THE OPTIMAL CUT-OFF POINTS OF BODY MASS INDEX WHICH REFLECT THE RISK FACTORS OF CARDIOVASCULAR DISEASE IN URBAN THAI MALE POPULATION

PRAPAPHORN KAEWBOONRUANG

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ABSTRACT

The objective of this cross sectional research was to examine the optimal cut-off points of body mass index which reflect the risk factors of cardiovascular disease in the urban Thai male population. The cardiovascular risk factors were hypertension and dyslipidemia. Thai males aged 35-50 years who worked and lived in Bangkok were surveyed. These respondents were 413 government officers who answered questionnaires regarding demographic and health data. Their weight and height were measured for calculating body mass index (BMI). Measurement of blood pressure and collection of blood samples for blood chemical analysis were performed. SPSS and epidemiological methods (sensitivity, specificity and ROC curve analysis) were used for data analysis.

Results show that more than 50 percent of respondents were overweight when using the Asia criteria of BMI ≥23 kg/m^2. While, the prevalence of overweight respondents using the WHO criteria of BMI ≥25 kg/m^2 was 32.7 percent. The mean BMI level of the respondents increased proportionately with blood pressure (SPB and DBP), TC and LDL-C, except, TG. Increasing of HDL-C levels reversed proportionately with BMI level. The optimal cut-off point of BMI which reflected hypertension was 23.5 kg/m^2. The optimal cut-off points of BMI which reflected high risk in TC, TG, LDL-C, and HDL-C were 22.0, 23.5, 23.0, and 24.0 kg/m^2, respectively.

The findings indicate that the cut-off points of BMI that reflect the risk factors of cardiovascular disease in this study were lower than WHO criteria but was almost matching the Asia criteria. Therefore, the cut-off points of BMI according to WHO criteria may not be appropriate for the Thai male population. These results demonstrate that if the physicians approach to specifying overweight as a BMI of ≥25 kg/m^2 is taken as an indication for therapeutic intervention, then the Thai male population will be ignored despite the profound relationship of weight gain to cardiovascular risk factors and incidence of disease such as hypertension and dyslipidemia.

KEY WORDS: BODY MASS INDEX/ CUT-OFF POINT FOR OBESITY/ CARDIOVASCULAR DISEASE/ HYPERTENSION/ DYSLIPIDEMIA

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จุดตัดของดัชนีมวลกายที่สะท้อนปัจจัยเสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือดในกลุ่มประชากรชายไทยที่อาศัยอยู่ในเขตเมือง (THE OPTIMAL CUT-OFF POINTS OF BODY MASS INDEX WHICH REFLECT THE RISK FACTORS OF CARDIOVASCULAR DISEASE IN URBAN THAI MALE POPULATION)

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บทคัดย่อ

การศึกษานี้เป็นการวิจัยแบบภาคตัดขวาง มีวัตถุประสงค์เพื่อศึกษาหาจุดตัดของดัชนีมวลกายที่สามารถสะท้อนปัจจัยเสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือดในกลุ่มประชากรชายไทยที่อาศัยอยู่ในเขตเมือง ปัจจัยเสี่ยงที่สำคัญต่อการเกิดโรคหัวใจและหลอดเลือด คือ ภาวะความดันโลหิตสูง และความผิดปกติของระดับไขมันในเลือด กลุ่มตัวอย่างเป็นประชากรชายไทยอายุ 35-50 ปี ที่ทำงานและอาศัยอยู่ในเขตกรุงเทพมหานคร โดยเป็นผู้ที่ทำงานในหน่วยงานราชการ จำนวน 413 คน เก็บรวบรวมข้อมูลโดยให้กลุ่มตัวอย่างเป็นผู้ตอบแบบสอบถามข้อมูลส่วนบุคคลและข้อมูลสุขภาพ ชั่งน้ำหนักและวัดส่วนสูง คำนวณค่าดัชนีมวลกาย วัดความดันโลหิต และเก็บตัวอย่างเลือดเพื่อวิเคราะห์ดัชนีบ่งชี้ทางชีวเคมี การวิเคราะห์ข้อมูลใช้โปรแกรม SPSS และวิธีทางระบาดวิทยา (ความไว, ความจำเพาะและ ROC curve)

ผลการศึกษาพบว่า เมื่อใช้จุดตัดของดัชนีมวลกายที่ ≥23 กก/ม^2 ตามข้อกำหนดของการศึกษาในกลุ่มประเทศเอเชียเพื่อบ่งบอกถึงภาวะน้ำหนักตัวเกิน พบว่า กลุ่มตัวอย่างมากกว่าร้อยละ 50 มีภาวะน้ำหนักตัวเกิน แต่ในเมื่อใช้จุดตัดของดัชนีมวลกายที่ ≥25 กก/ม^2 ตามข้อกำหนดขององค์การอนามัยโลก พบกลุ่มตัวอย่างมีภาวะน้ำหนักตัวเกินร้อยละ 32.7 นอกจากนี้ยังพบว่า ค่าเฉลี่ยของดัชนีมวลกายเพิ่มขึ้นเป็นสัดส่วนโดยตรงกับความดันโลหิตซีสโตลิค, ความดันโลหิตไดแอสโตลิค, โคเลสเตอรอลรวมและไลโปโปรตีนชนิดความหนาแน่นต่ำที่สูงขึ้น ยกเว้นไตรกลีเซอไรด์ และค่าเฉลี่ยของดัชนีมวลกายเพิ่มขึ้นเป็นสัดส่วนผกผันกับไลโปโปรตีนชนิดความหนาแน่นสูงที่ต่ำลง จุดตัดของดัชนีมวลกายที่สามารถสะท้อนถึงภาวะความดันโลหิตสูงในการศึกษานี้คือ จุดตัดของดัชนีมวลกายที่ 23.5 กก/ม^2 และจุดตัดของดัชนีมวลกายที่สามารถสะท้อนถึงภาวะโคเลสเตอรอลรวมในเลือดสูง, ไตรกลีเซอไรด์ในเลือดสูง, ไลโปโปรตีนชนิดความหนาแน่นต่ำในเลือดสูงและไลโปโปรตีนชนิดความหนาแน่นสูงในเลือดต่ำ ในการศึกษานี้คือ จุดตัดของดัชนีมวลกายที่ 22.0, 23.5, 23.0 และ 24.0 กก/ม^2 ตามลำดับ

ผลการศึกษาชี้ว่าจุดตัดของดัชนีมวลกายที่สะท้อนถึงปัจจัยเสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือดในการศึกษานี้มีค่าต่ำกว่าค่าที่องค์การอนามัยโลกได้กำหนดไว้ แต่มีค่าใกล้เคียงกับค่าที่กำหนดไว้ในการศึกษาของกลุ่มประเทศเอเชีย ดังนั้นจุดตัดของดัชนีมวลกายเพื่อบ่งบอกถึงภาวะน้ำหนักตัวเกินที่ได้กำหนดโดยองค์การอนามัยโลกอาจไม่เหมาะสมกับกลุ่มประชากรชายไทย หากบุคลากรทางการแพทย์ใช้จุดตัดของดัชนีมวลกายที่ ≥25 กก/ม^2 เป็นข้อบ่งชี้ถึงภาวะน้ำหนักตัวเกิน อาจจะทำให้กลุ่มประชากรชายไทยมีภาวะเสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือดมากขึ้น คือ มีภาวะความดันโลหิตสูง และมีความผิดปกติของระดับไขมันในเลือดเพิ่มขึ้น

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ABBREVIATIONS

CVD	Cardiovascular disease
CHD	Coronary heart disease
CAD	Coronary artery disease
BMI	Body mass index
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
TC	Total cholesterol
TG	Triglyceride
LDL-C	Low density lipoprotein cholesterol
HDL-C	High density lipoprotein cholesterol
PVD	Peripheral vascular disease
MI	Myocardial infarction
Lp(a)	Lipoprotein a
WHR	Waist to hip ratio
WC	Waist circumference
WHO	World Health Organization
ISH	International Society of Hypertension
NCEP	National Cholesterol Education Program
NIDDM	Non-insulin dependent diabetes mellitus
ROC	Receiver operating characteristic curve

CHAPTER I
INTRODUCTION

Rationale

In the past, weight gain and fat storage have been viewed as signs of health and prosperity. Nowadays, it threatens physical condition. Obesity is now well recognized as a disease in its own right. It is a major risk factor of many non-communicable diseases such as cardiovascular disease, hypertension and diabetes mellitus. Cardiovascular disease (CVD) in particular is significant as it is one of the five leading causes of death worldwide (1).

The prevalence of overweight and obesity has been increasing in most parts of the world. According to the World Health Organization Consultation on Obesity, the incidence of obesity has been increasing rapidly since 1990. The prevalence of obesity in adult population is 10-25% in most countries of Western Europe and 20-25% in some countries in the Americas (2). In Thailand, the Fourth Nations Food and Nutrition Survey (1995) by the Department of Health, Ministry of Public Health used the BMI of 25 kg/m^2 or more to classify of obesity and found that the prevalence rate of obesity was increasing in all adult age groups. In particular, the age groups 30-39, 40-49 and 50-59 years these rates were 20.8%, 40.2% and 35.0% respectively (3). Longitudinal study of Thai bank employees revealed that the majority of participants both male and female experienced weight gain. The prevalence of overweight and obesity increased in both sexes (4). This has resulted in an epidemic that has occurred concurrently with the changes in lifestyle and diet, along with an increase in life expectancy and urbanization.

In Thailand, mortality related to CVD has been increasing progressively each year since 1975 and has become one of the first three leading causes of death among the Thai population for more than 10 years. Health Information Division, Bureau of Health Policy and Strategy reported the mortality rate of heart disease were 56.0, 58.5,

62.5, 69.2, 77.4, and 71.2 per 100,000 population in 1992, 1993, 1994, 1995, 1996 and 1997 respectively (5).

A number of cardiovascular risk factors are influenced by overweight including hypertension, impaired glycaemic control, dislipidemia and haemostatic and rheological factors. Until recently it was thought that only severe degrees of excess weight increased the risk of coronary heart disease (CHD), but recent evidence shows a clear association with modest weight gain. In the nurses' study, Willett et al. (6) controlled for age, smoking, menopausal status, post menopausal hormone use and family history. They found that the risk of CHD increased 2-fold in women of body mass index (BMI) 25-28.9 kg/m^2 and by 3.6 for those with a BMI of 29 kg/m^2 or more. Weight gain from age 18 years increased the risk 1.6-fold for an 8-10.9 kg weight gain and 1.9-fold for an 11-19 kg gain. In males, a 10% increase in weight will increase the risk of coronary heart disease by 38% whereas a 20% weight rise corresponds with an 86% increase in risk (7). Blood pressure increased by 6 mmHg systolic and 4 mmHg diastolic for a 10% gain in body fat and those who are genetically more susceptible show the greater effect (7). Reisen et al. (8) have demonstrated that a weight loss of 11 kg produced a 20% decrease in both systolic and diastolic pressure in hypertensive patients even when the sodium intake was kept constant. It would appear that, as a general rule, blood pressure is reduced by 1 mmHg systolic and 2 mmHg diastolic for each 1% reduction in body weight (7). The most characteristic lipid disorders in obesity are elevated total cholesterol (TC) and triglycerides (TG), high low density lipoprotein cholesterol (LDL-C) and low high density lipoprotein cholesterol (HDL-C). A meta-analysis by Datillo and Kris-Etherton of some 70 published studies and other work reviewed, has indicated that for every 1 kg of weight lost, there is a corresponding reduction by about 1% in TC and LDL-C and a rise by 1% in HDL-C (9).

Early obesity detection, prevention and management offer the best of solution. There are many methods to assess fat distribution. Direct methods include computed tomography, magnetic resonance, ultrasound, and dual energy x-ray. While those methods are accurate, they are also complicated, expensive and cannot work in the field. Conversely, anthropometry are a range of indirect methods such as body mass index (BMI), waist to hip ratio (WHR), waist circumference (WC) and skinfold

thickness and these demonstrative ways have been successfully used for work in the field because they are simple and expedient (10-13). The World Health Organization (WHO) suggests using cut-off points for BMI and WC concurrently but declares that the use of WC to assess health risk would need to population specific and depends on the presence or absence of other risk factors (e.g. overweight, CVD, and type 2 diabetes) (1). Some studies pointed out the possible advantages of using anthropometric ratios. Suggested ratios were the WHR, the waist-to-thigh ratio, the waist-to-height ratio and the sagittal abdominal diameter-thigh ratio. The rationale for using these ratios is that the numerator reflects a combination of total and abdominal fat mass and the denominator reflects overall body size or a body tissue mass (e.g. muscle) that must be accounted for. However, since measurement errors may be compounded in a ratio, and because the interpretation of these ratios in pathophysiologic terms is difficult, the public health application of these ratios might be limited (11-13).

A BMI is an indicator of fatness and is used to clinically assess obesity. Its wide acceptance has been due to its simplicity and high specificity in screening for total body fatness (% Body Fat: %BF) (14). However, a number of studies have indicated limitation for its use. Length of legs or trunk in relation to overall stature influences BMI. Also BMI cannot differentiate the components of body composition, such as fat mass (FM) and fat-free mass (FFM), of subjects (15). Consequently BMI can be large for those with short legs in proportion to their height and for those with high muscularity. Consideration of these limitations is important when comparing BMI values obtained across a range of different ethnic groups, because their body proportions and body build are often different from each other (16).

Previous cross-ethnic studies that examined the BMI-%BF relationship in respect to age, gender and ethnicity have reported that Asians tend to possess greater %BF than Caucasians at the same BMI values (17-20). There are also reports that Asians have higher health morbidities at lower BMI values than Caucasians. In Asians, the cut-off classifications for overweight (≥23.0 kg/m^2) and obesity (≥25.0 kg/m^2) are lower than the WHO criteria (overweight ≥25.0 kg/m^2 and obesity ≥30.0 kg/m^2) (21-25). These findings suggest that the universal BMI classification proposed by the WHO may not be applicable to all populations. There is a need for studies to

determine if population-specific BMI classifications should be developed for different ethnic groups in order to allow BMI to be used as a better indicator of fatness (26).

In Thailand, Stevens NHC (27) studied 46 Thai women aged between 30-39 years old to test if body composition prediction equations using BMI, developed for Caucasian females can be used for Thai females. The results found that Thai female subjects have higher body fat than Caucasian females in the same level of BMI. Consequently, the universal BMI classification by WHO might not be a suitable prediction of body composition for Thai females. Chunchusak (28) studied 120 working men from factories in Bangpakong industrial estate. Their ages ranged between 35-60 years old and the results found that more than 70 percent of the subjects have normal BMI (20-24.9 kg/m^2) but their have dyslipidemia levels were rate as high normal levels of TC and TG were 60 percent and 23.58 percent, respectively. Therefore, the BMI classification proposed by WHO might not be applicable to the Thai male population. For this reason, the aim of this study was to examine the optimal cut-off points of BMI which be reflect the risk factors of CVD such as hypertension and dyslipidemia in the urban Thai male population.

Research Question

What are the optimal cut-off points of BMI in the selected urban Thai population which detect: high in blood pressure (BP), total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C) and low in high-density lipoprotein cholesterol (HDL-C)?

Research Objective

General Objective

To examine the optimal cut-off points of BMI which reflect the risk factors of CVD in the urban Thai male population.

Specific Objectives

1. To identify the optimal cut-off points of BMI which associate with the risk factors of CVD such as high in blood pressure, total cholesterol, triglyceride, low-density lipoprotein cholesterol and low in high-density lipoprotein cholesterol.

2. To compare the sensitivity of cut-off points of BMI which reflect the risk factors of CVD between Asia criteria (as 23 kg/m^2 or more) and WHO criteria (as 25 kg/m^2 or more).

Research Hypothesis

1. The cut-off points of BMI which detect the risk factors of CVD in this study is lower than WHO criteria (BMI $\geq$25.0 kg/m^2).

2. The sensitivity of cut-off points of BMI in Asia criteria is better than WHO criteria for detection the risk factors of CVD.

Scope of this Study

The subjects of this study are derived from Thai male population aged 35-50 years old and who live in Bangkok. Their employment duties are those of official works are official work, which have low levels of physical activity.

Limitations of this Study

1. This research aims at studying the selected Thai male population who live in Bangkok.

2. This research does not deal with eating habits that may affect the levels of BMI, blood pressure and plasma lipids.

3. This research dose not deal with family history of disease such as hyperlipidemia and hypertension because these diseases are the chronic disease which may not present signs and symptoms in the present time and so most of samples do not know their family history of disease.

Definition of Terms

1. Body mass index (BMI) is a simple index of weight-for-height that is commonly used to classify overweight and obesity in adults. It is calculated as the weight in kilograms divided by the square of the height in meters (kg/m^2).

$$\text{Body mass index} = \frac{\text{Body weight (kg)}}{\text{Body height (m)}^2}$$

2. Cardiovascular disease (CVD) risk factors are the factors associated with coronary heart disease (CHD) and cerebrovascular disease (stroke). These include hypertension and dyslipidemia.

2.1 Hypertension

Blood pressure levels are classified according to WHO-ISH Guideline for the Management of Hypertension (29).

- Optimal blood pressure means SBP <120 mmHg and DBP <80 mmHg.
- Normal blood pressure means SBP <130 mmHg and DBP <85 mmHg.
- High–normal blood pressure SBP 130-139 mmHg and DBP 85-89 mmHg.
- Hypertension is a SBP of 140 mmHg or greater and/or a DBP of 90mmHg.

2.2 Dyslipidaemia

Plasma lipids mean TC, TG, LDL-C and HDL-C that were classified according to the National Cholesterol education program (NCEP) (33).

2.2.1 Total cholesterol (TC)

- Acceptable levels means TC <200 mg/dl.
- Borderline levels means TC 200-239 mg/dl.
- High risk levels means TC ≥240 mg/dl.

