significant association among lifestyle factors and metabolic syndrome, but when age increased one year, they were more likely to have at least 3 from 5 criteria of metabolic syndrome (Table 4.17)**.**

Table 4.17 Adjusted association between lifestyle factors and other factors with metabolic syndrome in female participants.

Variable	Crude OR	95%CI	Adjusted OR	95%CI	p-value
Fruit and vegetable					
Low intake	1.42	0.86-2.34	1.64	0.91-2.97	1.00
High intake*	1.00		1.00		
Seafood					
Low intake	1.14	0.54-2.39	1.18	9.52-2.68	0.70
High intake*	1.00		1.00		
Physical activity by types of job					
Heavy*	1.00		1.00		
Light and moderate	1.24	0.75-2.06	1.14	0.66-1.95	0.64
Exercise					
Regular*	1.00		1.00		
Irregular	1.25	0.63-2.46	1.42	0.66-3.05	0.36
Alcohol drinking					
Non or ex drinker*	1.00		1.00		
Current drinker	1.58	0.62-4.08	1.92	0.68-5.45	0.22
Type A Behavior					
Non type A*	1.00		1.00		
Type A	1.60	0.93-2.76	1.75	0.96-3.18	0.07
Stress					
Mild and moderate*	1.00		1.00		
High and severe	1.39	0.85-2.27	1.51	0.88-2.57	0.13
Age (years)	1.06	1.02-1.10	1.05	1.01-1.10	0.02**

** Reference group* *** Significant risk*

*** adjusting for all variables in table including having history of hypertension in family and having history of DM in family member.

CHAPTER V
DISCUSSION

This study aimed to investigate unhealthy lifestyle factors associated with metabolic syndrome among population aged 35 to 60 years in Banphaeo district, Samutsakorn Province. The studied population who also participated in Routine Hypertension and Diabetes Screening Program (BRHDSP). This BRHDSP program has been implemented in this Banphaeo district for more than 3 years. Some of existing health promotion programs for modification of risk behavior for chronic diseases were also implemented such as exercise campaign, stroke screening, aging club, lifestyle modification package such as exercise festival, KAP modification, drug and smoking restriction both at workplace, community lifestyle intervention and etc. The results from this study was expected to be useful for monitoring, improvement of healthy lifestyle factors more specifically and to improve the effectiveness of existing the above health promotion programs.

1. Principle of the findings

The results of this study found that significant unhealthy lifestyle factors for metabolic syndrome in male which were physical activity by having heavy work occupation (OR 2.82; 95%CI 1.22-6.55) and high intake of seafood (OR 4.82, 95%CI 1.36-17.15). Among female participants, there was no significant unhealthy lifestyle factors found, but it was found that they were more likely to develop at least 3 from 5 criteria of metabolic syndrome by NCEP definition, when their age increased every single year.

2. Limitation of study

2.1 Study design and methodology

The population based case-control study was used to demonstrate associations between lifestyle factors and other factors associated with metabolic syndrome (MetS). Using case-control study for this study had weak points when compared to follow up study such as cohort study when the case-control study provided estimated odds ratio instead of comparing disease rates or risk.

Actually, follow up study as cohort study is usually the best study design in answering the etiologic question for lifestyle factors that produce MetS status by sequence of time in cause and effect. But because of limitation of time in follow up, this case-control study was used to determine the association between lifestyle factors and MetS instead.

The source population was people aged 35 to 60 years in Banphaeo district, Samutsakorn Province, but cases and controls were selected from people who enrolled in Banphaeo Routine Hypertension and Diabetes Screening Program (BRHDSP) during November 2007 to June 2008. The study population may not represent population of Banphaeo district. Because not all population aged 35 to 60 in Banphaeo district years participated in Routine Hypertension and Diabetes Screening Program (BRHDSP) during November 2007 to June 2008. The coverage rate was about 73.28% (23,691 of 32,330 population). Which means implication of the result to population of Banphaeo district may have some limitation.

2.2 Selection bias

Choices of selection of cases and control are usually major problem which have direct effect to size of odds ratio, especially in hospital based case-control study. In this study, population based case-control study was used, but some selection bias may exist. Cases and controls were selected from those who participated in BRHDSP which was screening program during period of November 2007 to June 2008. The duration of program did not cover entire year, some of people who did not enroll in BRHDSP may possessed interested potential lifestyle factors and may affect results of the study. Another selection bias was healthy worker effect, who enrolled BRHDSP usually had willingness to walk in to public health system and had more concern in

their health, while people who did not enrolled would have less concern on health. Other selection bias was cases and controls selected from participants who enrolled in BRHDSP, not selecting from the frame of total population database in the district. These may affected representativeness to total population of Banphaeo district.

2.3 Detection bias

Cases and controls group were selected by using 5 criterions of MetS from NCEP ATP III mainly from screening methods for diagnosis of hypertension and diabetes and from screening health center database. These methods of diagnosis were not considered as the standard methods of diagnosis, or it could be under standard of measurement screening such as blood pressure not measured after resting 5 minutes or fasting plasma glucose not fasting more than 8 hours. Also measuring of waist circumference may have different methods of measuring by using at umbilicus point as recommended by ministry of public health.

2.4 Recall bias

Recall bias is major weakness in cases-control study. Since in this study, the method of data collection for behavior or lifestyle factors in the past was from interviewing. Cases tended to try to think about unhealthy behavior in the previous 2-3 year more than controls, because they already had disease under study. Good behavior such as high frequency of exercise, eating fruit and vegetable were usually over reported. Nothing can be done to prevent this bias, but the results should be carefully used.

2.5 Interviewer bias

In this study, questionnaire was used for collecting information from participants by interviewers and could produce error in recording.

2.6 Sample size

Number of sample size was too small by the precision was 50%. It caused wide 95% confidence interval in some variables. It may also affected the analysis of odds ratio for some interesting unhealthy lifestyle variables.

3. Discussion in results

The results from these study demonstrated significant effect of lifestyle factors when controlled for genetic factors such as having HT, DM in family, hormonal factor, past history of some diseases, and then stratified by gender when analyzed. Significant factors from this study was physical activity by type of occupation and high intake of seafood in male participants. Among female participants, there was not found any significant lifestyle factors, but when age increased, they are more likely to develop metabolic syndromes .

Physical activity

The definition of physical activities in this study was measured by 2 types of physical activities which were physical activity by type of job and leisure time or exercise. In normal daily life, most of this study population (>50%) were agriculturist with nearly all day heavy working which considered as labor work. Results from this study found that physical activities by type of job in light work were more likely to develop metabolic syndrome 2.82 times compared those who worked as heavy work but not among female. This results was nearly the same as the results from others case-control study which showed significant effect compared to unemployed with blue collar with odds ratio of 2.0 (OR 2.0; 95% CI 1.4-2.8) in men and 1.7 (OR 1.7; 95%CI 1.0-2.7) in women [1]. The results from this study was also the same as the results from prospective study on physical activity which showed significant protective factor between physical activity over time compared with low activity with relative risk of 0.49 (RR 0.49; 95% CI 0.34-0.70). [2]

For exercise in leisure time, these study divided exercise into 2 groups; regular and non+irregular exercise. Regular exercise referred to having exercise 3 or more times per week and more than 30 minutes per time while irregular exercise referred to exercise less than 3 times per week and less than 30 minutes per time. Result from this study did not showed significant association of exercise to metabolic syndrome. When results from other studies showed that moderate exercise (2-3 sessions/week) was protective factors compared to low exercise (less than or equal to 1 session per week) (OR 0.6; 95% CI 0.5-0.9). [1]

However, this group of study population, most of cases and controls had no exercise in 85.20% and 81.60% and regular exercise were only only 10.71% and 13.68% in cases and controls respectively which might be because of lack of time from work and they might think that working on the job was equal value to exercise. This results recommended in perception strengthen exercises program, especially in workplace including other physical activity by daily walking to work place and back home.

Smoking status

The results from this study demonstrated prevalence of smoking were 12.76 % among cases, and 9.43% among controls which were quite low especially among female. The results did not demonstrated significant association between smoking and metabolic syndrome among them. There were the different results among studies. Other study found that current smoking associated with the metabolic syndrome regardless gender with OR 1.4 (95% CI 1.1-1.8) compared current smoker to non smoker in men [1].

Alcohol consumption

This study, alcohol consumption was divided into current drinker, ex-drinker and non drinker. But results did not demonstrated significant association between alcohol consumption and metabolic syndrome in men and women which because of most of cases and controls were non drinker which were 72.45% and 79.33% respectively.

Others studies, the results from cross sectional health survey showed light alcohol drinking (<15g/day) was protective factor for metabolic syndrome in women with an odds ratio of 0.8 (95% CI 0.7-0.9) [1].

Type A behavior

Type A behavior was determined by questionnaire and divided to type A and non type A behavior. The result from this study did not showed significant risk to metabolic syndrome in both male and female because of low proportion on type A in cases (7.69%) and controls (3.79%).

Others studies found that who had type A behavior had a moderating effect on the relationship between parameter of metabolic syndrome and the other study found relative risks of 0.99 in usual care, 0.81 in special intervention, and 0.87 overall (95% CI 0.59-1.28) [3, 4].

Stress

Stress status or mental health in this study, it was measured by using SPST-20 test. The results of test was classified to mild moderate high and severe stress and the result did not demonstrated association with metabolic syndrome in both male and female. It did not associated with MetS, but other study that showed chronic work stress more likely to have metabolic syndrome than those without work stress [5]

Dietary habit

Banphaeo district located near sea shore which have plenty of seafood. This study classified high intake of seafood as risk factors, but not included all kind of fish. As the lifestyle factors of eating habit, frequency of eating of seafood, and fruit and vegetable were classified into low intake and high intake. But both types of food did not show any significant association with metabolic syndrome among male and female in both crude and adjusted.

It was found that high intake of seafood more likely to develop MetS compared to low intake of seafood which was 4.82 times (OR 4.82 95% CI 1.36-17.15) in men. While in the other studies demonstrated that low intake crude fiber food was more likely to develop MetS 1.73 times compared to high intake of food. In cohort study, higher intake of dietary carbohydrates and lower intake of crude fiber were separately associated with an increased risk for metabolic syndrome (RR ranging from 1.3 to 1.9) [2].

Although, the prevalence of low intake of seafood were both high in 83.67% in case and 90.57% in controls, but cases had lower intake fruit and vegetable from and easily to access which was 55.61% and 59.91% respectively.

Age

In this present study, the results showed significant association between increasing of age and developing of MetS among female participants. These results implied that prevalence of MetS among women increased by age. Hormonal status can be used to explain this[6].

Menopause status

The frequency of metabolic syndrome usually increased from the time of the menopausal transition to the postmenopause. Abdominal obesity was the most frequent feature observed. In order to prevent cardiovascular disease, the metabolic syndrome must be evaluated from the time of the menopausal transition. Study in Korea report that postmenopausal status was associated with an increased risk of the metabolic syndrome independent of normal aging in Korean women. [86]

However, this study did not find the significant association of menopause status among female participant.

CHAPTER VI
CONCLUSION AND RECOMMENDATIONS

The metabolic syndrome is one of the major predisposing factors for CVD. This population based case-control study aimed to investigate the association between lifestyle factors and metabolic syndrome among population of Banphaeo district, Samutsakorn province. Data was collected during November 2007 to June 2008 . Cases and controls were selected from people who enrolled Banphaeo Routine Hypertension and Diabetes Screening Program (BRHDSP) and sampling by stratified random sampling with proportional to size of participants in each sub-district. There were 196 cases and 212 controls. Case and controls were age between 35-60 years old. The majority of case and controls were female, were couple, Buddhists, primary school level, agriculturists, personal income ≤ 5,000 bahts per month, had no family history of DM, HT and hypercholesterolemia and in female was not menopause status. The results from this study demonstrated both risk factor in male and female.

From the univariate analysis for association between lifestyle risk factors and metabolic syndrome. Significant factors in men were physical activity by type of job as non labor work compared to labor work and family history of DM in family while in female was not found any significant risk factors.

Final results from the unconditional logistic regression analysis was used to determine association between lifestyle factors to metabolic syndrome and controlling the effect of family history of hypertension, family history of hypercholesterolemia gender and age. The significant risk in men were still physical activity by type of occupation which was 2.82 (OR 2.82 95%CI 1.22-6.55) and high intake of seafood was 4.82 (OR 4.82 95% CI 1.36-17.15) while no significant risk in women.

By the results from this study, modificable risk factor in men was physical activity on working as a heavy work or most of time movements and low intake of seafoods. In women, there was no risk factor to develop metabolic syndrome found,

but prevalence of MetS increased when age increased, The need for specific good exercise programs to reduce prevalence of leisure time activity such as promote regular exercise at least 3 times per week, 30 minutes per time by intervene in workplace, community, pre-aging club or annual year exercise festival.

1. Recommendation for strengthening health program

Base on the finding of this study issues should be considered for prevention the development of metabolic syndrome by adjusting the modificable factor and thightly prevention on a person who are at risk.

1. Physical activity on daily working should be concern, men who did not work hard all day or office worker, rarely movement should be find the way to get more activity such as continuously exercise playing sports or working exercise.

2. High risk group such as men who aged 35 or above, women who going to become aging and being in transition of to menopause status should be carefully checking up for their health examination annually. Health promotion activities to reduce their waist circumference is necessary, such as exercise and controlling eating habits.

3. Government should promote exercise campaign continuously for choice of physical activity such as making sport areas, healthy community, healthy workplace

4. Lifestyle modification on high risk group of MetS such as people who possess one or two components of MetS to eliminate risk factors and prevention to MetS in the future.

2. Recommendation for further study

1. This study was case-control study that showed indirect approximation of risk to MetS not incidence rate, a follow up study such as cohort study which is the observational study should be performed to confirm the actually potential lifestyle factors, health promotion and disease prevention can be more specific and reduce the incidence of MetS in this population.

2. The further study might be considered to anothers definition of MetS such as from IDF, WHO to define the different ways of prevention.

3. The further study might be considered to screening program package of measurement method and health care worker practice.

4. The further study regarding to study population that can be represent to all population.

5. Increase sample size in order to increase the precision of study is recommended.

REFERENCES

1. Greenstone CL. The Metabolic Syndrome: A Lifestyle Medicine Foe Worthy of a Seek Am J Lifestyle Med. 2008;2((2)):109-12.
2. Torpy JM, Lynm C, Glass RM. The Metabolic Syndrome. 2006:850-.
3. Reaven GM. Role of insulin resistance in human disease. Diabetes. 1988;37:1595-607.
4. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection E, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Bethesda, MD: NCEP NHLBI NIH, 2001.
5. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 2001;285:2486.
6. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Circulation. 2005 Oct 25;112(17):2735-52.
7. American Heart Association I. Metabolic Syndrome. [cited; Available from: http://www.americanheart.org/presenter.jhtml?identifier=4756
8. M J. A dictionary of epidemiology: Oxford University Press, 1995.
9. Reaven GM. Diet and syndrome X. Atheroscler Rep. 2000;2(4):503-7.
10. Fung TT, Rimm EB, Spiegelman D, Rifai N, Tofler GH, Willett WC, et al. Association between dietary patterns and plasma biomarkers of obesity and cardiovascular disease risk. The American journal of clinical nutrition. 2001 Jan;73(1):61-7.
11. Ludwig DS, Pereira MA, Kroenke CH, Hilner JE, Van Horn L, Slattery ML, et al. Dietary fiber, weight gain, and cardiovascular disease risk factors in young adults. Jama. 1999 Oct 27;282(16):1539-46.

12. Pereira MA, Jacobs DR, Jr., Van Horn L, Slattery ML, Kartashov AI, Ludwig DS. Dairy consumption, obesity, and the insulin resistance syndrome in young adults: the CARDIA Study. Jama. 2002 Apr 24;287(16):2081-9.

13. Abbasi F, Brown BW, Jr., Lamendola C, McLaughlin T, Reaven GM. High carbohydrate diets, triglyceride-rich lipoproteins, and coronary heart disease risk. Journal of the American College of Cardiology. 2000 Sep 4;85(5):45-8.

14. Wirfalt E, Hedblad B, Gullberg B, Mattisson I, Andren C, Rosander U, et al. Food patterns and components of the metabolic syndrome in men and women: a cross-sectional study within the Malmo Diet and Cancer cohort. American journal of epidemiology. 2001 Dec 15;154(12):1150-9.

15. McAuley KA, Williams SM, Mann JI, Goulding A, Chisholm A, Wilson N, et al. Intensive lifestyle changes are necessary to improve insulin sensitivity: a randomized controlled trial. Diabetes care. 2002 Mar;25(3):445-52.

16. Meigs JB, D'Agostino RB, Sr., Wilson PW, Cupples LA, Nathan DM, Singer DE. Risk variable clustering in the insulin resistance syndrome. The Framingham Offspring Study. Diabetes. 1997 Oct;46(10):1594-600.

17. Aekplakorn W, Abbott-Klafter J, Khonputsa P, Tatsanavivat P, Chongsuvivatwong V, Chariyalertsak S, et al. Prevalence and management of prehypertension and hypertension by geographic regions of Thailand: the Third National Health Examination Survey, 2004. Journal of hypertension. 2008 Feb;26(2):191-8.

18. Aekplakorn W, Abbott-Klafter J, Premgamone A, Dhanamun B, Chaikittiporn C, Chongsuvivatwong V, et al. Prevalence and management of diabetes and associated risk factors by regions of Thailand: Third National Health Examination Survey 2004. Diabetes care. 2007 Aug;30(8):2007-12.