2.2.2 Triglyceride (TG)

- Acceptable levels means TG <200 mg/dl.
- Borderline levels means TG 200-400 mg/dl.
- High risk levels means TG >400 mg/dl.

2.2.3 Low density lipoprotein cholesterol (LDL-C)

- Acceptable levels means LDL-C <130 mg/dl.
- Borderline levels means LDL-C 130-159 mg/dl.
- High risk levels means LDL-C ≥160 mg/dl.

2.2.4 High density lipoprotein cholesterol (HDL-C)

- Acceptable levels means HDL-C >35 mg/dl.
- High risk levels means HDL-C <35 mg/dl.

CHAPTER II
LITERATURES REVIEW

This study aims to examine the BMI cut-off points level which are associated with the risk factors of CVD in the Thai male population who live in Bangkok.
The critical review of the literatures includes:

1. Cardiovascular disease
   1.1 Scope of problem
   1.2 Pathophysiology
   1.3 Risk factors of CVD
2. Obesity
   2.1 Scope of problem
   2.2 Etiology of obesity
   2.3 Risk factors associated with obesity
   2.4 Assessing of obesity
   2.5 Health problems related to obesity
3. Obesity and cardiovascular risk factors
   3.1 Hypertension
   3.2 Dyslipidaemia

1. Cardiovascular disease

1.1 Scope of problem

The American Heart Association classify nine types of cardiovascular disease (CVDs): CHD (sometimes called coronary artery disease; CAD or ischemic heart disease; IHD) (31), stroke, high blood pressure, arrhythmias, disease of the arteries, congestive heart failure, valvular heart disease, rheumatic fever/rheumatic heart disease and congenital heart disease (32).

CVD is an important cause of global morbidity and in five of six WHO regions it is the leading cause of morbidity (Table 1) (33). As health surveillance systems improve in non-industrialized countries, there is an emerging awareness of the magnitude of CVD as a cause of premature mortality. This is also true for industrialized societies, and in several regions there has been a rapid increase in CVD rate over the last two decades. In many countries in Asia and Africa limited data have been available on detailed CVD mortality patterns. Nevertheless, it is now widely recognized that stroke is a major cause of death among the elderly and CHD is an important public-health problem in urbanized populations in these regions (34).

In Thailand, the mortality rate of heart disease gradually increased for more than 10 years until 1996,then has been slightly decreased for both genders in both the absolute number of deaths and the mortality rate (Table 2).

The death rate from diseases of the circulatory system in 2002 was 52.6 per 100,000 population. Among these, cerebrovascular diseases were the leading cause of death with a rate of 21.5 and ischemic heart diseases were the secondary cause of death with a rate of 14.4 per 100,000 population. Moreover, males had higher death rate than females (5).

1.2 Pathophysiology

Disease of the coronary artery is almost without exception related to atherosclerosis in the heart's arteries. Atherosclerosis is not a single clinical disease entity because atherosclerotic lesions can occur at different anatomic sites and their clinical sequelae depend on additional factors, such as age and physiologic status. Atherosclerotic lesions most often occur within the inner most layer of the artery wall,

the intima, but secondary changers can also be found in the media of the artery underlying the lesion (35).

Table 1. CVD-related death in 2001 per 100,000 population, by WHO region

CVD	African Region	American Region	European Region	South-East Asia Region	Western Pacific Region	Eastern Mediterranean Region	World
CHD	333	967	2,423	1,972	963	523	7,181
Stroke	307	454	1,480	1,070	1,926	218	5,455
Hypertensive heart disease	54	131	175	138	285	91	874
All CVDs	985	1,979	5,042	3,797	3,745	1,037	16,585

Source: WHO. The World Health report 2002 'Reducing risk and promoting health life'. Geneva, WHO. 2002 (33).

Table 2. Death of heart disease by gender and rate per 100,000 population, 1991-1997

Year	Male		Female		Total	
	No. of deaths	Rate	No. of deaths	Rate	No. of deaths	Rate
1991	8,760	66.2	12,243	43.2	1,003	54.7
1992	19,746	68.7	12,385	43.2	32,131	56.0
1993	21,219	72.9	12,770	44.1	33,989	58.5
1994	22,729	77.4	13,951	47.6	36,680	62.5
1995	25,531	86.2	15,522	52.3	41,053	69.2
1996	28,630	96.0	17,656	58.9	46,286	77.4
1997	26,482	87.9	16,486	54.4	42,968	71.1

Source: Health Information Division, Bureau of Health Policy and Plan, Ministry of Public Health Statistic. 1997 (5).

1.2.1 The atherosclerotic lesion

1) The fatty streak

The early stages of fatty steak formation are characterized by the appearance of large subendothelial lumps covered by an intact layer of endothelial cells. Early fatty streaks consist mainly of macrophages and T-lymphocytes. Initially, monocytes and T-lymphocytes adhere to an intact endothelial surface at sites of predilection for lesion development. Endothelial cells in the affected areas express adhesion molecules. After migration through the endothelium the monocytes differentiate into macrophages. With expression of the lesion, smooth muscle cells also migrate into the intima. Macrophages and smooth muscle cells typically are lipid-laden, and at this stage the cholesterol-rich lipid droplets are intracellular. In a later stage during the development of the fatty streak, defects are found in the arterial endothelium through which macrophages can protrude, most likely to remove excess lipids from the arterial intima. The fatty streak is seen in many different populations, not only in those with a high increase of atherosclerosis; therefore, a lipid deposition as the fatty streak does not inevitably lead to an advanced atherosclerotic lesion. Rather, a number of additional factors are necessary if the fatty streak is to progress to the next, more complex stage of an atherosclerotic lesion, the fibrous plaque (35).

2) The fibrous plaque

The fibrous plaque occurs almost only in populations with a high incidence of atherosclerosis. Furthermore, it protrudes into the lumen of vessel. The fibrous plaque is made up of macrophages, T-lymphocytes**,** accumulating connective tissue matrix, cell debris, and intracellular lipid and extracellular lipid – all covered by proliferating smooth muscle cells, not only in the lumen but also at the bottom of the lesion. The presence of T-lymphocytes indicates that an immune response is an important factor in the development of the lesion and that this process may actually be driven by an autoimmune response (35).

3) The advanced or complex lesion

As it ages the fibrous plaque is vascularized from the lumen and from the media of the artery. The core of the lesion becomes increasingly necrotic and eventually is calcified. The surface of the intima may develop fissures and ulcerate. The developing complex lesion protrudes into the arterial lumen, which, when a

thrombus is formed, can become completely occluded. Alternatively, the media underling the lesion may atrophy, leading to the formation of an aneurysm. Plaque instability appears to be caused by the abundance of lipid-laden macrophages and extracellular lipids and by the death of smooth muscle cells and extracellular matrix proteins thought to stabilize the plaque (35).

1.2.2 Pathogenetic mechanisms of plaque formation

1) The response-to-injury hypothesis

The hypothesis is based on the thought that factors such as altered blood flow, chronic elevation of serum cholesterol, toxins and other injurious agents can lead to disintegration of the surface of the intima. The injury might lead to a chronic inflammatory response involving macrophages, T-lymphocytes, platelets, and smooth muscle cells and also such mediator substances as platelet-derived growth factor (PDGE). The endothelial cell monolayer acts as a permeability barrier that regulates the passage of substances from the plasma to the underlying arterial wall. An intact endothelial cell layer is thromboresistant, because the surface of the endothelial cells contains prostacyclin and endothelial-derived growth factor (EDGF) as well as glycoproteins and proteoglycans with antithrombolic properties (35).

2) The monoclonal hypothesis

The monoclonal hypothesis suggests that each lesion of atherosclerosis is a being neoplasm derived from a single transformed smooth muscle cell; the mutation of the cell may be induced by certain chemical components or other risk factors. This hypothesis is not uncontested: each lesion could arise from a population of cells that expresses the same isoenzyme, and therefore the lesion could be hyperplastic rather than distribution of the isoenzymes in cells of the artery wall (35).

3) The lipid hypothesis

This hypothesis proposes that remnants of triglyceride-rich lipoproteins or modified LDL of hyperlipidemic subjects are taken up by macrophages to form the early atherosclerotic lesion, and that chronic exposure of endothelium to these lipoproteins leads to cell injury. Cell necrosis in turn results in a deposition of lipid in the extracellular space. Injury to the endothelium and progression of the atherosclerotic lesion by exposure to chronically elevated levels of remnants and/or

modified LDL could be part of the sequence leading to the formation of occlusion plaques and to their clinical sequelae (35).

1.3 Risk factors of CVD

Over the last 2 decades a downward trend of incidence of CHD was observed in some countries, whereas in others CHD remained on the rise. It remains the major cause of death in the world. Therefore, the literature review on risk factors will be related to CHD (34). The term "risk factors" first appeared in the Framingham Heart study reported in mid-1960s (36). The Framingham Heart Study has clearly established that high cholesterol, hypertension and cigarette smoking are the CHD risk factors attributable to the majority of people. In addition to these factors, gender, family history, and age have been identified as non-modifiable risk factors (36). Categories of risk factors are important both for risk assessment and as a target for intervention where commonly, it is identified in relation to the strategy of intervention into two groups (37):

- Non-modifiable risk factors are age, gender, race/ethnic, family history and type A personality.
- Modifiable risk factors are hyperlipidemia, cigarette smoking, hypertension, diabetes mellitus, obesity, physical inactivity and others e.g. contraceptive used, alcoholic intake, and stress. Most of the modifiable risk factors can be influenced by diet.

The risk factors for CVD can also be categorized into three groups (38):

- Non-modifiable risk factors are age, male gender, black, and family history of CVD.
- Physiological risk factors that are modifiable include elevated LDL-C, hypertension, diabetes mellitus and obesity.
- Modifiable behavioral risk factors such as smoking, consumption of dietary fat, cholesterol, salt, alcohol and a sedentary lifestyle.

Grundy et al. (39) proposed a classification of factors for CHD into four categories according to emerging mechanisms as follows.

1) Causal risk factors

The major causal risk factors are cigarette smoking, high blood pressure, elevated serum cholesterol (or LDL-C), low HDL-C, and high plasma glucose. Although the precise mechanisms whereby these five risk factors promote atherosclerosis and predispose to CHD are not yet fully understood, abundant evidence supports a directly causal role. Moreover, they act independently of one another.

2) Conditional risk factors

Conditional risk factors consist of factors that are associated with an increased risk for CHD but whose causal link to CHD remains to be documented with certainly. Because of uncertainty about their role in atherogenesis, the conditional risk factors are not universally accepted as being major causal risk factors. The conditional risk factors include elevated concentrations of serum triglycerides, Lp(a), small LDL particles, homocysteine, and coagulation factors.

3) Predisposing risk factors

Predisposing risk factors consist of obesity, physical inactivity, and family history of premature CHD, male gender, and possibly behavioral, socioeconomic, and ethnic factors. Another predisposing risk factor is insulin resistance, a condition in which cellular action is impaired by metabolic aberration. The major predisposing risk factors, obesity and physical inactivity worsen insulin resistance, and their impact on causal and conditional risk factors may be mediated largely via this mechanism.

4) Plaque burden as a risk factor

The usual way for estimating plaque burden in the clinical setting is to use age as surrogate marker. The severity of coronary atherosclerosis rises with age. Hence older persons on average have a greater plaque burden than younger persons. This factors accounts for the well known claim that age is a risk factors for CHD. Use of age as an indicator of plaque burden has been generally accepted for primary prevention.

1.3.1 Major non-modifiable risk factors

1) Age and sex

Age is an independent risk factor for CVD. At any given LDL-C concentration, risk for a clinical CVD event is higher in older than in younger people (40). Eighty-five percent of Americans who die of CVD are age 65 or older. Given

that age is a surrogate for coronary atherosclerotic plaque burden, the true risk factor, in risk prediction algorithms, it has been suggested that risk algorithms would be improved by the replacement of the age factor by accurate measurement of plaque burden by noninvasive methods (41). At any given age, men are at a greater risk than women for a CVD event. The reason for the difference in risk by sex is not entirely understood. Both biologic and environment (including behavioral and psychosocial) factors have been suggested. The differences in CVD mortality rates between countries are greater than the differences between men and women, suggesting that biologic factors are not solely responsible (40).

2) History of atherosclerotic disease

Established CVD or clinical atherosclerotic disease of the aorta, carotid arteries, or arteries to the limbs confers very high risk for myocardial infarction (MI) or CHD death. Risk is increased five to seven times, and approximately one-half of MI and at least 70% of CVD deaths occur in patients with prior manifestations of atherosclerotic disease. Recent studies suggest that one-third of stroke patients have asymptomatic CVD, and cardiac events are the leading cause of death in stroke survivors. Patients with asymptomatic peripheral vascular disease (PVD) appear to have the same increased risk for CVD events and death as claudicates. Carotid intima-media thickness as determined by B-mode ultrasound appears to be a useful surrogate endpoint for atherosclerosis in distant beds and for clinical coronary events (42).

3) Family history

A family history of premature CVD indicates a high risk independent of traditional and nontraditional risk factors. Although heredity contributes to many risk factors (e.g. lipid concentrations, blood pressure, and obesity), it accounts for only a portion of the aggregation of CVD seen in families. As summarized by the United States National Cholesterol Education Program (NCEP) relative risk for CVD in first-degree relatives has been reported to range form 2 to as much as 12 times that of the general population. Risk increases with younger age at onset in the proband and with the number of primary relatives affected. Siblings of the proband appear to have the highest relative risk among primary relatives. Familial clustering of CVD risk most closely resembles diseases of polygenic origin and does not follow a mendelian dominant or recessive pattern that would suggest a single gene locus. Differences in

gene frequency and in demographic profile, environmental factors, and early childhood programming can all contribute to variations in risk for CVD among the population (43).

The American College of Cardiology Evaluation of Preventive Therapeutics (ACCEPT) study, a national survey conducted in 1996-1997, found that U.S. physicians do not appear to adhere to NCEP recommendations for the screening of family members of high-risk patients. Less than 1% of the screening of family members of CVD patients aged younger than 55 years. Within 6 months of the patient's cardiovascular event, family members were screened in only 17.8% of cases, and family screening was performed in only 19.6% of cases in which there was a recognized family history of premature CVD (44).

1.3.2 Major modifiable risk factors

1) Hypertension

Hypertension is established as one of the major independent risk factors for CVD and satisfies the criteria suggesting a causal relationship. Genetic and environmental factors, including factors such as weight gain, sodium intake and demographic factors such as age, sex, and ethnicity, contribute to population variations in blood pressure. Many prospective studies have related increasing SBP and DBP with risk for stroke, MI, congestive heart failure, and renal insufficiency. On average, SBP continues to rise with age, whereas DBP peaks in the fifth decade of life and then decreases. In the Multiple Risk Factor Intervention Trial (MRFIT) screens in 12 years' follow-up, both absolute and relative risk for CVD death increased with progressively higher SBP or DBP, independent of age, race, plasma cholesterol, cigarette-smoking status, or income. It is important to note that approximately two-thirds of hypertension-related CVD deaths in MRFIT occurred in individuals with only mild hypertension, and the relative risk for CVD death also increased with each succeeding SBP level in the normotensive range (45). In United States, clinical action limits for blood pressure are established by the Joint National Committee (JNC); classification of hypertension by the World Health Organization is in general accord with the JNC VI definitions (Table 3) (29).

Table 3. Classification of blood pressure in adults (≥18 years)

Category	Systolic (mmHg)		Diastolic (mmHg)
Optimal	<120	and	<80
Normal	<130	and	<85
High-normal	130-139	or	85-89
Hypertension			
Grade 1 (mild)	140-159	or	90-99
Grade 2 (moderate)	160-179	or	100-109
Grade 3 (severe)	≥180	or	≥110

Source: Guideline Subcommittee. 1999 World Health Organization-International Society of Hypertension Guidelines for the Management of Hypertension. J Hypertension 1999;17:151-83 (29).

On average, according to meta-analysis, antihypertensive drug therapy with lifestyle intervention significantly reduces the relative risk for CVD and for fatal CVD, each by 16% (2-3% for each reduction of 1 mmHg in DBP, reductions averaging 5-6 mmHg). Significant risk reduction extends even to older people with isolated systolic hypertension. Total stroke and stroke mortality rates are reduced 38% and 40%. However, the numbers of coronary and stroke deaths prevented are approximately the same because of the higher prevalence of CVD. There are clear-cut benefits in CVD risk from a decrease of even 5 or 6 mmHg in DBP. The Hypertension Optimal Treatment (HOT) trial, which enrolled 18,790 patients, from 26 countries, with hypertension and DBP of 100-115 mmHg, showed intensive lowering of blood pressure to be associated with a low rate of CVD events. The lowest incidence of major CVD events occurred at a mean achieved DBP of 82.6 mmHg; the lowest risk for CVD death occurred at 86.5 mmHg. The rate of adverse events was not increased or decreased with further reductions, although there was an apparent increase in the mortality rate when DBP was reduced to below 70 mmHg (46).

2) Dyslipidemia

Elevated plasma cholesterol

The strength of the independent, dose-response relation, the consistency of findings within and among populations and biologic plausibility all argue for elevated LDL-C as causal in the development of CVD. Perhaps the clearest demonstration of the importance of LDL-C in the pathogenesis of atherothrombotic disease is the occurrence of premature CVD events in individuals with inherited forms of hypercholesterolemia, even without the presence of other risk factors. In addition to the example from observational epidemiologic studies cited above and the major interventional trials, the meta-analysis by Law et al. (47) is of particular interest. There is remarkable consistency among large cohort studies in the relation of cholesterol to the incidence of CVD. The true relation between cholesterol concentration and CVD has tended to be underestimated because of regression dilution and surrogate underestimation. After allowance for these sources of error, analysis of the international studies shows that a 10% difference in cholesterol concentration is associated with a 38% difference in CVD mortality rate. A difference of 23 mg/dl in cholesterol in the cohort studies by this analysis would be associated with a CVD mortality difference of 54% at age 40, 39% at age 50, 27% at age 60, and 20% at age 70 (47). Traditionally, CVD risk has been considered to be 2-3% lower for each 1% decrease in TC concentration.