19. Aekplakorn W, Chaiyapong Y, Neal B, Chariyalertsak S, Kunanusont C, Phoolcharoen W, et al. Prevalence and determinants of overweight and obesity in Thai adults: results of the Second National Health Examination Survey. Journal of the Medical Association of Thailand = Chotmaihet thangphaet. 2004 Jun;87(6):685-93.

20. Boonyavarakul A, Choosaeng C, Supasyndh O, Panichkul S. Prevalence of the metabolic syndrome, and its association factors between percentage body fat and body mass index in rural Thai population aged 35 years and older. Journal of the Medical Association of Thailand = Chotmaihet thangphaet. 2005 Nov;88 Suppl 3:S121-30.

21. Pongchaiyakul C, Nguyen TV, Wanothayaroj E, Karusan N, Klungboonkrong V. Prevalence of metabolic syndrome and its relationship to weight in the Thai population. Journal of the Medical Association of Thailand = Chotmaihet thangphaet. 2007 Mar;90(3):459-67.

22. Lohsoonthorn V, Dhanamun B, Williams MA. Prevalence of metabolic syndrome and its relationship to white blood cell count in a population of Thai men and women receiving routine health examinations. Am J Hypertens. 2006 Apr;19(4):339-45.

23. Pongchaiyakul C, Pongchaiyakul C, Pratipanawatr T. Prevalence of dyslipidemia in rural Thai adults: an epidemiologic study in Khon Kaen province. Journal of the Medical Association of Thailand = Chotmaihet thangphaet. 2005 Aug;88(8):1092-7.

24. Chongsuvivatwong V, YipIntsoi T, Apakupakul N. Gender and ethnic differences in cardiovascular risks in Songkhla province, Thailand: the interASIA-south. Journal of the Medical Association of Thailand = Chotmaihet thangphaet. 2008 Apr;91(4):464-70.

25. Srisala K. Prevalence of the metabolic syndrome using the NCEP ATP III, WHO and IDF definitions in personel in the cardiovascular health program at the electricity generating authority of Thailand. 2007.

26. health Dom. Report of Mental Health survey.; 2008.

27. Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome: the Atherosclerosis Risk in Communities study. Circulation. 2008 Feb 12;117(6):754-61.

28. (NSO) NSO. Conclusion of Smoking and Drinking Behavior: National Statistical Office (NSO); 2008.

29. Hospital B. Report of Non-communicable disease year 2007; 2007.

30. Nagele U, Hagele EO, Sauer G, Wiedemann E, Lehmann P, Wahlefeld AW, et al. Reagent for the enzymatic determination of serum total triglycerides with improved lipolytic efficiency. Journal of clinical chemistry and clinical biochemistry. 1984 Feb;22(2):165-74.

31. Warnick GR, Benderson J, Albers JJ. Dextran sulfate-Mg2+ precipitation procedure for quantitation of high-density-lipoprotein cholesterol. Clinical chemistry. 1982 Jun;28(6):1379-88.

32. Katsouyanni K, Rimm EB, Gnardellis C, Trichopoulos D, Polychronopoulos E, Trichopoulou A. Reproducibility and relative validity of an extensive semi-quantitative food frequency questionnaire using dietary records and biochemical markers among Greek schoolteachers. Int J Epidemiol. 1997;26 Suppl 1:S118-27.

33. Physical Activity for Everyone: Glossary of Terms. 2008 [cited 2/8/2008]; Available from: http://www.cdc.gov/nccdphp/dnpa/physical/everyone/glossary/index.htm

34. DMH M. suanprung stress test. 2010 [cited 19/12/10]; Available from: http://203.157.56.1/test/stress/

35. Wikipedia. Education in Thailand. 2008 [cited 27 July 2008]; Available from: en.wikipedia.org/wiki/Education_in_Thailand

36. Soules MR, Sherman S, Parrott E, Rebar R, Santoro N, Utian W, et al. Executive summary: Stages of Reproductive Aging Workshop (STRAW). Fertility and sterility. 2001 Nov;76(5):874-8.

37. Cameron AJ, Shaw JE, Zimmet PZ. The metabolic syndrome: prevalence in worldwide populations. Endocrinology and metabolism clinics of North America. 2004 Jun;33(2):351-75, table of contents.

38. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet. 2005 Apr 16-22;365(9468):1415-28.

39. Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. Diabet Med. 1998 Jul;15(7):539-53.

40. Einhorn D, Reaven GM, Cobin RH, Ford E, Ganda OP, Handelsman Y, et al. American College of Endocrinology position statement on the insulin resistance syndrome. Endocr Pract. 2003 May-Jun;9(3):237-52.
41. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). Jama. 2001 May 16;285(19):2486-97.
42. Reaven GM. Insulin resistance, cardiovascular disease, and the metabolic syndrome: how well do the emperor's clothes fit? Diabetes care. 2004 Apr;27(4):1011-2.
43. Wikipedia. International Diabetes Federation - Wikipedia, the free encyclopedia. 2008 [cited 29/07/08]; Available from: http://en.wikipedia.org/wiki/International_Diabetes_Federation
44. Wikipedia. National Cholesterol Education Program - Wikipedia, the free encyclopedia. 2008 [cited 29/07/08]; Available from: http://en.wikipedia.org/wiki/National_Cholesterol_Education_Program
45. Wikipedia. Metabolic syndrome - Wikipedia, the free encyclopedia. 2008 [cited 29/07/08]; Available from: http://en.wikipedia.org/wiki/Metabolic_syndrome#NCEP
46. Grundy SM, Brewer HB, Jr., Cleeman JI, Smith SC, Jr., Lenfant C. Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. Circulation. 2004 Jan 27;109(3):433-8.
47. Ferrannini E, Haffner SM, Mitchell BD, Stern MP. Hyperinsulinaemia: the key feature of a cardiovascular and metabolic syndrome. Diabetologia. 1991 Jun;34(6):416-22.
48. Reaven GM. Role of insulin resistance in human disease (syndrome X): an expanded definition. Annual review of medicine. 1993;44:121-31.
49. Abbasi F, Brown BW, Jr., Lamendola C, McLaughlin T, Reaven GM. Relationship between obesity, insulin resistance, and coronary heart disease risk. Journal of the American College of Cardiology. 2002 Sep 4;40(5):937-43.

50. Lewis GF, Uffelman KD, Szeto LW, Weller B, Steiner G. Interaction between free fatty acids and insulin in the acute control of very low density lipoprotein production in humans. The Journal of clinical investigation. 1995 Jan;95(1):158-66.

51. Lewis GF, Steiner G. Acute effects of insulin in the control of VLDL production in humans. Implications for the insulin-resistant state. Diabetes care. 1996 Apr;19(4):390-3.

52. Taghibiglou C, Rashid-Kolvear F, Van Iderstine SC, Le-Tien H, Fantus IG, Lewis GF, et al. Hepatic very low density lipoprotein-ApoB overproduction is associated with attenuated hepatic insulin signaling and overexpression of protein-tyrosine phosphatase 1B in a fructose-fed hamster model of insulin resistance. The Journal of biological chemistry. 2002 Jan 4;277(1):793-803.

53. Shimomura I, Bashmakov Y, Ikemoto S, Horton JD, Brown MS, Goldstein JL. Insulin selectively increases SREBP-1c mRNA in the livers of rats with streptozotocin-induced diabetes. Proceedings of the National Academy of Sciences of the United States of America. 1999 Nov 23;96(24):13656-61.

54. Eckel RH, Yost TJ, Jensen DR. Alterations in lipoprotein lipase in insulin resistance. Int J Obes Relat Metab Disord. 1995 May;19 Suppl 1:S16-21.

55. Murakami T, Michelagnoli S, Longhi R, Gianfranceschi G, Pazzucconi F, Calabresi L, et al. Triglycerides are major determinants of cholesterol esterification/transfer and HDL remodeling in human plasma. Arteriosclerosis, thrombosis, and vascular biology. 1995 Nov;15(11):1819-28.

56. Brinton EA, Eisenberg S, Breslow JL. Increased apo A-I and apo A-II fractional catabolic rate in patients with low high density lipoprotein-cholesterol levels with or without hypertriglyceridemia. The Journal of clinical investigation. 1991 Feb;87(2):536-44.

57. de Graaf J, Hendriks JC, Demacker PN, Stalenhoef AF. Identification of multiple dense LDL subfractions with enhanced susceptibility to in vitro oxidation among hypertriglyceridemic subjects. Normalization after clofibrate treatment. Arterioscler Thromb. 1993 May;13(5):712-9.

58. Manzato E, Zambon S, Zambon A, Cortella A, Sartore G, Crepaldi G. Levels and physicochemical properties of lipoprotein subclasses in moderate hypertriglyceridemia. Clinica chimica acta; international journal of clinical chemistry. 1993 Oct 15;219(1-2):57-65.

59. Halle M, Berg A, Baumstark MW, Konig D, Huonker M, Keul J. Influence of mild to moderately elevated triglycerides on low density lipoprotein subfraction concentration and composition in healthy men with low high density lipoprotein cholesterol levels. Atherosclerosis. 1999 Mar;143(1):185-92.

60. Kwiterovich PO, Jr. Clinical relevance of the biochemical, metabolic, and genetic factors that influence low-density lipoprotein heterogeneity. The American journal of cardiology. 2002 Oct 17;90(8A):30i-47i.

61. Krauss RM. Dense low density lipoproteins and coronary artery disease. The American journal of cardiology. 1995 Feb 23;75(6):53B-7B.

62. Packard CJ. LDL subfractions and atherogenicity: an hypothesis from the University of Glasgow. Current medical research and opinion. 1996;13(7):379-90.

63. Lada AT, Rudel LL. Associations of low density lipoprotein particle composition with atherogenicity. Current opinion in lipidology. 2004 Feb;15(1):19-24.

64. Zambon A, Hokanson JE, Brown BG, Brunzell JD. Evidence for a new pathophysiological mechanism for coronary artery disease regression: hepatic lipase-mediated changes in LDL density. Circulation. 1999 Apr 20;99(15):1959-64.

65. Sacks FM, Campos H. Clinical review 163: Cardiovascular endocrinology: Low-density lipoprotein size and cardiovascular disease: a reappraisal. The Journal of clinical endocrinology and metabolism. 2003 Oct;88(10):4525-32.

66. Lee Y, Hirose H, Ohneda M, Johnson JH, McGarry JD, Unger RH. Beta-cell lipotoxicity in the pathogenesis of non-insulin-dependent diabetes mellitus of obese rats: impairment in adipocyte-beta-cell relationships. Proceedings of the National Academy of Sciences of the United States of America. 1994 Nov 8;91(23):10878-82.

67. Boucher A, Lu D, Burgess SC, Telemaque-Potts S, Jensen MV, Mulder H, et al. Biochemical mechanism of lipid-induced impairment of glucose-stimulated insulin secretion and reversal with a malate analogue. The Journal of biological chemistry. 2004 Jun 25;279(26):27263-71.

68. Joseph JW, Koshkin V, Saleh MC, Sivitz WI, Zhang CY, Lowell BB, et al. Free fatty acid-induced beta-cell defects are dependent on uncoupling protein 2 expression. The Journal of biological chemistry. 2004 Dec 3;279(49):51049-56.

69. Yaney GC, Corkey BE. Fatty acid metabolism and insulin secretion in pancreatic beta cells. Diabetologia. 2003 Oct;46(10):1297-312.

70. Bruning JC, Michael MD, Winnay JN, Hayashi T, Horsch D, Accili D, et al. A muscle-specific insulin receptor knockout exhibits features of the metabolic syndrome of NIDDM without altering glucose tolerance. Molecular cell. 1998 Nov;2(5):559-69.

71. Kulkarni RN, Bruning JC, Winnay JN, Postic C, Magnuson MA, Kahn CR. Tissue-specific knockout of the insulin receptor in pancreatic beta cells creates an insulin secretory defect similar to that in type 2 diabetes. Cell. 1999 Feb 5;96(3):329-39.

72. Ferrannini E, Buzzigoli G, Bonadonna R, Giorico MA, Oleggini M, Graziadei L, et al. Insulin resistance in essential hypertension. The New England journal of medicine. 1987 Aug 6;317(6):350-7.

73. Steinberg HO, Brechtel G, Johnson A, Fineberg N, Baron AD. Insulin-mediated skeletal muscle vasodilation is nitric oxide dependent. A novel action of insulin to increase nitric oxide release. The Journal of clinical investigation. 1994 Sep;94(3):1172-9.

74. DeFronzo RA, Cooke CR, Andres R, Faloona GR, Davis PJ. The effect of insulin on renal handling of sodium, potassium, calcium, and phosphate in man. The Journal of clinical investigation. 1975 Apr;55(4):845-55.

75. Anderson EA, Hoffman RP, Balon TW, Sinkey CA, Mark AL. Hyperinsulinemia produces both sympathetic neural activation and vasodilation in normal humans. The Journal of clinical investigation. 1991 Jun;87(6):2246-52.

76. Egan BM. Insulin resistance and the sympathetic nervous system. Current hypertension reports. 2003 Jun;5(3):247-54.

77. Hanley AJ, Karter AJ, Festa A, D'Agostino R, Jr., Wagenknecht LE, Savage P, et al. Factor analysis of metabolic syndrome using directly measured insulin sensitivity: The Insulin Resistance Atherosclerosis Study. Diabetes. 2002 Aug;51(8):2642-7.

78. Nakagawa T, Hu H, Zharikov S, Tuttle KR, Short RA, Glushakova O, et al. A causal role for uric acid in fructose-induced metabolic syndrome. American journal of physiology. 2006 Mar;290(3):F625-31.

79. Hallfrisch J. Metabolic effects of dietary fructose. Faseb J. 1990 Jun;4(9):2652-60.

80. Reiser S, Powell AS, Scholfield DJ, Panda P, Ellwood KC, Canary JJ. Blood lipids, lipoproteins, apoproteins, and uric acid in men fed diets containing fructose or high-amylose cornstarch. The American journal of clinical nutrition. 1989 May;49(5):832-9.

81. Mayoclinic.com. Metabolic syndrome Risk factors - Mayo Clinic.com. 2008 [cited 2/8/2008]; Available from: http://mayoclinic.com/health/metabolic%20syndrome/DS00522/DSECTION=risk-factors

82. Shah T, Jonnalagadda SS, Kicklighter JR, Diwan S, Hopkins BL. Prevalence of metabolic syndrome risk factors among young adult Asian Indians. Journal of immigrant health. 2005 Apr;7(2):117-26.

83. Hye Soon Park, Sang Woo Oh, Sung-Il Cho, Woong Hwan Choi, Kim YS. The metabolic syndrome and associated lifestyle factors among South Korean adults. International Journal of Epidemiology. 2004;33:328-36.

84. Azadbakht L, Mirmiran P, Esmaillzadeh A, Azizi F. Dairy consumption is inversely associated with the prevalence of the metabolic syndrome in Tehranian adults. The American journal of clinical nutrition. 2005 Sep;82(3):523-30.

85. MERCEDES R.CARNETHON SS, CATHERINE M.LORIA, PETER J.SAVAGE, JAMES O.HILL, KIANG LIU. Risk Factors for the Metabolic Syndrome. Diabetes care. 2004; 27(11):2707–15.

86. Ford. ES, Harold W. Kohl III, Alo H. Mokdad, Ajami. UA. Sedentary Behavior, Physical Activity, and the Metabolic syndrome among U.S. Adults. Obesity research. 2005;13(3):608-14.

87. Estanislao Ramrez-Vargas, Maria del Rosario Arnaud-Vinas, Dellsle. H. Prevalence of the metabolic syndrome and associated lifestyles in adult males from Oaxaca, Mexico. salud publica de mexico. 2007;49(2):94-102.

88. Demosthenes B. Panagiotakos, Christos Pitsavos, Skoumas. Y. The association between Food patterns and the metabolic syndrome using principal components analysis: The ATTICA study. American Dietetic Association. 2007;107(6):979-87.

89. Hee Man kim, So Yeon Ryu, Jong Park, kim. J. The effect of menopause on the metabolic syndrome among Korean women. Diabetes care. 2007;30(3):701-6.

90. Yeong Sook Yoon, Sang Woo Oh, Hyun Wook Baik, Hye Soon Park, Kim. WY. Alcohol consumption and the metabolic syndrome in Korean adults: the 1998 Korean Health and Nutrition Examination. The American journal of clinical nutrition. 2004;80:217-24.

91. Shekelle RB, Hulley SB, Neaton JD, Billings JH, Borhani NO, Gerace TA, et al. The MRFIT behavior pattern study. II. Type A behavior and incidence of coronary heart disease. American journal of epidemiology. 1985 Oct;122(4):559-70.

92. Tarani Chandola, Eric Brunner, Marmot. M. Chronic stress at work and the metabolic syndrome: prospective study. BMJ. 2006.

93. Ravaja N, Keltikangas-Jarvinen L, Viikari J. Type A behavior and metabolic syndrome precursors in young adults. Journal of clinical epidemiology. 1996 Mar;49(3):335-43.

94. Kim HM, Park J, Ryu SY, Kim J. The effect of menopause on the metabolic syndrome among Korean women: the Korean National Health and Nutrition Examination Survey, 2001. Diabetes care. 2007 Mar;30(3):701-6.