Although early observational studies suggested that CVD risk plateau at lower cholesterol concentrations, more recent evidence does not support such a plateau. Only in populations with a very low concentration of TC (<150 mg/dl) or LDL-C (<100 mg/dl) is a near absence of CVD events seen (47). In the Heart Protection Study, there was no evidence of a bottom LDL-C threshold beyond which statin therapy was not beneficial in preventing clinical events. Although there must be some LDL-C threshold, because cholesterol is required by the body, the LDL-C lowering in the Heart Protection Study led to clinical benefit even in patients whose values were lower than 116 mg/dl or lower than 100 mg/dl at entry into the study. Atherosclerosis identified in autopsy studies in adolescents and young adults correlates with plasma cholesterol concentration and other risk factors, and cholesterol

concentration in young adulthood predicts the development of CVD events later in life (48).

Reduced high-density lipoprotein cholesterol

In addition to genes, lifestyle factors strongly affect HDL-C concentrations, as with other lipoprotein fractions. The strong, curvilinear inverse relation between HDL-C concentration and CVD risk is well established by observational epidemiologic studies. Each 1 mg/dl increment in HDL-C is associated with a 2% risk decrement in men and 3% risk decrement in women. HDL-C also appears to be a stronger risk factor in type 2 diabetes mellitus and when low concentrations occur with elevated TG and LDL-C, and reduced HDL-C is a component of the metabolic syndrome. The HDL-C threshold for high CVD risk is much higher in patients with such additional risk factors (49).

Some evidence, including cross-sectional data on events and ultrasound assessment of carotid plaque morphology, has linked low HDL-C with increased risk for ischemic stroke (50).

Elevated plasma triglyceride

The relation between hypertriglyceridemia and atherothrombosis is complex. The hypertriglyceridemia state is heterogeneous with respect to underlying metabolic alteration. The various triglyceride-rich lipoprotein (TGRL) are heterogeneous in atherogenic potential; their metabolism is closely associated with the metabolism and blood coagulation and fibrinolytic pathways. Since the 1980s, a variety of mechanisms whereby TGRLs may be atherogenic and prothombotic have been identified. The epidemiologic association of total fasting plasma TG with CVD risk has, thus, been complicated by methodological difficulties. Association of the highest risk for CVD with moderate rather than severe hypertriglyceridemia, which can be understood in terms of the distribution of TG among different classes of plasma lipoproteins, is counterintuitive for many clinicians (51).

Meta-analysis of 17 population-based studies with an average follow-up of 8.4 years in men and 11.4 years in women, found an increase of 88.5 mg/dl in fasting TG to be associated with a 32% and 76% increased risk for incident CVD in men and women, which on multivariate analysis decreased to 14% and 37% but remained significant. Two years later, the 8 year follow-up of the prospective

Copenhagen Male Study showed that relative risk on multivariate analysis for a first CVD event was increased by 50% for the middle-third of fasting TG concentrations and more than doubled for the highest third of concentrations (52).

Lipid cut-off points are shown in Table 4 according to the current National Cholesterol Education Program (NCEP) initial classification based on TC, TG, LDL-C, and HDL-C levels (30).

Table 4. Classifications of blood lipid values in adults base on the National Cholesterol Education Program (NCEP)

Lipid (mg/dl)	Desirable level	Borderline level	High risk level
TC	<200	200-239	≥240
TG	<200	200-400	>400
LDL-C	<130	130-159	≥160
HDL-C	>35	-	<35

Source: Expert Panel on Detection, Education and Treatment of High Blood Cholesterol in Adults, Nation Cholesterol Education Program. Second report of the Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adult (Adult Treatment Panel II). Circulation 1994;89:1329-445 (30).

3) Diabetes mellitus

Major lipid abnormalities are more common in non-insulin-dependent diabetes mellitus (NIDDM) with the commonest lipid abnormality being hypertriglyceridemia with reduced HDL-C. Total cholesterol tends to be unaltered but there is more small dense atherogenic LDL-C with its increased susceptibility to oxidation. An analysis of National Health and Nutrition Examination Surveys (NHANES) I cohort data indicated that the declines in CHD mortality rate seen in the general U.S. population were not matched in subjects with diabetes. Between 1971 and 1975 and between 1982 and 1984, the age-adjusted CHD mortality rate fell by 36% among nondiabetic men and by 27% among nondiabetic women, but it fell by

only 13% among diabetic men and rose by 23% in diabetic women. It is estimated that the number of adults with diabetes in the world will be 300 million in 2025, compared with 135 million in 1995. The prevalence of diabetes in adults worldwide was estimated to be 4.0% in 1995 and is predicted to rise to 5.4% by the year 2025 (53).

Risk for all forms of CVD, including CHD, is increased substantially in type 1 as well as in type 2 diabetes mellitus, and patients with CHD are more likely to die if they have diabetes. Diabetes mellitus increases the risk for CHD by two to four times, and atherosclerosis is also rapidly progressive. Macrovascular complications are encountered more often in type 2 than in type 1 diabetes. Although multiple risk factors such as hypertension, dyslipidemia, and risk visceral obesity are common in adults with type 2 diabetes, they do not account for all the increased risk in diabetics. In a 12-year follow up in Multiple Risk Factor Intervention Trial (MRFIT), the absolute risk for cardiovascular death was much higher for diabetic than non-diabetic men at every age and risk factor level, and risk increased more steeply in the diabetic men as risk factor levels increased (54).

According to American Diabetic Association, diabetes mellitus is defined as a plasma glucose of 140 mg/dl and/or treatment of diabetes. Recently in 1997, Expert Committee on the Diagnosis and Classification of Diabetes defines diabetes mellitus as a fasting blood glucose of 126 mg/dl and/or treatment of diabetes. Multiple studies have demonstrated that the presence of diabetes mellitus (fasting plasma glucose >126 mg/dl) greatly increases the risk of cardiovascular disease (55).

4) Smoking

Cigarette smoking is the key public health challenge in developed nations. In 44 developed countries taken together, tobacco was responsible for 24% of all deaths in males and 7% of all deaths in females in 1990, rising to more than 40% in men in some former socialist economies. The average loss of life of smokers is 8 years; for those whose deaths are attributable to tobacco, it is approximately 16 years. Fifty percent of continuing cigarette smokers die of smoking-related causes approximately one-half of them in middle age (56).

Cigarette smoking can kill in at least 24 different ways. Among major chronic diseases for which smoking is a risk factor are CVD, cancers, including cancers of the lung and upper aerodigestive system, liver, pancreas, stomach, rectum,

renal pelvis, urinary bladder, uterine cervix, and penis, as well as leukemia, and nonmalignant respiratory diseases, for example, emphysema, bronchitis, and pneumonia (57).

Smoking and cardiovascular disease

Smoking is the single most important preventable cause of morbidity and mortality from CVD. In United States, approximately one-fifth of deaths from CVD is attributable to smoking, and CHD accounts for approximately one-half of these deaths. Smoking doubles to quadruples the risk for CHD event, and the risk is synergistically compounded by hypertension, hypercholesterolemia, glucose intolerance, or diabetes. Smoking confers at least a 70% excess rate of death from CHD. It increases risk for sudden cardiac death by ten times in men and 4.5 times in women. Smoking is predictive of chronotropic incompetence, and male smokers with chronotropic incompetence are at particularly high risk to suffer death or a CVD event (58).

Mechanisms of damage

Cigarette smoke contains 3000-4000 different components and each puff contains 10 free radicals. Studies in rats have demonstrated endothelial cell changes as a result of injury, and enhanced deposition of fibrinogen and lipid contributes to atherogenesis. Fibrinogen levels and platelet aggregation are increased and this leads to increased blood coagulability and viscosity. Smoking appears to reduce HDL-C and increase TG. By sympatho-adrenal stimulation, nicotine reduces coronary blood flow, most markedly in areas already compromised by ischemic damage, and this may precipitate arrhythmia or angina, potentially resulting in observed increased risk of sudden death among smokers (59).

Dose-response relation

The degree of risk of smoking is related to the number of cigarettes smoked and to cumulative consumption (age at beginning and duration) in a strong and consistent dose-response relation. Risk for MI and death from CVD is increased even with smoking only one to four cigarettes each day; thus, there is no safe level of tobacco use. Smoking so-called low-yield cigarettes does not reduce smoking-related risk for CVD (60).

5) Physical inactivity

Physical inactivity is an independent predictor of CHD and is directly related to CVD mortality rate. Manifestations of CHD are reduced in people whose jobs are physically demanding or who regularly participate in moderate to strenuous recreational activities, and survival rates are increased after MI when behavior is modified to include regular exercise. Most CVD mortality benefits of physical activity can be attained through moderately intense activity (61).

Exercise training increases maximum ventilatory oxygen uptake and decreases myocardial oxygen demands, and beneficial changes occur in hemodynamic, hormonal, metabolic, neurologic, and respiratory function. Exercise can help control diabetes and obesity and adds an independent blood pressure-lowering effect in certain hypertensive groups. Below a critical level of physical activity, chances for being overweight become substantial. Exercise training also favorably alters carbohydrate metabolism, adipose tissue distribution, and insulin sensitivity, and decreases fibrinogen concentrations. Although, importantly, unaccustomed vigorous exercise appears to be able to trigger acute coronary ischemic events, and endurance training reduces risk for thrombosis (62).

Exercise induces a number of positive changes in lipoprotein metabolism in diabetic and nondiabetic individuals. It increases HDL-C, in particular “cardioprotective”, and reduces, sometimes markedly, plasma TG. Effects on LDL-C are less consistent, although exercise often moderately lowers LDL-C, and it shifts LDL from small, dense particles to larger particles. In addition, it decreases the extent of postprandial lipemia, and endurance training increases lipoprotein lipase (LPL) activity. There appears to be little effect on Lp(a). Regular exercise in overweight individuals enhances the beneficial effect of a low-fat diet on lipid concentration. Furthermore, exercise protects against the adverse effects of dyslipidemia on the vascular wall and may improve endothelial function (63).

In conclusion, major modifiable risk factors are major risk factors of CVD and include hypertension, dyslipidemia, diabetes mellitus, smoking, and physical inactivity. For obesity is categorized as a major modifiable risk factors contributing to the major causal risk factors. Long-term prospective studies have consistently shown that excess body fat is related to increased levels of CHD. Many of the effects of

obesity, however, are mediated through increases in other characteristics and this has led to debate concerning the independence of obesity as a risk factor (64).

2. Obesity

2.1 Scope of problem

Obesity is a significant health problem in the United States and other industrialized nations. It is a complex, multifaceted chronic disease that involves metabolic, physiologic, biochemical, genetic, behavioral, social, and cultural factors and results from an imbalance between energy expenditure and caloric intake. Traditionally, obesity has been defined as an excess of body fat: 25% body fat in men and 33% in women. Obesity is associated with greater risk for a number of health problems, including cardiovascular disease, hypertension, dyslipidemia, and non-insulin dependent diabetes mellitus. It has been found to be a modifiable and preventable risk factor for these and other medical conditions that can be treated by making lifestyle modifications (65).

In the year 2000, 64.5% of the adult population in the United States was overweight (BMI of 25 kg/m^2 or higher), and 30.5% were obese (BMI of 30 kg/m^2or higher). Secular trend data suggest that obesity prevalence has been increasing steadily over the last 30 years (66). In Thailand, the fourth Nations Food and Nutrition Survey (1995) by Department of Health, Ministry of Public Health by used the BMI of 25 kg/m^2 or more to classify of obesity and found that the prevalence rate of obesity was increasing in all adult age groups. For age groups 30-39, 40-49, and 50-59 years, prevalence rate were 20.8%, 40.2%, and 35.0% respectively (3). Five year longitudinal studies of 1,019 Thai bank employee revealed that the majority of participants both male and female had experienced weight gain. The prevalence of overweight and obesity increased in both sexes (4).

At the international level, it appears that the degree of obesity is linked to the levels of urbanization, economic development, and nutrition. Although women appear to have higher rate of obesity than men, being overweight is more common in men than in women. The prevalence of obesity appears to be linked to levels of urbanization and development in particular regions. In Africa, little information has been collected on obesity trends, but there has been an increase in the rates of obesity

in the developing countries. For instance, in Mauritius the proportion of obese men increased from 3.4% to 5.3% and that of obese women form 10.4% to 15.2% over a 5-year period. In comparison, Brazil has undergone a transition from nutritional deficiencies to excess, as indicated by an increase in obesity across all groups of men (3.1% to 5.9%) and women (8.2% to 13.3%) over a 15-year period (1).

2.2 Etiology of obesity

The development of obesity is incompletely understood but involves social, behavioral, cultural, physiology, metabolic and genetic factors. Lifestyle my play a dominate role. Parents and offspring BMIs are correlated, including between biologic parents and adult adopters (but not between adoptive parents and adult adopters); furthermore, similarity of BMI is approximately twice as great in monozygotic as in dizygotic twins. The complex etiology makes the search for obesity genes challenging; several genome scanning projects with obesity phenotypes as a primary focus are currently under way (9).

It is being recognized more and more that obesity results in great measure from an environment that promotes excessive food intake and discourages physical activity through advances in technologic mechanisms that are strong in the defense against weight loss but weak against weight gain when there is abundant food supply. Nevertheless, altering the environment should not be viewed as insurmountable given the societal have been made in the United States against smoking and high–fat diets (67). Preventing the development of obesity in young adulthood and middle age should be high on the list of public health priorities, and even if weight control efforts fail in the first generation, they may succeed in later generations (9).

2.3 Risk factors associated with obesity

1) The importance of energy balance

Body weight is the integrated product of a lifetime's energy intake, offset by energy needs. Throughout the last century there has been a trend towards increased body weight and increases in body mass index. Data from the annual Health Survey for England shows that the average gain in weight of the adult population over the last 10 years has been approximately 0.35 kg/year, which is primarily adipose tissue, with

modest concomitant increases in lean tissue. At an individual level, excess weight gain may occur gradually, almost imperceptibly, over many years or, in intermittent episodes of more pronounced positive energy balance, perhaps related to holidays or festive periods when usual diet and activity habits are distorted. However, spontaneous weight loss is rare, except in association with pathological processes. This asymmetry in energy balance is underpinning the rise in obesity (76).

Energy balance is the product of both innate and discretionary processes. Energy expenditure consists predominately of three components; resting energy expenditure, thermogenesis and physical activity (77). Resting energy expenditure is a product of an individuals size, shape and body composition and accounts for 50-80 percent of energy needs. Additional energy (approximately 10 percent) is expended in the thermogenesis accompanying digestion and processing of food, or for thermoregulation. Only the energy expended in physical activity is discretionary and thus modifiable.

2) Physical activity

A comparison of contemporary living practices with historical accounts quickly reveals that habitual physical activity has declined, but data on secular trends using objective measures are lacking. Cross-sectional analyses of measures of activity and the risk of obesity consistently report a reduced weight or BMI with higher categorical levels of physical activity. This is particularly true for vigorous activity, which may reflect improved reporting of such activities in questionnaires relative to the lower intensity of activities of daily living. However, the interpretation of these cross-sectional analyses is limited due to difficulties in dissecting the directionality of the association. Physical activity may influence weight, but weight may also influence the intensity or nature of physical activity (78).

Prospective studies provide evidence that physical activity may attenuate weight gain, but there is no consistent evidence to suggest that activity can fully prevent or reverse age-associated increases in body weight. In the US Health Professionals Follow-up of almost 20,000 men those in the lowest activity category at baseline and follow up gained 1.1 kg compared to those in the highest activity category at the two time points who gained only 0.8 kg (79).

3). Energy intake

Analysis of the dietary factors associated with obesity is confounded by the difficulties in assessing food intake and eating behavior. Dietary surveys are increasingly beset by the problem of underreporting, probably related to the increased awareness of nutrition issues and concern over body weight, which leads individuals to consciously or sub-consciously mis-report their food intake.

Analysis of the dietary determinants of obesity is also confounded by the problems of post-hoc changes in consumption in response to increasing body weight. This makes it difficult to draw quantitative conclusions from cross-sectional or even prospective studies of food intake and body weight. Nonetheless increasingly refined recording tools and statistical analysis are seeking to understand more about the location and social context of eating episodes and using factor analysis to identify types of dietary patterns, which may inform future strategies to prevent and treat obesity (80).

4) Energy density

Energy density is a critical component in the regulation of human appetite and plays an important role in determining total energy intake. In the "real world" energy-dense diets are frequently high in fat, since fat (37 kJ/g) contains more than twice as much energy gram-for-gram as protein (17 kJ/g) or carbohydrate (16 kJ/g). Many low-fat foods, especially dairy products, contain substantially less energy than their full-fat equivalent, allowing consumers to maintain the bulk of food in the diet, while constraining energy intake. However, recent advances in food technology have resulted in some food ranges that are low in fat but where the energy content is similar to traditional equivalents. These foods, such as biscuits, cakes and desserts often contain large quantities of added sugars and might be expected to lead to similar passive over-consumption as high-fat foods of similar energy density.

Food served in most "fat-food" chains such as burger and chicken outlets are characterized by a particularly high energy density. These foods are frequently high in fat and have a low water content. The high energy density of these foods provides a plausible biological explanation for the epidemiological associations between "fast-food" consumption and obesity (81).

5). Portion size

The size of portions of food commonly served is increasing. This trend, previously observed only in the USA, has rapidly swept across restaurants throughout the world as part of the process of globalization. It has been exploited by marketers and now also affects household food from ready-meals to items such as crisps, confectionery and soft drinks.

A series of studies in both laboratory and free-living settings have demonstrated that large portions foster increased consumption in both adults and children of both genders and irrespective of adiposity (82).