APPENDICES

APPENDIX A

โครงการศึกษาวิถีชีวิตและโรคอ้วนลงพุง
ในประชากรอำเภอบ้านแพ้ว จังหวัดสมุทรสาคร
แบบเก็บข้อมูล

เรียนท่านผู้เข้าร่วมการศึกษา

แบบเก็บข้อมูลนี้ประกอบด้วยข้อถามเกี่ยวข้องกับวิถีชีวิตและการดำรงชีวิตประจำวันของท่านในช่วง 2-3 ปีที่ผ่านมา ได้แก่ การออกกำลังกาย การบริโภคอาหาร การดื่มแอลกอฮอล์ การสูบบุหรี่ รวมทั้ง แบบประเมินบุคลิกภาพ แบบประเมินความเครียด โดยผู้วิจัยจะนำไปศึกษาความสัมพันธ์กับการมีหรือไม่มีโรคอ้วนลงพุง โดยจะขอใช้ข้อมูลที่ท่านได้รับการตรวจร่างกายจากการเข้าร่วมในโครงการคัดกรองโรคความดันโลหิตสูงและเบาหวาน สำหรับประชากรอำเภอบ้านแพ้ว จังหวัดสมุทรสาคร โดยข้อมูลที่ได้จะนำไปใช้ประกอบเป็นแนวทางในการวางแผนป้องกันโรคอ้วนลงพุงและโครงการด้านการสร้างเสริมสุขภาพในอำเภอบ้านแพ้ว จังหวัดสมุทรสาคร เพื่อให้เกิดประโยชน์สูงสุดแก่ประชากรชาวบ้านแพ้วต่อไป

ข้อมูลมีทั้งหมด 6 ส่วน
โดยขอให้ท่านช่วยตอบคำถามในส่วนที่ 1, 2, 3, 4 และ 5 ดังนี้
ส่วนที่ 1 ข้อมูลทั่วไป
ส่วนที่ 2 ข้อมูลพฤติกรรมในอดีต
ส่วนที่ 3 รูปแบบการรับประทานอาหารในอดีต
ส่วนที่ 4 แบบวัดบุคลิกภาพ (Type A Behavior)
ส่วนที่ 5 แบบสอบวัดความเครียด (Suanprung Stress Test)
ส่วนที่ 6 เป็นข้อมูลประวัติทางด้านการแพทย์ของท่านที่ผู้วิจัยจะขออนุญาตคัดลอกจากแบบคัดกรองโรคความดันโลหิตสูงและเบาหวานจากของท่านจากการเข้าร่วมในโครงการคัดกรองโรคความดันโลหิตสูงและเบาหวาน อำเภอบ้านแพ้ว จังหวัดสมุทรสาคร ปี 2551 ซึ่งได้แก่ ผลการวินิจฉัยของแพทย์ถึงการเป็นหรือไม่เป็นโรคความดันโลหิตสูง โรคเบาหวาน ของท่าน น้ำหนักและส่วนสูง และผลการตรวจเลือดจากห้องปฏิบัติการ

<u>ขอให้ท่านนึกถึงคำตอบที่ใกล้เคียงความจริงกับตัวท่านมากที่สุด ที่ท่านได้ปฏิบัติในช่วง 2-3 ปีที่ผ่านมาแล้วจึงให้คำตอบหรือเลือกคำตอบ</u>

ผู้วิจัยขอรับรองว่าคำตอบที่ได้จากท่านจะถูกเก็บเป็นความลับ ไม่ถูกนำมาเปิดเผย โดยผลการวิจัยจะถูกนำเสนอในภาพรวม

ขอขอบพระคุณเป็นอย่างสูงที่ท่านสละเวลาในการตอบแบบเก็บข้อมูลฉบับนี้

นายนิติกร ภู่สุวรรณ ผู้วิจัย

<u>แบบเก็บข้อมูล</u>

“วิถีชีวิตและโรคอ้วนลงพุงในประชาชนอำเภอบ้านแพ้ว จังหวัดสมุทรสาคร”

เลขที่แบบสัมภาษณ์.....

( ) กลุ่มศึกษา ( ) กลุ่มเปรียบเทียบ

วัน/เดือน/ปีที่สัมภาษณ์.....

(สำหรับผู้วิจัย)

<u>ส่วนที่ 1 ข้อมูลทั่วไป</u>

1.1 อายุเต็มในปัจจุบันของท่าน.....ปี (วัน เดือน ปี เกิด ..... /……. /……..)

1.2 เพศของท่าน ( ) 1.ชาย ( ) 2. หญิง

1.3 สถานภาพสมรส

( ) 1. โสด ( ) 2. คู่ ( ) 3. หม้าย ( ) 4. หย่า/แยก

1.4 ท่านนับถือศาสนาใด

( ) 1. พุทธ ( ) 2. คริสต์ ( ) 3.อิสลาม ( ) 4. อื่น ๆ ระบุ .....

1.5 ที่อยู่ เลขที่.....หมู่ที่.....ตำบล.....อำเภอบ้านแพ้ว จังหวัดสมุทรสาคร

1.6 ระดับการศึกษาสูงสุดของท่าน

( ) 1. ไม่ได้เรียน ( ) 2. ประถมศึกษา ( ) 3. มัธยมศึกษาตอนต้น

( ) 4. มัธยมศึกษาตอนปลาย/ปวช./ปวส. ( ) 5. ปริญญาตรี

( ) 6. สูงกว่าปริญญาตรี

1.7 อาชีพหลักของท่าน ได้แก่

( ) 1. เกษตรกร ( ) 2. รับราชการ/รัฐวิสาหกิจ ( ) 3. รับจ้าง (ใช้แรงงาน)

( ) 4. รับจ้างทำงานบ้าน ( ) 5. รับจ้าง(งานสำนักงาน) ( ) 6. ค้าขาย/ธุรกิจส่วนตัว

( ) 7. ไม่ได้ทำงานอยู่บ้านเฉยๆ ( ) 8. อื่นๆ ระบุ.....

1.8 ท่านมีรายได้ของท่านต่อเดือนหรือไม่

( ) มี ยังไม่หักรายจ่าย (ระบุ ประมาณ).....บาท ( ) ไม่มีรายได้

1.9 ในครอบครัวของท่าน ได้แก่ บิดา มารดา พี่น้อง และบุตร มีใครเป็นโรคความดันโลหิตสูงหรือไม่

( ) 1 ไม่มี ( ) 2 มี ผู้ป่วยเป็น

( ) 2.1 บิดา ( ) 2.2 มารดา

( ) 2.3 พี่/น้อง ( ) 2.4 บุตร

1.10 ในครอบครัวของท่านได้แก่ บิดา มารดา พี่น้อง และบุตร มีใครเป็นโรคเบาหวานหรือไม่

( ) 1 ไม่มี ( ) 2 มี ผู้ป่วยเป็น

( ) 2.1 บิดา ( ) 2.2 มารดา

( ) 2.3 พี่/น้อง ( ) 2.4 บุตร

1.11 ในครอบครัวของท่านได้แก่ บิดา มารดา พี่น้อง และบุตร มีใครเป็นกลุ่มโรคไขมันในเลือดสูงหรือไม่

( ) 1 ไม่มี ( ) 2 มี ผู้ป่วยเป็น

( ) 2.1 บิดา ( ) 2.2 มารดา

( ) 2.3 พี่/น้อง ( ) 2.4 บุตร

1.12 ท่านเคยได้รับการวินิจฉัยหรือได้รับการบอก (จากแพทย์/บุคลากรหรือเจ้าหน้าที่สาธารณสุข) ว่าเป็นโรคต่อไปนี้หรือไม่

( ) 1. ไม่เคยตรวจ หรือ ไม่มีโรคดังกล่าว

( ) 2. เบาหวาน เมื่อ เดือน.....ปี.....

( ) 3. ความดันโลหิตสูง เมื่อ เดือน.....ปี.....

( ) 4. ไขมันในเลือดสูง เมื่อ เดือน.....ปี.....

( ) 5. โรคอัมพฤกษ์ อัมพาต เมื่อ เดือน.....ปี.....

( ) 6. โรคหัวใจ เมื่อ เดือน.....ปี.....

( ) 7. โรคประจำตัวอื่นๆ ระบุ ……เมื่อ เดือน.....ปี.....

1.13 สำหรับผู้ตอบแบบสอบถามเพศหญิง

1.13.1 ขณะนี้ท่านยังอยู่ในวัยที่มีประจำเดือน

( ) ใช่ ( ) ไม่ใช่ หมดแล้ว หมดมาแล้ว.....ปี .....เดือน

ส่วนที่ 2 ข้อมูลด้านพฤติกรรมส่วนบุคคลในอดีต

2.1 การสูบบุหรี่ใน 2-3 ปีที่ผ่านมา

2.1.1 ท่านสูบบุหรี่หรือไม่

( ) 1. ไม่สูบ และไม่เคยสูบเลยตลอดช่วงชีวิต (ข้ามไปข้อ 2.2)

( ) 2. เคยสูบ แต่ปัจจุบันเลิกสูบแล้ว

เริ่มสูบบุหรี่ ปี พ.ศ. .....

เลิกสูบมาได้.....ปีแล้ว เมื่ออายุ .....ปี

( ) 3. เคยสูบ และตอนนี้ยังสูบอยู่

เริ่มสูบบุหรี่ ปี พ.ศ. .....