6) Snacking

There is a clear secular decline in traditional three-meal-a-day eating patterns and a rise in more frequent, less formal eating occasions, commonly described as snacking. Epidemiological associations between eating frequency and obesity yield mixed results and heavily confounded by under-reporting of energy intake and hence potentially under-reporting of eating episodes too. In day-to-day life the impact on body weight is likely to be determined by the quantity and quality of snacks consumed rather than by eating frequency (82).

7) Soft drinks

The emphasis on dietary fat as an important determinant of energy density has tended to divert attention from sugar-rich foods and drinks. However, it is important to recognize the energy density theory of appetite control cannot be equally applied across solid and liquid foods alike. Liquids have a lower energy density than solids because of their high water content, yet there is poorer energy compensation following isoenergetic liquids relative to solid food, perhaps due to differences in viscosity (82).

It is evident that increases in soft drink consumption, as part of broader diet and lifestyle change, have paralleled the rise in obesity. Sugar-rich drinks are frequently alleged to be a determinant of obesity risk but within cross-sectional surveys there is little evidence of an association. Two free-living studies have tested the effects on sugar-rich drinks on body weight. In the first, daily consumption of 1150 g soda sweetened with a high-fructose corn syrup (530 kcal/day) versus aspartame (3 kcal/day) or no beverage, over 3 weeks each, showed that the high-fructose corn syrup drink significantly increased energy intake and body weight relative to both the

aspartame and control treatments (83). Secondly, a 10 week trial examined the impact of sugar-rich versus artificially-sweetened foods and drinks in which >80 percent of all intervention foods were beverages. Over 10 weeks, weight increased by 1.6 kg in the high sugar group and decreased by 1.0 kg in the group in whom sugar rich foods and beverages were replaced by artificially sweetened varieties ($p < 0.001$). Overall, this data supports the hypothesis that consumption of sugar-rich drinks is a risk factor for obesity (84).

2.4 Assessing of obesity

There are several methods of body-composition analysis, including anthropometry (skinfold measurements, waist to hip ratio, waist circumference, and BMI), near infrared interactance (NIA), hydrostatic weighing, air displacement, dual energy x-ray absorptiometry (DEXA), and bioelectrical impedance (BIA).

2.4.1 Direct methods

1) Near-infrared interactance (NIA)

In this method, a fiber optic probe is connected to a digital analyzer that measures the tissue composition (fat and water) at various sites on the body. This method is based on studies that show optical densities are linearly related to subcutaneous and total body fat. The biceps is the most often used single site for estimating body fat using the NIA method. The NIA light penetrates the tissues and is reflected off the bone bake to the detector. The NIA data is entered into a prediction equation with the person's height, weight, frame size, and level of activity to estimate the percent body fat.

This method has become popular outside of the laboratory because it is simple, fast, noninvasive, and relatively inexpensive. However, the amount of pressure applied to the fiber optic probe during measurement may affect the values of optical densities, and skin color and hydration level may be potential sources of error. To date, studies conducted with this method have produced mixed results: a high degree of error has occurred with very lean and very obese people, and the validity of a single-site measurement at the biceps is questionable. Much more research is needed to substantiate the validity, accuracy, and applicability of this method, and it is therefore not recommended (68).

2) Hydrostatic weighing

Hydrostatic weighing measures whole-body density by determining body volume. There is a variety of equipment available to do underwater weighing ranging in sophistication from the standard stainless steel tank with a chair or cot mounted on underwater scales to a chair and scale suspended from a diving board over a pool or hot tub.

The individual is first weighed outside the tank, then immersed totally in water and weighed again. Since bone and muscle are more dense and fat is less dense than water, an individual with more bone and muscle will weigh more in water and therefore have a higher body density and lower percentage of body fat than an individual who has a higher proportion of body fat. The volume of the body is calculated and the individual's body density is determined using standard formulas. Body fat percentage is then calculated from body density using standard equations.

The underlying assumption with this method is that densities of fat mass and fat-free mass are constant. However, underwater weighing may not be the appropriate gold standard for everyone. For example, athletes tend to have denser bone and muscles than non-athletes, which may lead to an underestimation of body fat percentage. At the same time, the body fat of elderly patients suffering form osteoporosis may be overestimated. To date, specific equations have not been developed to accommodate these different population groups.

Another important consideration in this method is residual lung volume, which can be estimated or measured, but a direct measure is desirable and it should be taken in the tank whenever possible (68).

3) Air displacement

Based on the same principle as underwater weighing, the BOD POD is a fiberglass plethysmograph that measures body volume by changes in pressure in a closed chamber. The system uses computerized sensors to measure how much air is displaced while a person sits within the chamber. It uses a calculation to determine body density, and then estimates body fat. The system is safe, relatively quick, results are easily replicated, and the chamber can accommodate a wide range of body shapes and sizes. However, the equipment is very expensive and limited in availability. Another drawback to this method is that subjects need to be clothed in very thigh-

fitting attire, including a tight-fitting swim cap, for accurate measurement. Since the equipment is measuring air displacement, uncovered scalp hair and loose clothing can underestimate body fat by more than two percent and five percent, respectively (68).

4) Dual energy x-ray absorptiometry (DEXA)

The use of DEXA for body composition analysis is based on a three-compartment model that divides the body into total body mineral, fat-free lean mass and fat tissue mass. DEXA uses a whole body scanner that has two low-dose x-ray at different sources that read bone and soft tissue mass simultaneously. The sources are mounted beneath a table with a detector overhead. The scanner passes across the individual's reclining body and data is collected at 0.5-cm intervals. The full scan takes 10 and 20 minutes. It is safe and noninvasive, although the individual must lie still throughout the procedure.

DEXA is becoming regarded as the new "gold standard" in body composition analysis because it provides a higher degree of precision in only one measurement, and can also show the distribution of fat tissue throughout the body. It is very reliable and the results are reproducible. However, the equipment is not widely available, although it is moving more from the laboratory and into clinical settings. In evaluating fat mass by DEXA as compared to bioelectrical impedance or skinfold thickness, body fat is underestimated by the other methods as compared to DEXA, with better precision obtained by the DEXA. In nonobese patients, skinfold thickness or bioelectrical impedance is appropriate for routine monitoring, but DEXA may be the method of choice in obese patient monitoring, since reproducibility gains special importance (68).

5) Bioelectrical impedance (BIA)

Bioelectrical impedance analysis is a relatively quick, simple, and fairly accurate way to determine body composition. For the analysis, the patient only needs to remove the shoe and sock on one foot. Gel electrodes are placed on the hands and feet, and a very mild electrical current is sent though the body. Muscle, because it contains water and electrolytes, will conduct the current; fat tissue acts as an insulator and resists the current. The differences in conduction between the two tissues provide the machine with a measure of electrical impedance, which is then applied to a mathematical formula to calculate lean body mass. The entire procedure takes only a

few minutes, and the instrument provides information on fat mass in pounds and percentage of total bodyweight, lean body mass in pounds and percent, basal metabolic rate based on lean body mass, and target weight.

During the first week of caloric restriction, there is loss of body weight in excess of the loss of lean and fat tissue due to a water diuresis. If patients are measured at their first visit and then frequently thereafter, it is possible to find that patients are apparently gaining fat as they lose weight using BIA. Since lean body mass is assessed based on both body water and muscle, the loss of water leads to an apparent decrease in lean body mass, which in most cases exceeds the loss of fat in the first week of dieting, leading to a seeming increase in percent body fat. The bioelectrical impedance measurement is most useful at first visit for assessing type of obesity, and not useful for multiple serial determinations.

A second potential problem is overemphasis on the quantitative accuracy of body fat estimation. Small changes cannot be measured using this device, and it is important to stress this fact to patients. The changes observed in percent fat often do not impress patients as much as the ratio of the absolute change in fat mass in pounds compared to changes in lean mass (68).

2.4.2 Indirect methods

1) Skinfold thickness measurement

Using hand-held calipers that exert a standard pressure, the skinfold thickness is measured at various body locations. A calculation is then used to derive a body fat percentage based on the sum of the measurements. The caliper method is based upon the assumption that the thickness of the subcutaneous fat reflects a constant proportion of the total body fat and that the sites selected for measurement represent the average thickness of the subcutaneous fat. Skinfold measurements are made by grasping the skin and underlying tissue, shaking it to exclude any muscle, and pinching it between the jaws of the calipers. Duplicate readings are often made at each site to improve the accuracy and reproducibility of the measurements. Generally speaking, skinfold measurements are easy, inexpensive, and portable. However, results can be very subjective as precision ultimately depends on the skill of the person making the measurement and the site measured. The quality of the calipers is also a factor; they should be accurately calibrated and have a constant specified pressure.

Inexpensive models sold for the home use are usually less accurate than those used by an accredited caliper technician. The more obese the subject, the more difficult it is to "pinch" the skinfold correctly, requiring even more skill to obtain an accurate measurement (69).

2) Waist to hip ratio

Since the pioneering work of Jean Vague in the 1940s, it has slowly become accepted that different body morphology or types of fat distribution are independently related to the health risk associated with obesity. Starting with Jean Vague's brachio-femoral adipo-muscular ratio as an index of fat distribution (which was based on a ratio of skinfolds and circumferences of the arms and thighs), more recent indices have been adopted to predict specifically intra-abdominal fat. The most popular among all measures is the waist to hip circumference ratio (69).

Over the last 10 years, a high WHR (WHR >1.0 in men and >0.85 in women) has become accepted as the clinical method of identifying patients with abdominal fat accumulation (13). However, some experts consider that the hip measurement contains additional valuable information related to gluteofemoral muscle mass and bone structure (11). The WHR may therefore remain a useful research tool but individuals can be identified as being at increased risk of obesity-related illness by using the waist circumference alone as an initial screening tool (1).

3) Waist circumference (WC)

Waist circumference is a convenient and simple measurement that is unrelated to height, correlates closely with BMI and WHR, and is an approximate index of intra-abdominal fat mass and total body fat (11). Furthermore, changes in WC reflect changes in risk factors of CVD (10). Waist circumference is measured at the midpoint between the lower border of the ribcage and the iliac crest (Table 5) (1).

Table 5. Sex-specific cut-off points for waist circumference that denote "increased risk" and "substantially increased risk" of metabolic complications associated with obesity in Caucasians

	Risk of obesity-associated metabolic complications	
	Increased	Substantially increased
Men	≥94 cm (~ 37 inches)	≥102 cm (~ 40 inches)
Women	≥80 cm (~ 32 inches)	≥88 cm (~ 35 inches)

Source : WHO. The World Health Report Organization Report of a WHO consultation on obesity. Obesity: preventing and managing the global epidemic. Geneva, WHO. 1998 (1).

4) Body mass index

Body mass index (BMI) is a simple index of weight–for–height that is commonly used to classify overweight and obesity in adults. It is calculated as the weight in kilograms divided by the square of the height in meters (kg/m^2). BMI has a high correlation with adiposity, but it does not quantify total body adiposity or convey information concerning regional fat distribution. However as reviewed by Kuczmarski and Flegal (67), BMI is an easily obtained measure that has been recommended for use in all age groups. Most clinical studies assessing the health effect of overweight and obesity rely on BMI. From a public health perspective, the use of BMI cut-off points to define overweight and obesity is a necessary to help describe populations. BMI is an index that can be used for comparison across studies both in the United States and internationally. Although it may be useful to use similar criteria when comparing data from different studies and countries, health risks associated with overweight and obesity are part of a continuum and at a given BMI may vary when a specific population is observed. These BMI cut-off points should be considered as a guide to allow for the comparisons among various populations and over time. Currently, the National Institutes of Health (65), Healthy People 2010 (70), the 2002 Dietary

Guidelines for America (71), and WHO (1) all use similar cut-off points of BMI for defining overweight (BMI ≥25 kg/m^2) and obesity (BMI ≥30 kg/m^2).

Internationally, there has been some deliberation concerning the universal application of these cut-off points of overweight and obesity. A report cosponsored by the WHO Western Pacific Region (26) recently recommended different ranges for classifying regions. Increase in health related risk factors and co-morbidities associated with obesity occur at a lower level in Asian populations than in other ethnic groups. By contrast, Pacific Islanders appear to be more muscular and have comparably lower levels of body fat at a given BMI. Thus, on the basis of the respective health related risk factors and co-morbidities in these populations, lower cut-off points for Asians were identified for overweight (BMI ≥23 kg/m^2) and obesity (BMI ≥25 kg/m^2) (Table 6).

Some support for cut-off points comes from data on Chinese living in Hong Kong (72). Similar data have been published from the Chinese in Singapore (73) and on Indian Asians living in Mauritius, where there is a significantly increased risk of Type 2 diabetes and hypertension among those with a BMI between 23 to 24.9 kg/m^2 compared to those within the normal range. Clearly these cut-off points do not apply to Pacific Islanders. In these populations, higher cut-off points are required to define overweight and obesity of BMI ≥26 kg/m^2 and BMI ≥32 kg/m^2 respectively (74).

Relative risk of obesity associated health problems

Although obesity should be considered as a disease in its own right, it can also be recognized as one of the key risk factors for other non-communicable diseases, such as CVD and non-insulin dependent diabetes mellitus (NIDDM). The key risk factors for other non-communicable are obesity, high blood pressure, hypercholesterolemia and smoking (7).

The more life-threatening, chronic health problems associated with obesity fall into four main areas (Table 7) (1):

1. Cardiovascular problems including hypertension, stroke and CHD.
2. Conditions associated with insulin resistance, namely NIDDM.

3. Certain types of cancers, mainly the hormonally related and large-bowel cancer.

4. Gallbladder disease.

Table 6. Classification of overweight in adult Europids and Asians according to BMI

Classification	BMI		Risk of co-morbidities
	Europids	Asians	
Underweight	<18.5	<18.5	Low (but increased risk of other clinical problems)
Normal range	18.5 – 24.9	18.5 –22.9	Average
Overweight	≥ 25	≥ 23	
Pre –obese	25 –29.9	23 –24.9	Increase
Obese I	30 –34.9	25 –29.9	Moderate
Obese II	35 –39.9	≥ 30	Severe
Obese III	≥ 40		Very Severe

Source : The Asia-Pacific perspective : Redefining obesity and its treatment. 2000 (26).

It is important to recognize that ethnic differences will have a bearing on the prevalence of a particular disease; some minority populations in the USA have a higher prevalence of certain obesity-related disease (particularly NIDDM but, for black Americans, also CVD, stroke and osteoarthritis of the knee) compared with the white population (65). Nevertheless, the absolute prevalence may vary, the relative risk of any particular disease (whether the risk is slightly, moderately or greatly increased for an obese person compared to a lean person) is fairly consistent throughout the world.

Table 7. Health risk associated with obesity (WHO 1998)

Greatly Increased (RR > 3)	Moderately Increased (RR 2-3)	Mildly Increased (RR 1-2)
-Type 2 diabetes -Gallbladder disease -Dyslipidemia -Metabolic Syndrome -Breathlessness -Sleep apnea	-Coronary heart disease -Hypertension -Osteoarthritis (knees and hips) -Hyperuricemia and gout	-Cancer (breast cancer in post menopausal women, colon cancer) -Reproductive hormone -Abnormalities polycystic ovary syndrome -Impaired fertility -Low back pain -Increase anaesthetic risk -Fetal defects associated with maternal obesity

Source: WHO. The World Health Report Organization Report of a WHO consultation on obesity. Obesity: preventing and managing the global epidemic. Geneva, WHO. 1998 (1).

3. Obesity and cardiovascular risk factors

Several cardiovascular risk factors are associated with obesity including hypertension and dyslipidemia. Many studies investigating the effect of obesity on cardiovascular risk factors are based on Europids and show increase in risk associated with high BMI (1, 7, 67). In Asia, risks of the same magnitude occur at a lower BMI. In Japan, there is an increases in mortality from CHD for those with a BMI >30 kg/m^2. If other risk factors such as hypertension, diabetes mullitus and dyslipidaemia are also present, then there is a higher risk of CHD morbidity among those with a BMI of between 25 and 29.9 kg/m^2 (75).

3.1 Hypertension

The association between hypertension and obesity is well documented. Both systolic and diastolic blood pressures increase with BMI and obese individuals are at a higher risk of developing hypertension than are lean subjects. The reason for the

association between increased body weight and elevated blood pressure is unclear. One possibility is that obesity is associated with higher circulating levels of insulin which enhances renal retention of sodium, resulting in increased blood pressure (31). As exercise is known to improve insulin sensitivity, this would perhaps explain why exercise also reduces blood pressure. Other possible etiological factors include elevated plasma remain or enchanted catecholamine activity (32).

Blood pressure is increased by 6 mmHg systolic and 4 mmHg diastolic for a 10 % gain in body fat with those genetically more susceptible showing the greater effect. Reisen et al. (9) have demonstrated that a weight loss of 11 kg produced a 20% decrease in both systolic and diastolic pressure in hypertensive patients even when the sodium intake was kept constant. It would appear that, as a general rule, blood pressure is reduced by 1 mmHg systolic and 2 mmHg diastolic for each 1% reduction in body weight (25).

The relationship of BMI and hypertension

Both systolic and diastolic blood pressure increase with BMI and obese individuals are at higher risk of developing hypertension than are lean subjects. In obese adults aged 20–45 years, the relative risk of developing high blood pressure is five to six times greater than in lean individuals. A weight reduction of as little as 3 – 5 kg can significantly reduce blood pressure for both treated and untreated individuals and can eliminate the need for pharmacologic agents in up to 50 % of cases. As there currently is insufficient research to suggest a specific body weight target for hypertension on an individual basis, relying on the expert panel's guidelines for healthy (70).

In Japan, there is a two- fold increase in the risk of hypertension for those with a BMI >25.0 kg/m^2 compared to those with a BMI 22.0 kg/m^2 (39). It has recently been suggested that the optimal BMI cut-off point to predict hypertension in Hong Kong Chinese is 23.8 kg/m^2 among men and 24.1 kg/m^2 for women (34).