2.1.3 ปกติท่านสูบบุหรี่หรือเคยสูบบุหรี่วันละกี่มวน โดยประมาณ……..มวนต่อวัน

2.1.4 ชนิดบุหรี่ที่เคยสูบหรือสูบอยู่

( ) 1. มีก้นกรอง ยี่ห้อ ..... ( ) 2. ไม่มีก้นกรอง ( ) 3. มวนเอง

2.2. การดื่มสุราในช่วง 2-3 ปีที่ผ่านมา

2.2.1. ท่านดื่มแอลกอฮอล์หรือไม่

( ) 1. ไม่เคยดื่มเลยในช่วงชีวิตที่ผ่านมา (ข้ามไปข้อ 2.3)

( ) 2. เคยดื่ม ตอนนี้เลิกแล้ว เลิกดื่มเมื่อ อายุ……ปี

( ) 3. ตอนนี้ยังดื่มอยู่

2.2.2 ส่วนใหญ่ ท่านดื่มเครื่องดื่มแอลกอฮอล์ชนิดใดและอย่างไรบ้าง**ใน 2-3 ปีที่ผ่านมา**

ประเภทเครื่องดื่ม แอลกอฮอล์	จำนวนวันที่ดื่ม ในแต่ละสัปดาห์ (< 1,1,2,…,7)	ปริมาณที่ดื่มต่อครั้ง (จอก/แก้ว/กระป๋อง) (1,2,3…..,9,10,11)
1.สุรากลั่นพื้นบ้าน(จอก)		
2.สุราผสม(แก้ว) เช่น หงษ์ทอง หงษ์ไท แม่โขง		
3.เบียร์ ยี่ห้อ.....(แก้ว)		
4. สุราสรรพสามิต		
5.วิสกี้/บรั่นดี (แก้ว)		
6.ไวน์ (แก้ว)		
7. อื่น ๆ ระบุ.....		

2.2.3 ปริมาณหรือขนาด**<u>ในการดื่มแต่ละครั้ง</u>** ท่านเคยดื่มลักษณะนี้หรือไม่ และอย่างไร

ลักษณะการดื่มแต่ละครั้ง	ไม่เคยดื่มเลย	เคยดื่ม	(จำนวนครั้งต่อเดือน) (< 1,1,2,3,….31)
เหล้าผสมโซดาตั้งแต่ 8 แก้ว หรือ 8 เป๊กขึ้นไป			
เหล้าขาว 40 ดีกรีตั้งแต่ครึ่งขวดขึ้นไป			
เหล้าขาว 28 ดีกรี ตั้งแต่ครึ่งขวดขึ้นไป			
เบียร์ตั้งแต่ 4 กระป๋องหรือขวดใหญ่ 2 ขวดครึ่ง ขึ้นไป			
ไวน์ หรือ แชมเปญ ตั้งแต่ 7 แก้วขึ้นไป			
น้ำตาลเมา สาโท กระแช่ ขนาดเท่าขวดเบียร์ขนาดใหญ่ 630 ซีซี ตั้งแต่ขวดครึ่งขึ้นไป			
ไวน์คูลเลอร์ หรือเหล้าผสมผลไม้ ตั้งแต่ 5 ขวด ขึ้นไป			

<u>2.3 ลักษณะของการเคลื่อนไหวร่างกาย (Physical activity)</u>

<u>2.3.1 การเคลื่อนไหวจากการทำงาน ให้ท่านบอกลักษณะการทำงาน ใน 2-3 ปี ที่ผ่านมา</u>

2.3.1.1. <u>การเคลื่อนไหวจากงาน</u>

ระบุงานที่ท่านทำเป็นประจำ.....

วิธีการเดินทางไปสถานที่ทำงาน ( ) ด้วยการเดิน ( ) ขี่จักรยาน

( ) ซ้อน/ขับจักรยานยนต์ ( ) นั่ง/ขับรถยนต์ ( ) อื่นๆ ระบุ

.....

ลักษณะ<u>งานที่ท่านทำประจำ</u> (อาจเลือกได้มากกว่า 1 ข้อ) คือ

( ) 1. ไม่ได้ทำงานนอกบ้าน ทำงานแม่บ้าน ทำงานบ้าน

( ) 2. นั่งขายของ บางทีก็เดินไปเดินมา

( ) 3. ทำงานในสำนักงาน / บริษัท / นั่งโต๊ะทำงานเป็นส่วนมาก

( ) 4. เป็นงานที่ต้องยืนหรือเดินต่อเนื่องเกือบทั้งวันเป็นส่วนใหญ่ เช่น ขายของ ค้าขาย

( ) 5. เป็นงานที่ต้องยืน/เดินต่อเนื่องแต่ไม่ทั้งวัน เช่นเฉพาะตอนเช้าที่ตลาด ขายของ ค้าขาย

( ) 6. งานที่ต้องใช้แรงงาน เช่น งานก่อสร้าง เกษตรกรรม ยกของหนัก ขนของหนัก

( ) 7. อื่นๆระบุ……

2.3.2 <u>การเคลื่อนไหวจากกิจกรรมนอกเวลาทำงานที่ทำบ่อยๆ</u>

เมื่อท่านเสร็จจากการทำงานหรือกิจกรรมการทำงานประจำในวันหยุดของท่าน ท่านมักจะใช้เวลาว่างทำอะไรบ้างทางกิจกรรมต่อไปนี้ ได้แก่

<u>นั่งๆ นอนๆ เพื่อพักผ่อน</u>

( ) 1. ท่านมักจะนั่งๆนอนๆเป็นส่วนมาก เช่น ดูโทรทัศน์/อ่านหนังสือ คุยโทรศัพท์กับเพื่อน เป็นต้น

( ) 2. มักจะนั่งๆนอนๆพักผ่อนเช่น ดูโทรทัศน์/อ่านหนังสือ **<u>แต่</u>** ก็ออกกำลังกายบ้างแต่ไม่เป็นประจำ

ประมาณ.....วัน ต่อสัปดาห์

ครั้งละประมาณ.....นาที

ระบุวิธีการออกกำลังกายของท่าน.....

<u>ออกกำลังกาย</u>

( ) 3. ท่านออกกำลังกายหรือเล่นกีฬาอะไรเป็นประจำ

( ) เดินช้า/รำไทเก็ก จำนวน.....วัน/สัปดาห์ ครั้งละ .....นาที

( ) เดินเร็ว/วิ่งปานกลาง จำนวน.....วัน /สัปดาห์ ครั้งละ .....นาที

( ) ขี่จักรยาน/วิ่งเหยาะ ๆ จำนวน..... วัน /สัปดาห์ ครั้งละ .....นาที

( ) แอโรบิค จำนวน..... วัน/สัปดาห์ ครั้งละ .....นาที

( ) วิ่งระยะไกล/ว่ายน้ำ/จักรยานระยะไกล จำนวน....วัน /สัปดาห์ ครั้งละ .....นาที

( ) อื่นๆระบุ.....จำนวน.....วัน /สัปดาห์ ครั้งละ .....นาที

( ) 4. ไม่เคยออกกำลังกายเลย

( ) 5. ทำงานอดิเรก คือ .....

( ) 5. ท่านมักจะหาเวลานอนพักและหลับหลังอาหารกลางวันเสมอ

( ) ทุกครั้ง ( ) ส่วนมาก ( ) บางครั้ง ( ) ไม่เคยนอน

ส่วนที่ 3 พฤติกรรมการบริโภคอาหาร

3.1 พฤติกรรมการบริโภค

3.1.1 รสชาตินำ (หวาน เปรี้ยว เค็ม เผ็ด จืด) ได้แก่ รส.....