3.2 Dyslipidemia

The most characteristic dyslipidaemia in obesity is elevated total cholesterol and triglycerides, high LDL-C and low HDL-C. A meta-analysis of 70 published studies on the effects of weight loss on plasma lipid concentrations showed a decrease

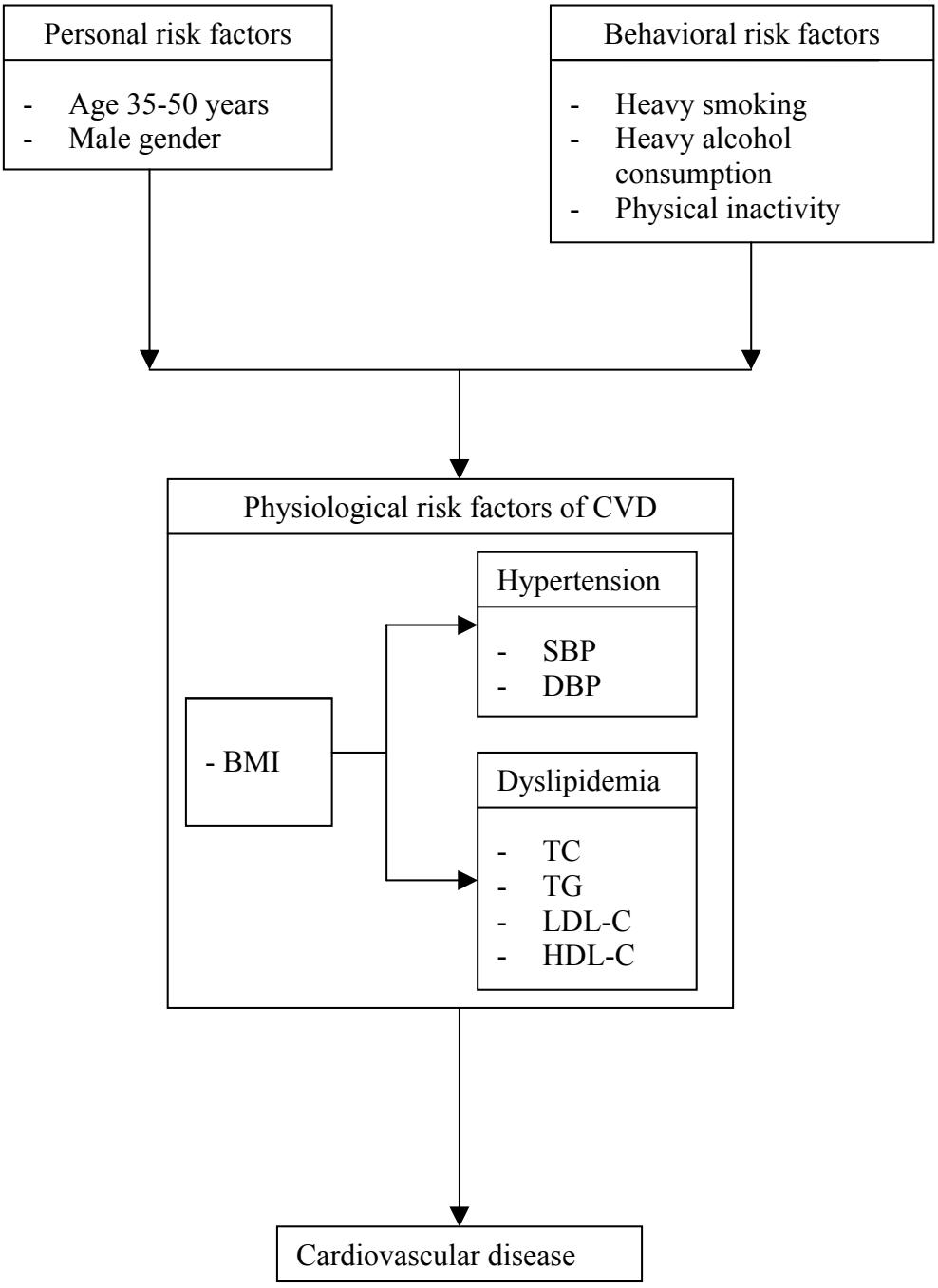
of 1.9 mg/dl in total cholesterol, 0.8 mg/dl in LDL-C and 1.3mg/dl in triglycerides and increase in HDL-C of 0.27 mg/dl during active weight loss and 0.35 mg/dl on weight stabilization for each 1 kg of weight loss (36).

The relationship of BMI and dyslipidemia

The lowest mortality related to cardiovascular disease occurs in individuals who remain lean throughout life. A healthy weight-one that optimizes the normal lipidemia corresponds to a BMI of 22.6 kg/m^2 for men and 21.1 kg/m^2 for women. If this BMI were achieved there would be 25% fewer cases of coronary heart disease and 35% fewer strokes or episodes of cardiac failure. Weight loss of 205 in obese individuals would reduce cardiovascular risk by 40% (69).

In Japan, there is a two–fold increase in the risk of hypertriglyceridemia, hypercholesterolemia and decreased HDL cholesterol in those with a BMI >25.0 kg/m^2 compared to those with a BMI of 22.0 kg/m^2 (39). It has recently been suggested that the optimal BMI cut-off point to predict dyslipidemia in Hong Kong Chinese is 23.0 kg/m^2 among men and 24.1 kg/m^2 for women. (34).

Conceptual framework of the study



CHAPTER III
MATERIALS AND METHODS

Research Design

This is a cross sectional study. The study was designed to examine the BMI cut-off point levels, which reflect the risk factors of CVD (SBP, DBP, TC, TG, LDL-C and HDL-C) in the urban Thai male population.

Population and Sample

The population in this study were Thai nationality, male employees who undertook annual physical check ups by the Office of Public Health and Environmental Technology Service, Faculty of Public Health, Mahidol University during 2nd February – 31st March 2004. The subjects were purposive sampling as describe by the following criteria:

Inclusion criteria

1. Males aged 35 – 50 years old who live in Bangkok
2. Thai nationality

Exclusion criteria

1. The subjects who were taking medication for controlling of blood pressure and/or plasma lipid.
2. The subjects who were heavy smokers as smoking 16 rolls per day or more.
3. The subjects who were heavy, daily consumer alcohol.
4. The subjects who have any complication conditions or diseases that induce increase or decrease weight change such as:
   - Renal disease
   - Liver disease
   - Thyroid disease
   - Post orthopedic operation with plate and screw

Sample Size

The sample size was determined by the following formula:

$$n = \frac{Z^2_{\alpha/2} P Q}{d^2}$$

n = The sample size

$Z_{\alpha/2}$ = The standard estimate under normal curve at $\alpha = 0.1$ and $Z_{\alpha/2} = 1.645$

P = The prevalence of at least one of five physiological risk factors for CVD was 42.2 (five physiological risk for CVD are SBP and/or DBP≥140/90 mmHg, FBS >126 mg/dl, TC >200 mg/dl and HDL-C <35 mg/dl). This data were collected from 249 male employees of the Government Saving Bank in 2000 by Temcharoen (5).

Q = 1- P = 0.58

d = Error between sample and population.
= 10% of P = 0.042

Calculation of formula:

$$n = \frac{(1.645)^2 (0.42) (0.58)}{(0.042)^2}$$

$$= 373.69$$

Therefore, a minimum of 374 samples were necessary in this study.

Research Instruments

1. Self-administered questionnaires for collecting personal general information.

Part I Demographic data: age, nationality, religion, marital status and educational level.

Part II Health data: health history, smoking habits, alcohol consumption behaviors and exercise habits.

2. Anthropometric data

Body weight was measured using a beam balance scale.

Body height was measured using a measuring height instrument (fixed to the beam balance scale).

3. Blood pressure was measured using a mercury manometer.

4. Laboratory measurements

Blood samples were collected by laboratory instruments (syringe 5 ml, needle No. 23, standard sampling tube laboratory) and analyzed by blood chemical analysis.

Collection of Data

1. The self-administered questionnaires were provided to subjects on the examination day. Incomplete questionnaires would be sent back to subjects to complete.

2. Body weight was measured to the nearest 0.1 kilogram. Being, the subjects stand in the center of the weighing platform with wearing light clothes, with emptied pocked and without shoes.

3. Body height was measured to the nearest 0.1 centimeter. The subjects were barefoot in a standing position with feet together, arms to the side, legs straight, shoulders relaxed and head in the Frankfort horizontal plane (looking straight ahead). This plane is represented by a line between the lowest point on the margin of the orbit (the bony socket of the eye) and the tragion (the notch above the tragus, the cartilaginous projection just anterior to the external opening of the ear). Heels, buttocks, scapular (shoulder blades), and the back of the head all rested against the vertical surface of the height instrument.

4. BMI was calculated by the body weight in kilograms divided by the body height in meters squared (kg/m^2).

5. Blood pressure was measured in the sitting position from the left arm after at least 5 minutes of physical rest. The subjects, who have high blood pressure, SBP $\geq$ 140 mmHg and/or DBP $\geq$90 mmHg, repeated the measurement of blood pressure again after at least 30 minutes of physical rest.

6. Blood sampling and analysis.

A blood sample volume of 3-5 ml was collected. Blood samples were obtained from vein of the subjects in the morning after at least 8 hours of overnight fast and at least 24 hours of drinking alcohol. The following quantitative assays were performed:

- Serum levels of total cholesterol, triglyceride and high density lipoprotein cholesterol were assayed by enzymatic colorimetric test.

- Low density lipoprotein cholesterol was calculated from Freidewald's formula.

LDL-C (mg/dl) = TC – HDL-C – (TG/5)

Type of Variables

1. Independent variables
   - Body mass index
2. Dependent variables
   - Systolic blood pressure
   - Diastolic blood pressure
   - Plasma total cholesterol
   - Plasma triglyceride
   - Plasma low-density lipoprotein cholesterol
   - Plasma high-density lipoprotein cholesterol
3. Controlled variables
   - Age
   - Nationality
   - Personal illness
   - Taking medication controlling blood pressure and plasma lipid
   - Smoking
   - Alcohol drinking

Statistical Analysis

The data were analyzed by the Statistical Package for Social Science (SPSS) program.

1. The number of distribution and percentage were used to analyze the demographic data and health data.

2. The mean and standard deviations were used to analyze the weight, height, BMI, blood pressure, plasma TC, plasma TG, plasma LDL-C and plasma HDL-C.

3. The multiple regression analysis was used to find the effect of the confounding variables on blood pressure, plasma TC, plasma TG, plasma LDL-C and plasma HDL-C.

4. Sensitivity and specificity analysis were used to find the optimal cut-off points of BMI which reflect the risk factors of CVD. The receiver operating characteristic (ROC) curve analysis were plotted using measures of sensitivity and 100- specificity for each BMI cut-off value (Appendix B).

Sensitivity is the ability of a test to correctly identify those who have the disease; the percentage of those who have the disease and are proven to have the disease as demonstrated by a test. Sensitivity shows the proportion of truly diseased persons in a population who underwent screening and who are correctly identified as being diseased by the screening test (88).

Specificity is the ability of a test to correctly identify the percentage of those who do not have the disease; those who do not have the disease and are proven to not have the disease as demonstrated by a test. Specificity shows the proportion of nondiseased persons in a population who underwent screening and who are correctly identified as not being diseased by the screening test (88).

An ROC curve is a plot of a test's sensitivity (plotted on the y axis) versus its false positive fraction (FPF), or (100 – specificity and plotted on the x axis). Each point on the graph is generated by a different decision threshold.

In this study the optimal cut-off points of BMI for each of the risk factors of CVD were denoted by high in sensitivity value and each value of sensitivity and specificity being more than 50%. For the each of the risk factors of CVD, the sensitivity associated with each optimal BMI cut-off value was greater than its attendant specificity. The overall performance of each ROC curve was evaluated by calculating the area under the curve (AUC), which is a measure of the diagnostic power of a test, and describes the probability that a test will correctly identify subjects with the disorder. The closer the curve to the (0, 100) point (left upper corner), the greater the AUC and the better test (89).

Ethical Issue

The research was conducted after the final permission was granted by the Committee on Human Rights Related to Human Experimentation, Faculty of Graduated Studies, Mahidol University. The researcher was well aware of research ethics. Therefore, the utilization of the data collected from the subjects would be used in consideration of the integrity, value, and possible effects on the subjects. As such, before the data collection process began, the potential subjects would have explained to them about the research objectives and the data collected from them. They would be assured that their participation in the study was based purely on a voluntary basis and that they could withdraw from the study at any time. They also had a chance to ask questions for clarification, and they were asked to sign the informed consent from (Appendix A) before data collection started.

CHAPTER IV
RESULTS

This research aimed to examine the cut-off points of BMI which reflect the risk factors of CVD (high in BP, TC, TG, LDL-C and low in HDC-C) in Thai males populations who live in Bangkok. The results of the study are presented in 4 parts as follows:

1. Characteristics of the samples
   1.1 General characteristics
   1.2 Health behavior characteristics
2. Health status of the samples
   2.1 Anthropometric data and body mass index
   2.2 Blood pressure
      2.2.1 Systolic blood pressure
      2.2.2 Diastolic blood pressure
   2.3 Lipidemia
      2.3.1 Total cholesterol level
      2.3.2 Triglyceride level
      2.3.3 Low density lipoprotein cholesterol level
      2.3.4 High density lipoprotein cholesterol level
3. The effect of confounding age, smoking habits, alcohol drinking behavior and BMI on variables
4. The optimal cut-off points of BMI which reflect the risk factors of CVD

1. Characteristics of the samples

1.1 General characteristics

General characteristics of 413 samples in terms of age, marital status, and educational level are shown in Table 8. The age range of samples was 35 to 50 years with a mean of 43 years. Most of them were married and had completed Bachelor degree education level.

Table 8. The number and percentage distribution of general characteristics of samples

Characteristic	Number	Percentage
Age (years)		
35 - 40	144	34.9
41 - 45	119	28.8
46 - 50	150	36.3
Total	413	100.0
Mean ± SD	43.05 ± 5.01	
Min – Max	35 – 50	
Marital Status		
Single	71	17.2
Married	290	70.2
Divorced / Widowed / Separated	52	12.6
Total	413	100.0
Educational level		
Diploma	109	26.4
Bachelor degree	274	66.3
Masters degree or higher	30	7.3
Total	413	100.0

1.2 Health behavior characteristics

Heath behavior characteristics of the samples in terms of smoking habits, alcohol drinking behavior, and exercise habits are shown in Table 9. More than 50 percent of samples were smokers by 29.5 percent smoked 1-5 rolls per day, 19.9 percent smoked 6-10 rolls per day and 7.5 percent smoked ≥11 rolls per day.

In regards to alcohol drinking behavior of the samples, it was found that 51.3 percent of the samples drank alcohol ≤1 time per month, and 16.5 percent of the samples did not drink alcohol.

For exercise habits, 46.5 percent of the samples did not exercise. There were 12.3 percent of the samples exercised ≥3 days per week and ≥30 minutes per time, while 5.6 percent were exercised ≥3 day per week but <30 minutes per time.

Table 9. The number and percentage of smoking habits, alcohol drinking behavior and exercise habits of samples

Characteristic	Number	Percentage
Smoking habits		
No	178	43.1
Yes		
1 - 5 rolls / day	122	29.5
6 -10 rolls / day	82	19.9
11 -15 rolls / day	31	7.5
Total	413	100.0
Alcohol drinking behavior		
No	68	16.5
Yes		
≤1 time / month	212	51.3
1 time / week	87	21.1
2 – 3 times / week	37	8.9
≥4 times / week	9	2.2
Total	413	100.0

Table 9. (Continued)

Characteristic	Number	Percentage
Exercise habits		
No	192	46.5
Yes		
<3 days / week	147	35.6
≥3 days / week and <30 minutes / time	23	5.6
≥3 days / week and ≥30 minutes / time	51	12.3
Total	413	100.0

2. Health status of the samples

2.1 Anthropometric data

2.1.1 Body weight and body height

Most of samples had a body weight 60 to 60.9 kg. The minimum body weight was 44 kg, while 110 kg was the maximum recorded body weight. The mean of body weight was 67.87±10.89 kg (Table 10).

For body height, the average of body height was 168.32±6.43 cm. Body height ranged from 153 cm for the minimum and 186 cm for the maximum. Most of samples had a body height of 160 to 169 cm (Table 10).

Table 10. The number and percentage by weight and height of samples

Variable	Number	Percentage
Body weight (kg)		
<50	11	2.7
50 –59.9	66	16.0
60 –69.9	172	41.6
70 –79.9	112	27.1
80 – 89.9	34	8.2
≥90	18	4.4
Total	413	100.0
Mean ± SD	67.87 ± 10.59	
Min – Max	44 - 110	
Body height (cm)		
<160	31	7.5
160 – 169	215	52.0
170 – 179	156	37.8
≥ 180	11	2.7
Total	413	100.0
Mean ± SD	168.32 ± 6.43	
Min – Max	153 - 186	

2.1.2 Body mass index

The body mass index of the samples was classified by two criteria: WHO and Asia criteria (Table 11). According to WHO criteria, the results found that most of the samples were normal weight with a BMI of 18.5–24.9 kg/m^2. While, 27.9 percent of the sample found to be pre-obese (BMI 25.0-29.9 kg/m^2) and 4.8 percent were obese level I (BMI 30.0-34.9 kg/m^2). By Asia criteria, the results found that most of the samples were in the overweight group with a BMI ≥23 kg/m^2. 25.1 percent of

the samples were pre-obese (BMI 23.0-24.9 kg/m^2), 27.9 percent were obese level I (BMI 25.0-29.9 kg/m^2) and 4.8 percent of the samples were obese level II (BMI≥30.0 kg/m^2). The mean BMI of the samples was 23.33 ± 3.21 kg/m^2.

Table 11. The number and percentage of BMI of samples classified by WHO and Asia criteria

Variable	Number	Percentage
BMI (WHO)		
Underweight (<18.5 kg/m^2)	23	5.6
Normal (18.5 – 24.9 kg/m^2)	255	61.7
Overweight (≥25.0 kg/m^2)	135	32.7
Pre-obese (25.0-29.9 kg/m^2)	115	27.9
Obese I (30.0-34.9 kg/m^2)	20	4.8
Obese II (35-39.9 kg/m^2)	0	0.0
Obese III (≥40 kg/m^2)	0	0.0
Total	413	100.0
BMI (Asia)		
Underweight (<18.5 kg/m^2)	23	5.6
Normal (18.5 – 22.9 kg/m^2)	151	36.6
Overweight (≥23 kg/m^2)	239	57.8
Pre-obese (23.0-24.9 kg/m^2)	104	25.1
Obese I (25.0-29.9 kg/m^2)	115	27.9
Obese II (≥30.0 kg/m^2)	20	4.8
Total	413	100.0
Mean ± SD	23.93 ± 3.21	
Min – Max	16.71 – 34.33	

2.2 Blood pressure and BMI

2.2.1 Systolic blood pressure and BMI

The results found that the mean systolic blood pressure of the samples was 125.16 ± 12.16 mmHg (100–160 mmHg) (Table 12). The percentage that had systolic blood pressure at hypertensive levels were 19.6 percent. The mean of body mass index of the hypertensive group was 26.21 ± 3.46 kg/m^2, and 32.3 percent of samples had high-normal systolic blood pressure. The results showed that BMI level increased proportionately with SBP.

2.2.2. Diastolic blood pressure and BMI

The samples had diastolic blood pressure of between 60-100 mmHg with a mean of 80.29 ± 8.13 mmHg (Table 12). An analysis to identify the samples who had diastolic blood pressure of hypertensive level were 24.4 percent. The mean body mass index was 25.85 ± 3.33 kg/m^2. There were 50.4 percent of the samples who had normal diastolic blood pressure and the mean body mass index was 23.72 ± 2.91 kg/m^2. The results showed that BMI level increased proportionately with DBP.

Table 12. The mean BMI of the study subjects classified by blood pressure level.

Variables	Number	Percentage	BMI Mean ± SD
SBP (mmHg)			
Optimal (<120mmHg)	84	20.3	22.84 ± 3.29
Normal (<130mmHg)	115	27.8	23.00 ± 2.78
High-normal (130-139mmHg)	133	32.3	24.02 ± 2.58
Hypertension (≥140mmHg)	81	19.6	26.21 ± 3.46
Mean ± SD	125 ± 12.16		
Min – Max	100 – 160		

Table 12. (Continued)

Variables	Number	Percentage	BMI Mean ± SD
DBP (mmHg)			
Optimal (<80mmHg)	104	25.2	22.48 ± 2.74
Normal (<85mmHg)	208	50.4	23.74 ± 2.91
High-Normal (85-89mmHg)	-	-	-
Hypertension (≥90mmHg)	101	24.4	25.85 ± 3.30
Mean ± SD	80.29 ± 8.13		
Min – Max	60 – 100		

2.3 Lipidemia and BMI

2.3.1 Total cholesterol level and BMI

The mean level of total cholesterol was 221.07 ± 39.22 mg/dl (range was 113–398 mg/dl) (Table 13). The results showed that 24.7 percent of the samples had total cholesterol in an acceptable level, 47.7 percent had a borderline total cholesterol level and 27.6 percent had a high-risk total cholesterol level. The mean of body mass index classified by total cholesterol level 22.60 ± 3.60, 23.82 ± 2.77 and 25.28 ± 3.03 kg/m^2, respectively. The results revealed that BMI level increased proportionately with TC.

2.3.2 Triglyceride level and BMI

The mean level of triglyceride was 162.35 ± 91.27 mg/dl (47–596 mg/dl) (Table 13). More than 70 percent of the samples had an acceptable triglyceride level, 23.3 percent had a borderline triglyceride level and 2.4 percent had a high-risk triglyceride level. The mean of body mass index classified by triglyceride level were 23.36 ± 3.02, 25.69 ± 3.13 and 24.39 ± 3.05 kg/m^2, respectively. These results indicated that BMI levels did not increase proportionately with TG.

2.3.3 Low-density lipoprotein cholesterol level and BMI

The mean level of low-density lipoprotein cholesterol was 139.40 ± 37.06 mg/dl (44.2–269.8 mg/dl) (Table 13). The results showed that 38.7 percent of the samples had an acceptable low-density lipoprotein cholesterol level, 33.5 percent had a borderline low-density lipoprotein cholesterol level and 27.8 percent had a high-risk level. The mean of body mass index classified by low-density lipoprotein cholesterol level were 23.25 ± 3.57, 23.79 ± 2.91 and 25.02 ± 2.70 kg/m^2, respectively. These results indicated that BMI levels increased proportionately with LDL-C.

2.3.4 High-density lipoprotein cholesterol level and BMI

The mean level of high-density lipoprotein cholesterol was 49.16 ± 12.63 mg/dl (18-99 mg/dl) (Table 13). The results showed that 90.1 percent of the samples had an acceptable high-density lipoprotein cholesterol level and 9.9 percent had a high-risk high-density lipoprotein cholesterol level. The mean of body mass index classified by high-density lipoprotein cholesterol level were 23.74 ± 3.14 and 25.63 ± 3.34 kg/m^2, respectively. These results suggested that BMI levels reversed proportionately with HDL-C.

Table 13. The mean BMI of the study subjects, classified by lipid level

Variables	Number	Percentage	BMI Mean ± SD
TC (mg/dl)			
Acceptable (<200 mg/dl)	102	24.7	22.60 ± 3.60
Borderline (200-239 mg/dl)	197	47.7	23.82 ± 2.77
High-risk (≥240 mg/dl)	114	27.6	25.28 ± 3.03
Mean ± SD	221.07 ± 39.22		
Min – Max	113 – 398		

Table 13. (Continued)

Variables	Number	Percentage	BMI Mean ± SD
TG (mg/dl)			
Acceptable (<200 mg/dl)	307	74.3	23.36 ± 3.02
Borderline (200-400 mg/dl)	96	23.3	25.69 ± 3.13
High-risk (>400 mg/dl)	10	2.4	24.39 ± 3.50
Mean ± SD	162.35 ± 91.27		
Min – Max	42 – 596		
LDL-C (mg/dl)			
Acceptable (<130 mg/dl)	160	38.7	23.25 ± 3.57
Borderline (130-159 mg/dl)	138	33.5	23.79 ± 2.91
High-risk (≥160 mg/dl)	115	27.8	25.02 ± 2.71
Mean ± SD	139.40 ± 37.06		
Min – Max	44.2 – 269.8		
HDL-C (mg/dl)			
Acceptable (>35 mg/dl)	372	90.1	23.74 ± 3.14
High-risk (<35 mg/dl)	41	9.9	25.63 ± 3.34
Mean ± SD	49.16 ± 12.63		
Min – Max	18 – 99		

3. The effect of confounding age, smoking habits, alcohol drinking behavior and BMI on the variables

Form the literature review found that age, smoking habits, alcohol drinking behavior and BMI were effected to blood pressure and lipidemia. Therefore, to ensure that the effect of age, smoking habits, alcohol drinking behavior and BMI were controlled, the multiple regression analysis was used in this study.

Table 14. The effect of confounding age, smoking habits alcohol drinking behavior and BMI on the variables

Variables	Confounding Factors	R	R^2	R^2 Change	F
SBP	Age Smok Alc	0.270	0.073	-	0.000**
	Age Smok Alc BMI	0.425	0.181	0.108	0.000**
DBP	Age Smok Alc	0.248	0.062	-	0.000**
	Age Smok Alc BMI	0.406	0.165	0.103	0.000**

Table 14. (Continued)

Variables	Confounding Factors	R	R^2	R^2 Change	F
TC	Age Smok Alc	0.532	0.283	-	0.000**
	Age Smok Alc BMI	0.552	0.305	0.022	0.000**
TG	Age Smok Alc	0.610	0.372	-	0.000**
	Age Smok Alc BMI	0.623	0.388	0.016	0.000**
LDL-C	Age Smok Alc	0.375	0.140	-	0.000**
	Age Smok Alc BMI	0.395	0.156	0.016	0.000**

Table 14. (Continued)

Variables	Confounding Factors	R	R^2	R^2 Change	F
HDL-C	Age Smok Alc	0.198	0.039	-	0.000**
	Age Smok Alc BMI	0.217	0.047	0.008	0.000**

Note: ** Significant at p < .01, Smok = Smoking habits, Alc = Alcohol drinking behavior, BMI = Body mass index

Table 14 showed that the age, smoking habits and alcohol drinking of samples effects SBP and DBP; and the unique effects exerted by BMI on these two risk factors are larger than 10%. For TC, TG, LDL-C and HDL-C the results showed that these factors were slightly but still significantly effected by BMI after controlling age, smoking habits and alcohol drinking behavior.

4. The optimal cut-off points of BMI which reflect the risk factors of CVD

Epidemiological methods have been used to examine the cut-off points of BMI which reflect the risk factors of CVD. Figure 1-6 and Table 15 showed the epidemiological methods to identify the optimal cut-off points of BMI which reflect high in SBP, DBP, TC, TG and LDL-C and low in HDL-C, the risk factors of CVD, by receiver operatic characteristic (ROC) curve analysis.

4.1 The optimal cut-off points of BMI and hypertension

4.1.1 Systolic blood pressure

The ROC curves for the cut-off points of BMI which reflect high systolic blood pressure was illustrated in Figure 1.

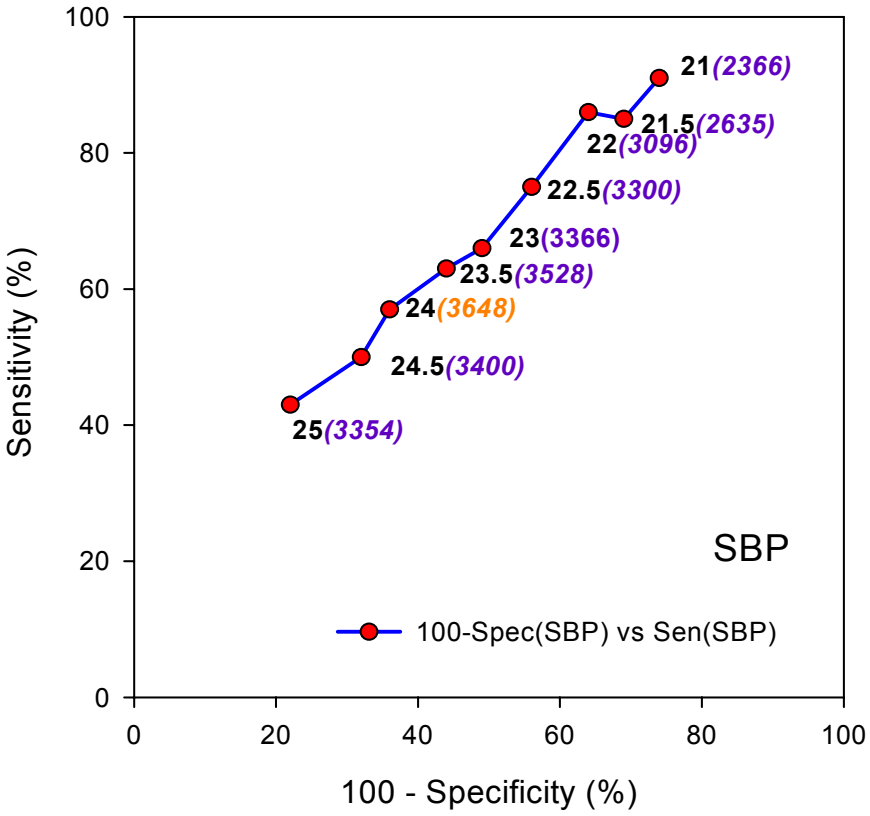


Figure 1. The receiver operating characteristic (ROC) curve for the cut-off points of BMI which reflect high systolic blood pressure

The cut-off point of BMI which had the most of area under the curve (AUC) was 24 kg/m^2 (AUC = 3648) but it had sensitivity value (57%) lower than specificity value (64%). According to the criteria for this study, the optimal cut-off points of BMI for each the risk factors of CVD were denoted by high in sensitivity value and each value of sensitivity and specificity were more than 50%. For the each of risk factors of CVD, the sensitivity associated with each optimal BMI cut-off value was greater than its attendant specificity. Moreover the optimal cut-off points would had the most of area under ROC curve. When, the cut-off point of BMI at 23.5 kg/m^2 was considered, it had sensitivity value (63%) higher than specificity value (56%) and AUC = 3528.

Considering, the cut-off point of BMI at 23 kg/m^2 also had sensitivity value (66%) higher than specificity value (51%) but it had AUC (3366) which was lower than the cut-off point of BMI at 23.5 kg/m^2. According to the epidemiological methods criteria for this study, the optimal cut-off point of BMI which reflects high systolic blood pressure was 23.5 kg/m^2.

4.1.2 Diastolic blood pressure

The ROC curves for the cut-off points of BMI which reflect high diastolic blood pressure was illustrated in Figure 2.

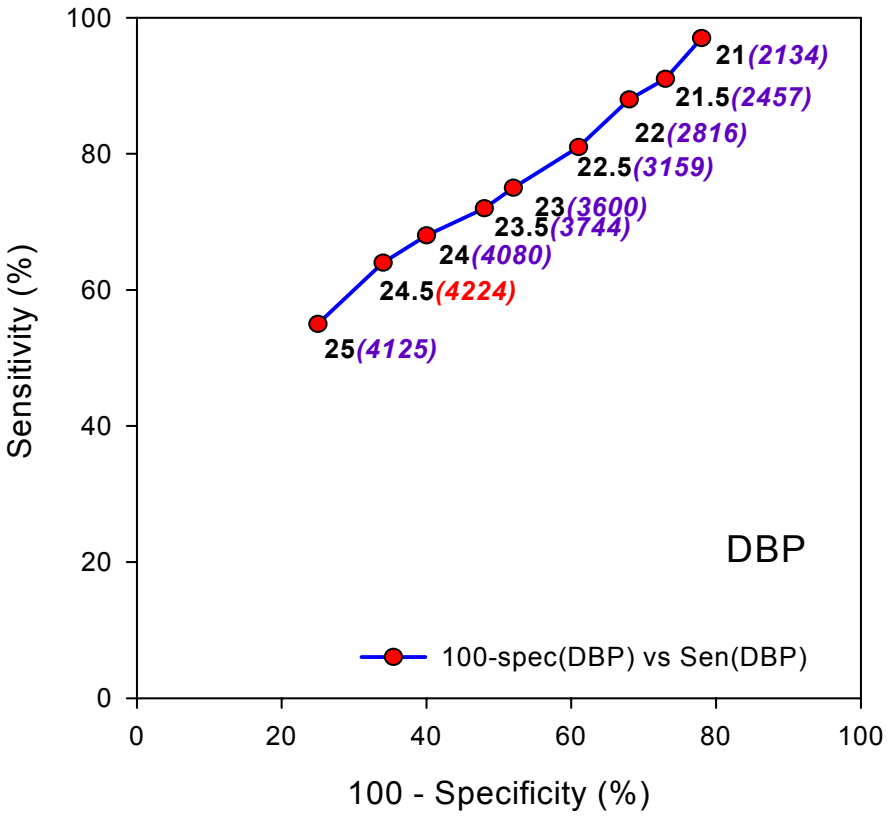


Figure 2. The receiver operating characteristic (ROC) curve for the cut-off points of BMI which reflect high diastolic blood pressure

The cut-off point of BMI which had the most of area under the curve (AUC) was 24.5 kg/m^2 (AUC = 4224) but it had sensitivity value (64%) lower than specificity value (66%). Considering, the cut-off point of BMI at 24.0 kg/m^2 found that the sensitivity was 68% and specificity value was 60% (AUC = 4080), it would be the optimal cut-off point of BMI for reflect high DBP. When the cut-off point of BMI at

23.5 kg/m^2 was considered, it found that sensitivity value was 72% and specificity value was 52% (AUC = 3744). However, the cut-off point at 23.5 kg/m^2 had sensitivity value (72%) more than the cut-off point at 24.0 kg/m^2 (68%). According to the epidemiological methods criteria for this study, the optimal cut-off point of BMI which reflects high diastolic blood pressure was 23.5 kg/m^2.

4.2 The optimal cut-off points of BMI and dyslipidemia

4.2.1 Total cholesterol level

The ROC curves for the cut-off points of BMI which reflect high total cholesterol level was illustrated in Figure 3.

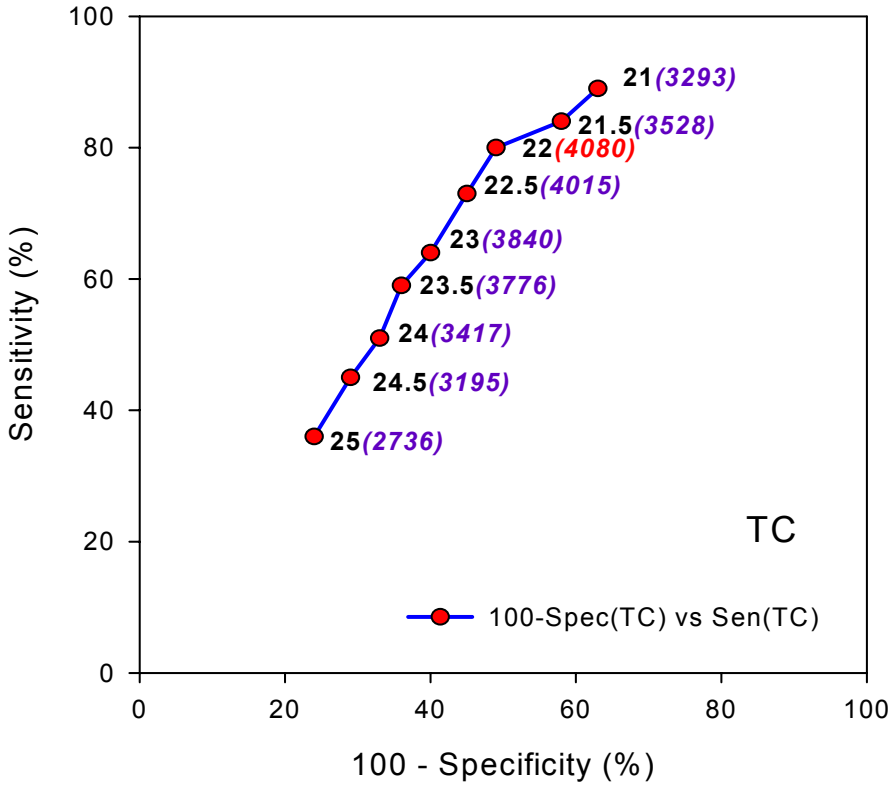


Figure 3. The receiver operating characteristic (ROC) curve for the cut-off points of BMI which reflect high total cholesterol level

The cut-off point of BMI which had the most of area under the curve (AUC) was 22.0 kg/m^2 (AUC = 4080), sensitivity value was 80% and specificity value was 51%. According to the epidemiological methods criteria for this study, the optimal cut-off points of BMI which reflect high diastolic total cholesterol level was 22.0 kg/m^2.