3.1.2 น้ำมันพืช (ยี่ห้อ) ที่ใช้ในครัวเรือน คือ .....,……

3.1.3 แบบแผนการรับประทานอาหาร

( ) รับประทานอาหารน้อยกว่า 3 มื้อต่อวัน

( ) รับประทานอาหาร 3 มื้อต่อวัน

( ) รับประทานอาหารมากกว่า 3 มื้อต่อวัน

3.1.4 ความถี่ในการรับประทานอาหารขณะดูโทรทัศน์ .....ครั้ง/สัปดาห์

3.2 ความถี่ของการบริโภคอาหาร

ขอให้ท่านคิดถึงพฤติกรรมการบริโภคอาหารของท่านในช่วง 2-3 ปีที่ผ่านมา แล้วกรอกปริมาณความถี่ของการรับประทานอาหารดังนี้

ที่	ชนิดอาหาร	ความถี่ในการบริโภคอาหาร (ให้กรอกเป็นจำนวนครั้งต่อสัปดาห์)
	หมวดเนื้อสัตว์บก (เช่น เนื้อหมู เนื้อไก่ เนื้อวัว เป็นต้น)	
	เนื้อสัตว์ติดมันนำมาต้ม, ปิ้ง, ย่าง	
	เนื้อสัตว์ติดมันนำมาผัด	
	เนื้อสัตว์ติดมันนำมาทอด	
	เนื้อสัตว์ไม่ติดมันนำมาต้ม, ปิ้ง, ย่าง	
	เนื้อสัตว์ไม่ติดมันนำมาผัด	
	เนื้อสัตว์ไม่ติดมันนำมาทอด	
	หมวดเนื้อสัตว์ทะเล(เช่น ปลาหมึก, กุ้ง, หอย เป็นต้น)	
	เนื้อสัตว์ทะเลนำมาต้ม, ปิ้ง, ย่าง	
	เนื้อสัตว์ทะเลนำมาผัด	
	เนื้อสัตว์ทะเลนำมาทอด	

ที่	ชนิดอาหาร	ความถี่ในการบริโภคอาหาร (ให้กรอกเป็นจำนวนครั้งต่อสัปดาห์)
	หมวดเนื้อสัตว์น้ำ	
	ปลาน้ำจืด/น้ำเค็ม (เช่น ปลาดุก, ปลาช่อน, ปลาทู, ปลาอินทรีย์ เป็นต้น) นำมาทอด ต้ม ปิ้ง ย่าง ผัด	
	ผลิตภัณฑ์จากปลาที่ผ่านการถนอมอาหาร (เช่น ปลาเค็ม ปลาแดดเดียว ปลากระป๋อง เป็นต้น) นำมาทอด ต้ม ปิ้ง ย่าง ผัด	
	หมวดข้าว แป้ง	
	ข้าวขัดสีน้อย เช่น ข้าวกล้อง ข้าวซ้อมมือ	
	ข้าวขัดสี เช่น ข้าวขาว	
	ก๋วยเตี๋ยวเส้นเล็ก เส้นใหญ่ เส้นหมี่	
	ก๋วยเตี๋ยวเส้นบะหมี่	
	หมวดผัก/ผลไม้	
	ผักสดทุกชนิด	
	ผักต้มทุกชนิด	
	ผลไม้ให้รสหวาน เช่น กล้วย มะละกอ	
	ผลไม้รสหวานน้อย เช่น ส้ม แก้งมังกร ฝรั่ง	
	หมวดนมและผลิตภัณฑ์จากนม	
	นมสด (หนึ่งหน่วยเท่ากับ 200 cc.)	
	นมพร่องมันเนย (หนึ่งหน่วยเท่ากับ 200 cc.)	
	นมเปรี้ยว (หนึ่งหน่วยเท่ากับ 200 cc.)	
	นมข้นหวาน (หนึ่งหน่วยเท่ากับ 2 ช้อนชา)	
	หมวดขนมหวาน	
	ใส่กะทิ เช่น ลอดช่อง วุ้นกะทิ เต้าส่วน เป็นต้น	
	ไม่ใส่กะทิ เช่น เฉาก๊วย ถั่วเขียวต้มน้ำตาล เป็นต้น	

ที่	ชนิดอาหาร	ความถี่ในการบริโภคอาหาร (ให้กรอกเป็นจำนวนครั้งต่อสัปดาห์)
	หมวดเครื่องดื่ม	
	เครื่องดื่มน้ำตาลสูง เช่น ชานมเย็น น้ำอัดลม โอวัลติน ไมโล	
	เครื่องดื่มน้ำตาลน้อย/ไม่มีน้ำตาล เช่น ชาจีน	
	เครื่องดื่มมีคาเฟอีน เช่น กาแฟ เครื่องดื่มชูกำลัง	

ส่วนที่ 4 แบบวัดบุคลิกภาพ (Type A Behavior)

ให้ท่านลองพยายามนึกถึงลักษณะที่ใกล้ตัวท่านมากที่สุด ก่อนที่จะตอบว่า “ถูก” หรือ “ผิด”

ข้อคำถาม	ถูก	ผิด
1. เวลาท่านโกรธ ท่านมักจะไม่ให้คนอื่นรู้		
2. ท่านคิดว่าคนส่วนมากจะเห็นแก่ตัวและนึกถึงแต่ตัวเอง		
3. คนส่วนมากจะบอกว่าท่านเป็นคนใจเย็นและง่ายๆ		
4. ท่านจะรู้สึกทุกข์ใจหรือกลุ้มใจเมื่อท่านต้องอยู่ว่างๆ		
5. ท่านมักจะเชื่อใจคนที่ท่านทำงานด้วยเสมอ		
6. ท่านคิดว่าคนทั่วไปโดยปกติเป็นคนดีมาแต่เกิด		
7. ท่านจะหงุดหงิดรำคาญใจ เมื่อต้องรอคอยอะไร		
8. ท่านมักจะใจเย็นแม้ว่าอยู่ในสถานการณ์ฉุกเฉิน		
9. ท่านจะอดทนได้เสมอเมื่อต้องรอคอยคนอื่น		
10. ท่านจะคำนึงถึงความรู้สึกตนเองเสมอ		
11. ท่านมักจะชอบทำงานแข่งกับเวลา		
12. ท่านคิดว่าการมีวันหยุดพักผ่อนตามปกติเป็นเรื่องสำคัญ		
13. ท่านมักจะให้ความสนใจในการแก้ปัญหาทีละเรื่อง		
14. ท่านเป็นคนที่ไม่ค่อยไว้ใจคนอื่น		
15. มีคนอื่นที่พูดว่าท่านเป็นศัตรูและไม่น่าไว้ใจ		
16. ท่านจะรับฟังความคิดเห็นผู้อื่นเสมอ		
17. ท่านมักจะชอบทำงานแข่งกับเวลาแม้ว่าไม่จำเป็นต้องรีบทำ		
18. ท่านมักสงสัยหรือระแวงคนอื่นอยู่เสมอๆบ่อยๆ		
19. ท่านมักทำงานไม่ดีนักในระยะเวลาที่จำกัด		
20. ท่านมักจะพยายามใจเย็นเมื่อทุกอย่างดูเชื่องช้าลง		

ส่วนที่ 5 แบบวัดความเครียดสำหรับคนไทย (Suanprung Stress Test)

ให้ท่านอ่านหัวข้อข้างล่างนี้ แล้วสำรวจดูว่าในระยะ **6 เดือนที่ผ่านมา**มีเหตุการณ์ในข้อใดเกิด ขึ้นกับตัวท่านบ้าง ถ้าข้อไหนไม่ได้เกิดขึ้นให้ข้ามไปไม่ต้องตอบ แต่ถ้ามีเหตุการณ์ในข้อใดเกิดขึ้นกับตัวท่านให้ประเมินว่าท่านมีความรู้สึกอย่างไรต่อเหตุการณ์นั้น แล้วทำเครื่องหมายให้ตรงช่องตามที่ท่านประเมิน โดย

คะแนนความเครียด 1 คะแนน หมายถึง ไม่รู้สึกเครียด

คะแนนความเครียด 2 คะแนน หมายถึง รู้สึกเครียดเล็กน้อย

คะแนนความเครียด 3 คะแนน หมายถึง รู้สึกเครียดปานกลาง

คะแนนความเครียด 4 คะแนน หมายถึง รู้สึกเครียดมาก

คะแนนความเครียด 5 คะแนน หมายถึง รู้สึกเครียดมากที่สุด

ข้อ	ในระยะ 6 เดือนที่ผ่านมา	คะแนนความเครียด				
		1	2	3	4	5
1	กลัวทำงานผิดพลาด					
2	ไปไม่ถึงเป้าหมายที่วางไว้					
3	ครอบครัวมีความขัดแย้งกันในเรื่องเงินหรือเรื่องงานในบ้าน					
4	เป็นกังวลกับเรื่องสารพิษ หรือมลภาวะในอากาศ น้ำ เสียง และดิน					
5	รู้สึกว่าต้องแข่งขันหรือเปรียบเทียบ					
6	เงินไม่พอใช้จ่าย					
7	กล้ามเนื้อตึงหรือปวด					
8	ปวดหัวจากความตึงเครียด					
9	ปวดหลัง					
10	ความอยากอาหารเปลี่ยนแปลง					
11	ปวดศีรษะข้างเดียว					
12	รู้สึกวิตกกังวล					
13	รู้สึกคับข้องใจ					
14	รู้สึกโกรธ หรือหงุดหงิด					
15	รู้สึกเศร้า					
16	ความจำไม่ดี					

17	รู้สึกสับสน					
18	ตั้งสมาธิลำบาก					
19	รู้สึกเหนื่อยง่าย					
20	เป็นหวัดบ่อย ๆ					

ข้อมูลทางการแพทย์ (สำหรับผู้วิจัยเท่านั้น)

ส่วนที่ 6 ข้อมูลประวัติทางด้านการแพทย์(คัดลอก)

6.1. วัน เดือน ปี ที่ได้รับการตรวจคัดกรอง...../……/…….

6.2. วัน เดือน ปี ที่ได้รับการวินิจฉัยว่าป่วยด้วยโรคเบาหวาน

...../……/…….