4.2.2 Triglyceride level

The ROC curves for the cut-off points of BMI which reflect high triglyceride level was illustrated in Figure 4.

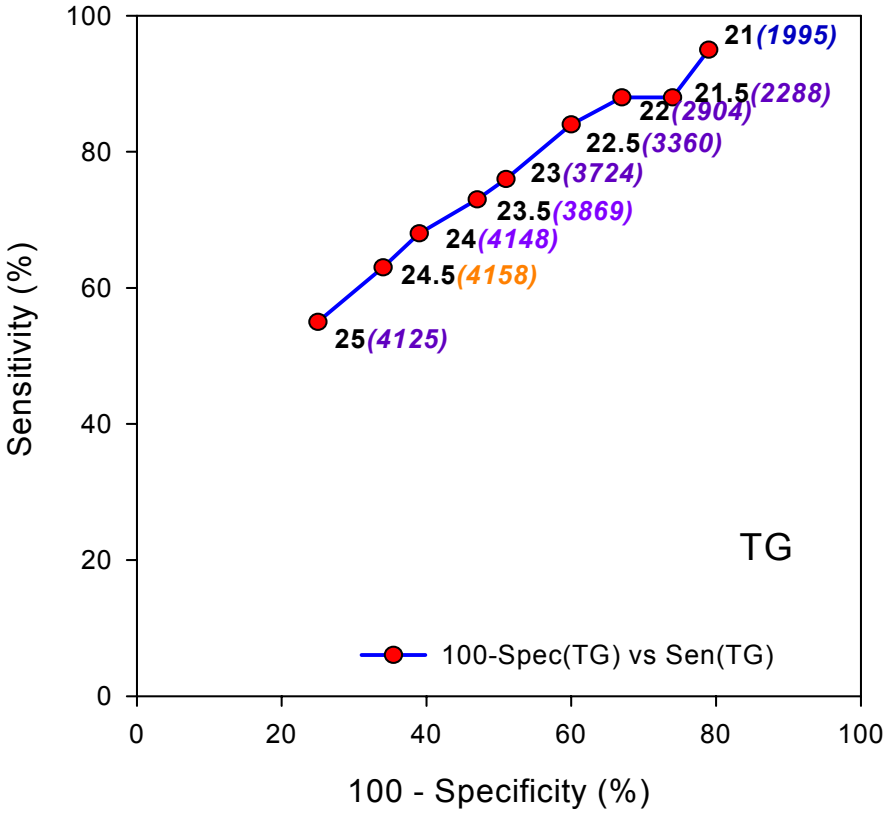


Figure 4. The receiver operating characteristic (ROC) curve for the cut-off points of BMI which reflect high triglyceride level

The cut-off point of BMI which had the most of area under the curve (AUC) was 24.5 kg/m^2 (AUC = 4158) but it had sensitivity value (63%) lower than specificity value (66%). Considering, the cut-off point of BMI at 24.0 kg/m^2 found that the sensitivity value was 68% and specificity value was 61% (AUC = 4148), it would be the optimal cut-off point of BMI for reflect high TG. When the cut-off point of BMI at 23.5 kg/m^2 was considered, it found that sensitivity value was 73% and specificity value was 53% (AUC = 3869). However, the cut-off point at 23.5 kg/m^2 had sensitivity value (73%) more than the cut-off point at 24.0 kg/m^2 (68%). According to the epidemiological methods criteria for this study, the optimal cut-off points of BMI which reflect high triglyceride level was 23.5 kg/m^2.

4.2.3 Low-density lipoprotein cholesterol level

The ROC curves for the cut-off points of BMI which reflect high low-density lipoprotein cholesterol level was illustrated in Figure 5.

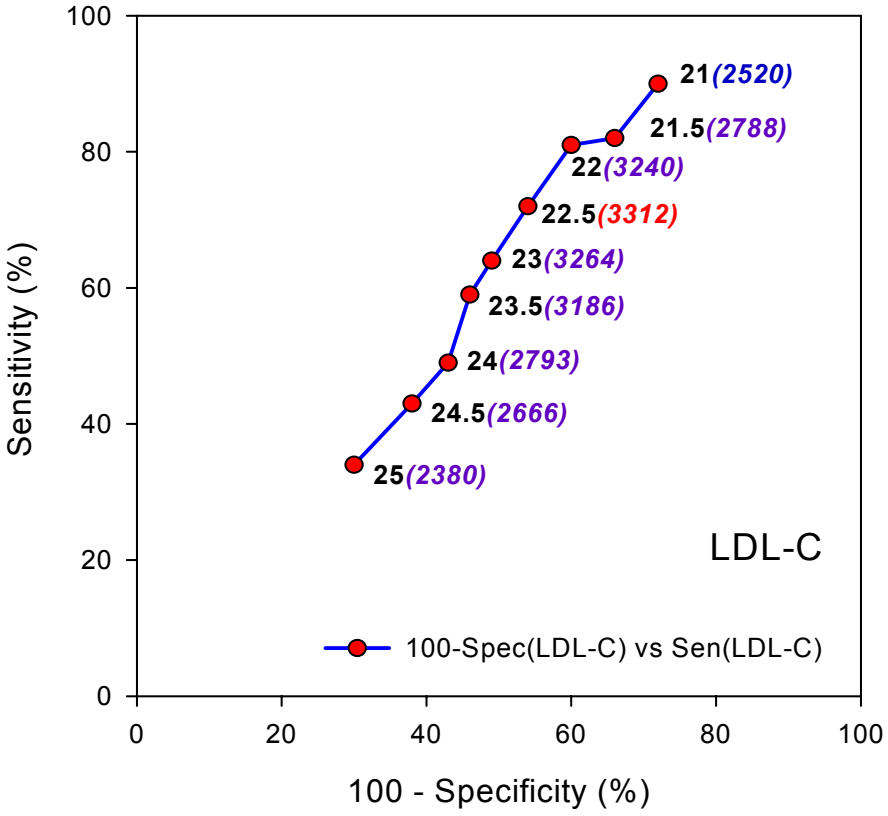


Figure 5. The receiver operating characteristic (ROC) curve for the cut-off points of BMI which reflect high low-density lipoprotein cholesterol level

The cut-off point of BMI which had the most of area under the curve (AUC) was 22.5 kg/m^2 (AUC = 3312) but it had specificity value (46%) lower than 50%. When the cut-off point of BMI at 23.0 kg/m^2 was considered, it had sensitivity value 64% and specificity value 51% (AUC = 3264). According to the epidemiological methods criteria for this study, the optimal cut-off points of BMI which reflect high LDL-C level was 23.0 kg/m^2.

4.2.4 High-density lipoprotein cholesterol level

The ROC curves for the cut-off points of BMI which reflect low high-density lipoprotein cholesterol level was illustrated in Figure 6.

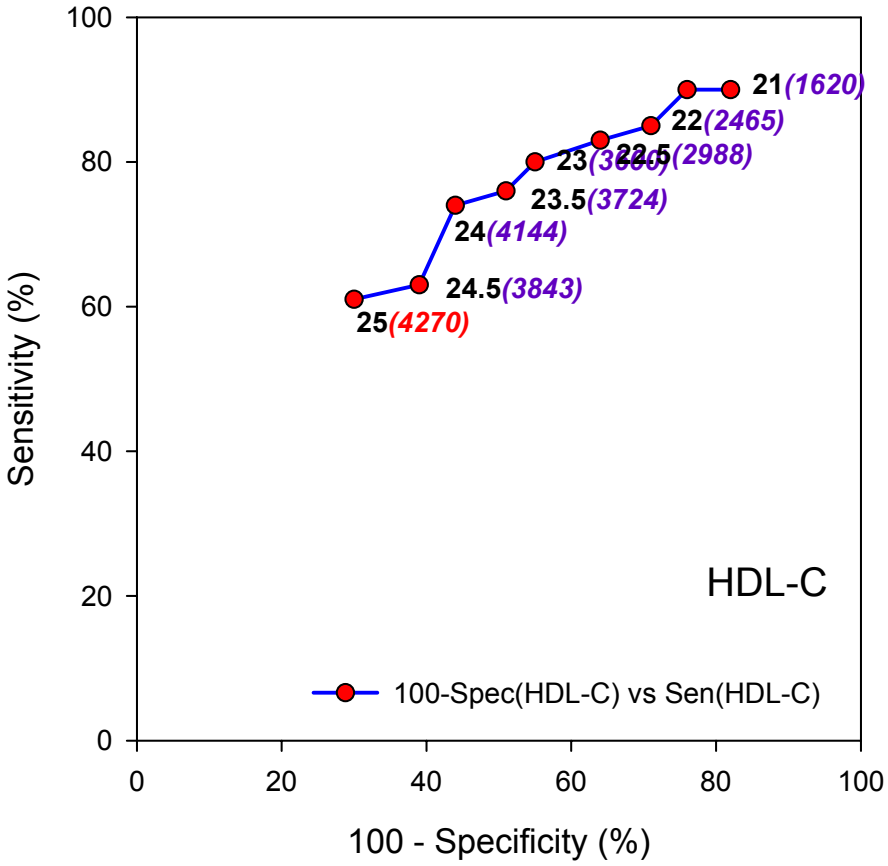


Figure 6. The receiver operating characteristic (ROC) curve for the cut-off points of BMI which reflect low high-density lipoprotein cholesterol level

The cut-off point of BMI which had the most of area under the curve (AUC) was 25.0 kg/m^2 (AUC = 4270) but it had sensitivity value (61%) lower than specificity value (70%). Considering, the cut-off point of BMI at 24.5 kg/m^2 found that the sensitivity value was 63% and specificity value was 61% and AUC was 3843. When the cut-off point of BMI at 24.0 kg/m^2 was considered, it found that sensitivity value was 74%, specificity value was 56% and AUC was 4144. The results showed that the cut-off point at 24.0 kg/m^2 had sensitivity value and AUC more than the cut-off point at 24.5 kg/m^2. According to the epidemiological methods criteria for this study, the optimal cut-off points of BMI which reflect low HDL-C level was 24.0 kg/m^2.

Table15. The sensitivity and specificity of the samples by SBP, DBP, TC, TG, LDL-C, HDL-C to identify those with the cut-off points of BMI

	SBP		DBP		TC		TG		LDL-C		HDL-C	
BMI	Sens. (%)	Spec. (%)	Sens. (%)	Spec. (%)	Sens. (%)	Spec. (%)	Sens. (%)	Spec. (%)	Sens. (%)	Spec. (%)	Sens. (%)	Spec. (%)
25.0	43	78	55	75	36	76	55	75	34	70	61	70
24.5	50	68	64	66	45	71	63	66	43	62	63	61
24.0	57	64	68	60	51	67	68	61	49	57	**74**	**56**
23.5	**63**	**56**	**72**	**52**	59	64	**73**	**53**	59	54	76	49
23.0	66	51	75	48	49	60	76	49	**64**	**51**	80	45
22.5	75	44	81	39	73	55	84	40	72	46	83	36
22.0	86	36	88	32	**80**	**51**	88	33	81	40	85	29
21.5	85	31	91	27	84	42	88	26	82	34	90	24
21.0	91	26	97	22	89	37	95	21	90	28	90	18

In conclusion, the optimal cut-off points of BMI which reflect to each of the risk factors of CVD are listed in Table 16. The BMI cut-off values for reflect hypertension was 23.5 kg/m^2 and dyslipidemia were range from 22 to 24 kg/m^2.

Table 16. The optimal cut-off values, sensitivity, specificity, and area under curve of BMI indices to predict hypertension and dyslipidemia based on ROC curve analysis

	Males (N= 413)			
CVD risk factors	Cut-off	Sensitivity	Specificity	AUC
Hypertension				
- SBP	23.5	63	56	3528
- DBP	23.5	72	52	3744
Dyslipidemia				
- TC	22.0	80	51	4080
- TG	23.5	73	53	3869
- LDL-C	23.0	64	51	3264
- HDL-C	24.0	74	56	4144

5. The cut-off points of BMI according to WHO and Asia criteria

Comparison of the sensitivity of cut-off points of BMI which detects the risk factors of CVD between WHO criteria (≥25 kg/m^2) and Asia criteria (≥23 kg/m^2). The results showed that the cut-off point of BMI at ≥25 kg/m^2 had the sensitivity lower than the cut-off point of BMI at ≥23 kg/m^2. Therefore, the sensitivity of the cut-off point of BMI in Asia criteria is better than the WHO criteria for detecting the risk factors of CVD for these samples.

Table 17. The sensitivity of the samples by SBP, DBP, TC, TG, LDL-C and HDL-C to identify the cut-off point of BMI according to WHO criteria (≥25 kg/m^2) and Asia studies criteria (≥23 kg/m^2)

	Sensitivity (%)					
BMI	SBP	DBP	TC	TG	LDL-C	HDL-C
25	43	55	36	55	34	61
23	66	75	49	76	64	80

CHAPTER V

DISCUSSION

This cross sectional study examined the cut-off points of BMI which reflect the risk factors of CVD including hypertension and dyslipidemia. The 413 participants were Thai males aged 35-50 years and who live in Bangkok. They all work in the office, which involves low levels of physical activity.

Weight-height indexes are well accepted as indexes for body fat content, especially for BMI. Therefore, most researchers in nutrition use BMI for the assessment of nutritional status. The universal BMI classification proposed by the WHO suggested cut-off points for overweight (BMI $\geq$25 kg/m^2) and obesity (BMI $\geq$30 kg/m^2). These criteria were based on observational studies in Europe on the relationship between morbidity and mortality with BMI, and so may not be applicable to Thai populations. Because, the BMI have limitations for its use as length of legs or trunk in relation to overall stature. In addition BMI cannot differentiate the components of body composition, such as FM and FFM of subjects (15). Consequently BMI can be large for those with short legs in proportion to their height and for those with high muscularity. Consideration of these limitations is important when comparing BMI values obtained across a range of people each quite different to each other (16).

Most of the samples were married males with a mean age of 43.05 years old. The majority of them have completed a Bachelor degree (Table 8). Age is an independent risk factor for CVD. The risk for a clinical CVD event is high in older than in younger people. Because, the atherosclerotic lesion or plaque developed over a long period of time, which is in line with the culmination of processes that may have been developing a series of changes to the walls of the coronary arteries. The severity of coronary atherosclerosis rises with age, hence older people on average, have a greater plaque burden than younger persons. This factor supports the well known claim that age is a risk factor for CHD. Use of age as an indicator of plaque burden has

been generally acceptable for primary prevention (64). Eighty-five percent of Americans who die of CVD are aged 65 or older. In addition, men are at greater risk than women for CVD event (64).

The respondents were the government officials who working in offices, were physically inactive (sedentary lifestyles) and most of them did not exercise (Table 9). Physical inactivity is directly related to CVD mortality rate. Manifestation of CVD are reduced in people whose jobs are physically demanding or who regularly participate in moderate to strenuous recreational activity and survival rates are increased after myocardial infarction when behavior is modified to include regular exercise. Most CVD mortality benefits of physical activity can be attained through moderately intense activity (61). Exercise induces a number of positive changes in lipoprotein metabolism in both diabetic and non-diabetic individuals. It increases HDL-C in particular "cardioprotective" and reduces, sometimes markedly, plasma TG. Effects on LDL-C are less consistent, although exercise often moderately lower LDL-C (63).

Furthermore, the majority of the respondents were smokers and drinkers, which were the modifiable behavioral risk factors for CVD (Table 9). For participants who smoked, most of them smoked 1-5 rolls per day and the maximum number smoked was 15 rolls per day but this study did not limit the cigarettes commutative consumption (age at beginning and duration). Cigarette smoking appears to reduce HDL-C, increase TG (59) and risk for death from CVD is increased even when smoking only one to four rolls of cigarettes each day. Thus, there is no safe level of cigarettes smoking (60).

In regards to alcohol consumption, the potential participants who were heavy alcoholic drinkers, as defined as those drinking alcohol everyday and or those drinking alcohol within 24 hours before blood samples were obtained, were excluded from the study group. The respondents who drank alcohol, most of them drank only once pre month or more and drank on a maximum of 4 occasions per week. However, this study did not limit the amount of alcohol drunk per occasion. Alcohol produced a beneficial rise in HDL-C but only at moderate intakes beyond which there is a concomitant rise in TG and LDL-C. The effect contributes to the J-shaped relation between alcohol and total mortality rate. Lowest mortality occurred in those who consume 1 or 2 drink per day and total mortality rises rapidly with increasing number of drinks consumed as

these exceed 3 drinks per day (59). The NCEP recommend that daily alcohol consumption should not exceed approximately 30 g for men, the amount in approximately 2 drinks and approximately 15 g, or 1 drink, for women (59).