6.3. วัน เดือน ปี ที่ได้รับการวินิจฉัยว่าป่วยด้วยโรคความดันโลหิตสูง

...../……/…….

6.4. วัน เดือน ปี ที่ได้รับการวินิจฉัยว่าป่วยด้วยโรคไขมันในเส้นเลือด

...../……/…….

6.5. ผลการคัดกรอง

6.5.1. น้ำหนัก .....กิโลกรัม

6.5.2. ส่วนสูง .....เซนติเมตร

6.5.3. รอบเอว ..... เซนติเมตร

6.5.4. ความดันโลหิต Systolic…….mmHg

Diastolic…….mmHg

6.5.5. ระดับน้ำตาลในเลือด (Fasting Plasma Glucose)

…….mg/dl

6.5.6. ระดับไขมันในเลือด (Total cholesterol) …….mg/dl

6.5.7. ระดับไตรกลีเซอไรด์ (Triglyceride) ……..mg/dl

6.5.8. ระดับ HDL ……..mg/dl

APPENDIX B

Table B-1 Demographic characteristics of study population by gender.

Characteristics	Male (n=147)		Female (n=261)	
	N	%	N	%
Age (Year)				
35-44	32	21.77	69	26.44
45-54	68	46.26	107	41.00
55-60	47	31.97	85	32.57
Mean	50.31		49.85	
SD	6.40		6.98	
Marital status				
Couple	134	91.16	188	72.03
Widowed	3	2.04	17	6.51
Divorce	1	0.68	7	2.68
Single	9	6.12	49	18.77
Religion				
Buddhist	147	100.00	260	99.62
Christian	0	0.00	1	0.38
Education level				
Illiterate	4	2.72	19	7.28
Primary	117	79.59	214	81.99
Pre-high school	15	10.20	12	4.60
High school and over	11	7.48	16	6.13
Occupation				
Agriculture	90	61.22	131	50.19
Servant	3	2.04	9	3.45
Employee(labor)	26	17.69	50	19.16
Housework	1	0.68	5	1.92
Office employee	6	4.08	4	1.53
Merchant	12	8.16	19	7.28
No work	7	4.76	35	13.41
Others	2	1.36	8	3.07

Table B-1 Demographic characteristics of study population by gender (continued).

Characteristics	Male (n=147)		Female (n=261)	
	N	%	N	%
Personal Income per month (Baht)				
No income	20	13.61	63	24.14
≤ 5,000	53	36.05	101	38.70
5,001 – 10,000	59	40.14	88	33.72
≥10,001	15	10.20	9	3.45
Mean	8,880.96		6,860.26	
S.D.	8,665.32		6,190.06	
Family history of hypertension				
Yes	50	34.01	80	30.77
No	97	65.99	180	69.23
Family history of DM				
Yes	40	27.21	53	20.31
No	107	72.79	207	79.62
Family history of hypercholesterolemia				
Yes	28	19.05	41	15.71
No	119	80.95	220	84.29
Note: 1 data missing				
Menopause Status (only in female)				
Yes			91	35.00
No			169	65.00

Table B-2 Prevalence (%) of each components of metabolic syndrome among cases and controls.

components	Case		Control	
	N	%	N	%
Gender: Male	73	37.24	74	34.91
Female	123	62.76	138	65.09
WC ≥ normal*	159	81.12	3	1.42
TG ≥ 150 mg/dl	163	83.16	2	0.94
HDL ≤ normal**	117	59.69	0	0.00
BP > 130/85 mmHg	158	80.61	28	13.21
FPG ≥ 100 mg/dL	112	57.14	0	0.00

***WC** (waist circumference) : ≥ 90 cm in men or ≥ 80 cm in women by Asian definition

****HDL** (serum HDL) : concentration < 40 mg/dl in men and <50 mg/dl in women (or on drug treatment for reduced HDL-C)

Table B-3 Prevalence of smoking among total study population in male and female.

Characteristics	**Male (n=147)**		**Female (n=261)**	
	N	**%**	**N**	**%**
Smoking				
Current smoker	39	26.53	6	2.30
Ex-smoker	34	23.13	3	1.15
Non-smoker	74	50.34	252	96.55

Table B-4 Prevalence of smoking quantity among total study population in male and female.

Characteristics	**Male (n=123)**		**Female (n=258)**	
	N	**%**	**N**	**%**
Smoking quantity				
Non-smoker	75	60.98	252	97.67
1-10 cigarettes a day	34	27.64	6	2.33
11s and more a day	14	11.38	0	0.00

Table B-5 Prevalence of alcohol drinking among the total study populations in male and female.

Characteristics	Male (n=144)		Female (n=260)	
	N	%	N	%
Alcohol				
Current drinker	56	38.89	19	7.31
Ex-drinker	20	13.89	2	0.77
Non-drinker	68	47.22	239	91.92

Table B-6 Prevalence of physical activity at daily traveling to work among the total study populations in males and females.

Characteristics	Male (n=147)		Female (n=261)	
	N	%	N	%
Physical activity at daily traveling to work				
Light	62	42.18	62	23.75
Heavy	70	47.62	144	55.17
Others	15	10.20	55	21.07

Table B-7 Prevalence of physical activity by type of occupations among the total study populations in males and females.

Characteristics	Male (n=147)		Female (n=257)	
	N	%	N	%
Type of occupation				
Light and moderate work	42	28.57	100	38.91
Heavy work	105	71.43	157	61.09

Table B-8 Prevalence of exercise habits among the total study populations in males and females.

Characteristics	Male (n=147)		Female (n=261)	
	N	%	N	%
Leisure time activity				
Regular exercise	10	6.80	40	15.33
Irregular exercise	9	6.12	9	3.45
No exercise	128	87.07	212	81.23

Table B-9 Prevalence of sleeping after right main meals among total study populations in males and females.

Characteristics	Male (n=136)		Female (n=238)	
	N	%	N	%
Sleep after right main meals				
Usually	10	7.35	11	4.62
Mostly	17	12.50	23	9.66
Sometimes	90	66.18	154	64.71
Never	19	13.97	50	21.01

Table B-10 Prevalence of preferrent main taste among total study population in males and females.

Characteristics	Male (n=145)		Female (n=259)	
	N	%	N	%
Main taste				
Normal	20	13.79	45	17.38
Sweety	30	20.69	64	24.71
Sour	21	14.49	64	24.71
Salty	45	31.03	64	24.71
Spicy	29	20.00	22	8.49

Table B-11 Prevalence of having main meals among total study population in males and females.

Characteristics	Male (n=141)		Female (n=259)	
	N	%	N	%
Frequency of eating pattern per day				
Less than 3 times	19	13.48	38	14.67
3 times a day	111	78.72	215	83.01
More than 3 times	11	7.80	6	2.32

Table B-12 Prevalence of eating while watching T.V. among the total study populations in males and females.

Characteristics	Male (n=101)		Female (n=168)	
	N	%	N	%
Eating while watching per week				
0-7 times**	96	95.05	160	95.24
More than 7 times	5	4.95	8	4.76

Table B-13 Frequency of eating by types of food among the total study populations in males and females.

Characteristics	Male (n=147)		Female (n=261)	
	N	%	N	%
All meat				
Low intake	115	78.23	192	74.13
High intake	32	21.77	67	25.87
Seafood				
Low intake	127	86.39	229	87.74
High intake	20	13.61	32	12.26
Fish				
Low intake	78	53.06	114	43.68
High intake	69	46.94	147	56.32
Brown Rice				
Low intake	124	84.35	238	91.19
High intake	23	15.65	23	8.81
Fruit and vegetable				
Low intake	71	48.30	101	38.70
High intake	76	51.70	160	61.30

Table B-14 Prevalence of behaviors among the total study populations in males and females.

Characteristics	Male (n=147)		Female (n=259)	
	N	%	N	%
Definite type B	6	4.08	5	1.93
Type B	53	36.05	72	27.80
Both A and B trait	83	56.46	164	63.32
Type A**	5	3.40	18	6.95

Table B-15 Prevalence of stress among total study population in males and females.

Characteristics	Male (n=147)		Female (n=261)	
	N	%	N	%
Stress score				
Mild Stress	9	6.12	16	6.13
Moderate stress	72	48.98	131	50.19
high stress	51	34.69	90	34.48
severe stress	15	10.20	24	9.20

BIOGRAPHY

NAME	Mr.Nitikorn Phoosuwan
DATE OF BIRTH	29 June 1982
PLACE OF BIRTH	Angthong, Thailand
INSTITUTIONS ATTEND	Sirindhorn College of Public Health, 2005 Bachelor of Public Health Mahidol University, 2011 Master of Science (Public Health)
OFFICE POSITION	Health Promotion and Prevention Department, Banphaeo Hospital (Public Organization), Samutsakorn Position : Public Health Technical Officer Tel. 081-7918914
HOME ADDRESS	196/252 Moo 1, Banphaeo, Banphaeo, Samutsakorn 74120