According to the WHO classification of obesity, using a BMI ≥30 kg/m^2, the prevalence of obesity was approximately 10-20% and 15-25% in European men and women, respectively (1). By contrast, the prevalence of obesity using the same criteria for Asian populations is less than 3% in Japan and China (69). Based on these definitions, Asian populations, such as the Chinese, are often considered as a non-obese population. Previous cross-ethnic studies that examined the BMI-%BF relationship in respect to age, gender and ethnicity have reported that Asians tend to possess greater %BF than Caucasians at the same BMI values (17-20). There are also reports that Asians have higher health morbidities at lower BMI values than Caucasians. In Asians, the cut-off point of BMI for overweight (≥23.0 kg/m^2) and obesity (≥25.0 kg/m^2) are lower than the WHO criteria (overweight ≥25.0 kg/m^2 and obesity ≥30.0 kg/m^2) (21-25).

This study illustrated that use of the Asia criteria of BMI ≥23 kg/m^2 demonstrated almost a twice greater prevalence of overweight in the respondents when compared using the WHO criteria of BMI ≥25 kg/m^2. Therefore, the cut-off point of BMI according to the Asia criteria may be appropriate for the Thai male population. Therefore, this finding supports using lower BMI criteria to define obesity.

These results (Table 12) showed that the BMI levels increased positively with both systolic blood pressure and diastolic blood pressure. The relationship of which remained statistically significance after excluding the effect of age, smoking habits and alcohol drinking behaviors (Table 14). More than 50 percent of the samples were high normal and hypertensive in systolic blood pressure. For subjects who had systolic blood pressure at the hypertensive level, an average BMI of 26.21±3.46 kg/m^2 was recorded. Those with diastolic blood pressure at the hypertensive level had an average BMI of 25.85±3.30 kg/m^2. Both systolic and diastolic blood pressure increased with BMI and obese individuals are at higher risk of developing hypertension than lean subjects (68). The benefits of a 10 kg loss of weight showed a decrease of 10 mmHg in SBP and 20 mmHg in DBP (36). Moreover, hypertension increased in prevalence with increasing age.

The most characteristic dyslipidemia in obesity are elevated levels of TC, TG, LDL-C and low levels of HDL-C. An elevated plasma cholesterol concentration has been shown to be a major risk factor for CHD. Hypercholesterolemia is estimated to account for 40% of all MI. Moreover, for each 10 mg/dl increased in serum cholesterol, the overall death rate increased by 5% and the cardiovascular related death rate by 9% (84). Eventhough major factors affecting these biochemical parameters were age, smoking habits, alcohol drinking behaviors and BMI played a minor rate but its effects were still of statistical significance after controlling age, smoking habits and alcohol drinking behaviors (Table 14).

In this study, it was showed that the BMI levels were positively increased with TC level. The samples who had TC in high-risk levels (≥240 mg/dl), recorded an average BMI of 25.28±3.03 kg/m^2. The benefits of a 10 kg loss of weight showed a decrease of 10 percent in TC (35).

Triglyceride level is well accepted as a coronary risk factor. The studies of men surviving a MI showed that approximately 8% had elevated cholesterol levels, 7% had elevated TG and 15% had combined elevations of cholesterol and TG (85).

This study showed that the BMI levels did not positively increase with TG level. The samples who had TG in high-risk levels (>400 mg/dl), had an average BMI of 24.39±3.50 kg/m^2. The samples who had TG in borderline levels (200-400 mg/dl), yielded an average BMI of 25.69±3.13 mg/m^2. These results were not consistent with other studies, because the number of samples, who had TG in the high-risk levels, represented only 2.4 percent of the total sample group. The benefits of a 10 kg loss of weight showed a decrease of 30 percent in TG (35).

Low-density lipoprotein cholesterol increased with age and weight gain. They also may be elevated in the presence of hypothyroidism, nephrotic syndrome, and estrogen deficiency. Moreover, the concentration of TC or LDL-C in the blood is significantly associated with subsequent CHD morbidity and mortality, especially in middle-age men (86).

In this study, it was showed that the BMI levels positively increased with LDL-C level. The samples who had LDL-C in the high-risk levels (≥160 mg/dl), had an average BMI of 25.02±2.71 kg/m^2. The benefits of a 10 kg loss of weight showed a decrease of 15 percent in LDL-C (35).

A low high-density lipoprotein cholesterol level is a major risk factor for CHD, but a high HDL-C is a negative risk factor. In multiple clinical trials, for every 1 mg/dl increase in HDL-C, a 2-3 percent reduction of risk for CHD occurred (87).

In this study, it was showed that BMI levels were negatively increased with HDL-C level. The samples who had HDL-C in the high-risk levels (<35 mg/dl), had an average BMI was 25.63±3.34 kg/m^2. The benefits of a 10 kg loss of weight showed an increase of 8 percent in HDL-C (35).

This finding (Table 13) illustrated that only 24.7 percent of samples had TC in acceptable levels, while 90.1 percent of samples had HDL-C in acceptable levels so that it may result in to high level of TC, the majority of which were in borderline and high-risk level. However, only 38.7 percent of the samples who had LDL-C in acceptable levels and more than 60 percent had LDL-C in borderline and high-risk levels. It was interesting to note that the cut-off point of BMI according to the Asia criteria detected the overweight proportion of the samples as 57.8 percent (the WHO criteria detected only 32.7 percent), the percentage of which is similar to that of the samples who had LDL-C in a borderline and high-risk level.

The optimal cut-off points of BMI which reflect the risk factors of CVD

The optimal cut-off points of BMI and hypertension

Results from this study suggested that the optimal cut-off points of BMI which reflect hypertension (both systolic blood pressure and diastolic blood pressure) to be 23.5 kg/m^2. The sensitivity and specificity of these values to predict hypertension were more than 60% and 50%, respectively. The Hong Kong study suggested that the optimal BMI cut-off points to predict hypertension in Hong Kong Chinese was 23.8 kg/m^2 among men and 24.1 kg/m^2 among women (34). The Singapore study suggested that the optimal BMI cut-off point to predict hypertension was 23.4 kg/m^2 in Singaporean women (88).

The optimal cut-off points of BMI and dyslipidemia

This study suggested the optimal cut-off points of BMI which reflect elevated TC at 22.0 kg/m^2, elevated TG at 23.5 kg/m^2, elevated LDL-C at 23.0 kg/m^2, and reduced HDL-C at 24.0 kg/m^2. The sensitivity and specificity of these values to

predict dyslipidemia were more than 80% and 50%, respectively. The Hong Kong study suggested that the optimal BMI cut-off points to predict dyslipidemia in Hong Kong Chinese was 23.0 kg/m^2 among men and 24.1 kg/m^2 among women (34). The Singapore study suggested that the optimal BMI cut-off point to predict dyslipidemia was 23.9 kg/m^2 in Singaporean women (88).

Alternatively, the difference in findings may reflect the different decision rules used to determine cut-off values. In this study, the optimal cut-off points of BMI for each the risk factors of CVD were denoted by high in sensitivity value and each value of sensitivity and specificity were more than 50%. For each of the risk factors of CVD, the sensitivity (63% - 80%) associated with each optimal BMI cut-off value was greater than its attendant specificity (51% - 56%). In the study by Yong-Hao et al. (87), the optimal cut-off points for each index was denoted by the value that yielded the largest sum of sensitivity and specificity. Ostensibly, applying this decision rule in this study may potentially lower the cut-off points of the BMI indices by enhancing sensitivity at the expense of reduced specificity. Notwithstanding these methodological differences, it must be acknowledged that all anthropometric cut-off points are arbitrary, and any decision rules to determine cut-off values must balance the need to prevent a significant proportion of CVD events, and the clinical practice burden of labeling a patient as being at risk for CVD (87).

These findings (Table 15) showed that the optimal cut-off points of BMI which reflect the risk factors of CVD (hypertension and dyslipidemia) were 22.0, 23.0, 23.5 kg/m^2 (only HDL-C had the cut-off point of BMI at 24.0 kg/m^2). These optimal cut-off points of BMI were closely matched with the Asia criteria. The results of this study indicate that the optimal cut-off values of BMI were lower than those recommended by the WHO. Therefore, the current WHO criteria to classify overweight and obesity may not be appropriate for Thai male populations who aged 35-50 years.

CHAPTER VI

CONCLUSION AND RECOMMENDATIONS

Conclusion

The aim of the study was to examine the optimal cut-off points of body mass index which reflect the risk factors of cardiovascular disease in the urban Thai male population aged 35-50 years and who live in Bangkok.

The design of this study was a cross sectional research. The 413 respondents worked in Bangkok. The samples worked in the government offices and undertook annually physical check up by the office of Public Health and Environmental Technology Service, Faculty of Public Health, Mahidol University. The samples were purposive sampling. Respondents who were taking medication for controlling of blood pressure and plasma lipid, were heavy smokers who smoked 16 rolls or more per day, were heavy daily consumers of alcohol, and who had any complication condition or disease that induces increases or decreases weight change such as renal disease, liver disease, thyroid disease or post orthopedic operation with plate and screw were excluded of this study.

The research instruments were two part self-administered questionnaires: demographic data and health data, weight and height scale, mercury manometer for measurement of blood pressure and laboratory instruments for collecting blood samples and blood chemical analysis. The body mass index of each participant was calculated. The collected data were analyzed and presented in absolute number, percent, mean, standard deviation, minimum, and maximum value. The relationship among BMI, SBP, DBP, TC, TG, LDL-C, and HDL-C were analyzed by multiple regression analysis. The optimal cut-off points of BMI that associated with high in SBP, DBP, TC, TG, LDL-C, and low in HDL-C were analyzed by epidemiological methods (sensitivity, specificity and ROC curve analysis). From the results of the

study it can be concluded that, most of the respondents were married males in the middle age group (43.05±5.01 years). The majority of them completed Bachelor degrees and were consumer of cigarettes and alcohol. Moreover, the respondents were government officials who working in an office and who were physically inactive (had sedentary lifestyles) and most of them did not exercise. The body mass index was classified by two criteria. The first group classified according to WHO criteria found that the most of the respondents were of normal weight (BMI 18.5-24.9 kg/m^2). The second group classified according to Asia criteria found that the most of the respondents were overweight (BMI ≥23 kg/m^2). BMI levels positively increased with the risk factors of cardiovascular disease such as SBP, DBP, TC, LDL-C, and negatively increased with HDL-C. However, BMI levels did not positively increased with TG. The cut-off points of BMI were classified into 9 levels as 21.0, 21.5, 22.0, 22.5, 23.0, 23.5, 24.0, 24.5, and 25.0 kg/m^2. Epidemiological methods (sensitivity, specificity and ROC curve analysis) have been used to examine the optimal cut-off points of BMI which reflect the risk factors of CVD. The optimal cut-off points of BMI which reflect hypertension was 23.5 kg/m^2, elevated TC was 22.0 kg/m^2, elevated TG was 23.5 kg/m^2, elevated LDL-C was 23.0 kg/m^2, and reduced HDL-C was 24.0 kg/m^2.

Recommendations

1. The samples of this study were not population based and were limited to Thai male who aged 35 – 50 years and lived in Bangkok. Hence, this results need to be interpreted with caution.

2. This study demonstrated that the optimal cut-off points of BMI which reflect the risk factors of CVD were lower than the criteria suggested by the WHO, but were in agreement with Asia criteria. Therefore, using the optimal cut-off point of BMI at 23.0 kg/m^2 according to the Asia criteria represented the optimal cut-off points of BMI at 22.0, 23.5 and 24.0 kg/m^2, would be convenience to detect the risk factors of CVD for Thai male populations who are aged 35 -50 years.

3. This the multiple regression analysis indicated that age, smoking and alcohol drinking of the samples slightly effects on the risk factors of CVD. Therefore, using

these results would be aware of the age, smoking habits and alcohol drinking behavior in other Thai male population.

Recommendations for further study

1. Expanded study in other Thai male populations in different work place characteristics or the general population or Thai female populations. Furthermore, the optimal cut-off points of BMI as the most appropriate screening tool for Thai populations would be further strengthened if there were supporting data from the larger sample sizes than that undertaken in this study.

2. Eating habits questionnaires should include information on health data related to the risk factors for CVD both in terms of quality and quantity of food intake.

3. Smoking habits questionnaires should include age at beginning smoking and the duration of smoking. Moreover, the further studies should limit the number of cigarettes smoked that may be affect the levels of blood pressure and plasma lipid.

4. Alcohol drinking behavior questionnaires should include the volume of alcohol consumed at each intake event.

REFERENCES

1. World Health Organization Report of a WHO Consultation on obesity. Obesity: preventing and managing the global epidemic. Geneva: world Health Organization, 1998.

2. Seidell JC. Obesity: a growing problem. Acta Paediater 1999;88:46-50.

3. กรมอนามัย กระทรวงสาธารณะสุข. รายงานการสำรวจภาวะอาหารและโภชนาการของประเทศไทย. Fact sheet 2541;3(5):1-5.

4. Temcharoen P. A Longitudinal causal relationship among cardiovascular risk factors in the employee of the government saving bank. A thesis submitted in partial fulfillment of the requirements for the degree of Doctor of Public Health. Faculty of Graduate Studies Mahidol University, 2001.

5. ส่วนข้อมูลข่าวสาธารณสุข, สำนักนโยบายและแผนสาธารณสุข, กระทรวงสาธารณสุข. สถิติสาธารณสุข พ.ศ. 2545.

6. Willett WC. Manson JE, Stampfer MJ. Weight, weight change and coronary heart disease in women. JAMA 1995;27:1461–5.

7. Jung RT. Obesity as a disease. British Medecal Bulletin 1997;53:307-321.

8. Reisen E, Abel R, Modan M, Sliverberg DS, Eliahou HE, Modan B. The effect of weight loss without salt restriction on the reduction in blood pressure inoverweight hypertensive patients. N Engl J Med 1998;298:1-6.

9. Dattilio AM, Kris-Ethertum PM. Effects of weight reduction on blood lipids and lipoproteins: a meta analysis. Am J Clim Nutr 1992;56:302-8.

10. Han TS, Leer EM, Seidell JC, Lean MEJ. Waist circumference action level in the identification of cardiovascular risk factors: prevalence study in a random sample. BMJ 1995;311:1401-5.

11. Lean MEI, Han TS, Morrison CE. Waist circumference as a measure for indicating need for weight management, BMJ 1995;311:158-61.

12. Leenen R, Van der Kooy K, Seidel JC, Deurenberg P. Visceral fat accumulation measured by magnetic resonance imaging in relation to serum lipid in obese men and women. Atherosclerosis 1992;94:171-81.

13. Lemieux S, Prud'homme D, Bouchard C, Tremblay A, Despres J-P. A single threshold value of waist circumference identifies normal-weight and overweight subjects with excess visceral adipose tissue. Am J Clin Ntur 1996; 64:685-93.

14. Roche AF, Guo S. Development, testing and use of predictive equation for body composition measure. In: Kral JG, Vanitallie TB, editors. Recent developments in body composition analysis: Methods and applications. London: Smith-Gordon and Company Limited Nishimura Company Limited, 1993:1-16.

15. Garn SM, Leonard WR, Hawthorne VM. Three limitations of the body mass index. Am J Clin Nutr 1986;44:996-7.

16. Deurenberg P, Deurenberg-Yap M, Guricci S. Asians are different from caucasians and from each other in their body mass index/body fat percent relationship. Obesity Reviews 2002;3:141-6.

17. Deurenberg P, Deurenberg-YapM, Staveren WAV. Body mass index and percent body fat: a meta-analysis among different ethnic groups. Int J Obesity 1998; 22:1164-71.

18. Gurric S, Hartriyanti Y, Hautvast JGAJ, Deurenberg P. Relationship between body fat and body mass index: Difference between Indonesians and Dutch Caucasians. Eur J Clin Nutr 1998;52:779-83.

19. Deurenberg P, Deurenberg-Yap M, Wang J, Lin FP, Schmidt G. The impact of body build on the relationship between body mass index and percent body fat. Int J Obesity 1999;23:537-42.

20. Gallagher D, Heymsfield SB, Heo M, Jebb SA, Murgatroyd PR, Samoto Y. Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. Am J Clin Nutr 2002;72:694-701.

21. Moon OR, Kim NS, Jang SM, Yoon TH, Kim SO. The relationship between body mass index and the prevalence of obesity-related diseases based on the

1995 National Health Interview Survey in Korea. Obesity Reviews 2002;3:191-6.

22. Deurenberg-Yap M, Chew SK, Deurenberg P. Elevated body fat percentage and cardiovascular risks at low body mass index levels among Singaporean Chinese, Malays and Indians. Obesity Reviews 2002;3:209-15.
23. Jia WP, Xiang KS, Chen L, Lu JX, Wu YM. Epidemiological study on obesity and its comorbidities in urban Chinese older than 20 years of age in Shanghai, China. Obesity Reviews 2002;3:157-65.
24. Lee ZSK. Critchley JAJH, Ko GTC. Obesity and cardiovascular risk factors in Hong Kong Chinese. Obesity Reviews 2002;3:173-82.
25. Li G, Chen X, Jang Y. Obesity coronary heart disease risk factors and diabetes in Chinese: an approach to the criteria of obesity in the Chinese population. Obesity Reviews 2002;3:167-72.
26. WHO/IASO/IOTF. The Asia-Pacific perspective: Redefining obesity and its treatment. Health Communications Australia Pty Limited; 2000.
27. Stevans NHC. The correlation and prediction of body composition from dual energy X-ray absorbtiometry and anthropometric measurements in Thai women aged 30 to 39 years. (M.S. Thesis in Public Health (Nutrition)) Bangkok; Faculty of Graduate Studies, Mahidol University; 1997.
28. Chunchusak S. Comparative study between the relationship of waist circumference and waist to hip ratio and serum lipid in men. A thesis submitted in partial fulfillment of the requirement for the degree of Master of Science in Public Health Nutrition. Faculty of Graduate Studies Mahidol University, 1997.
29. Guidelines Subcommittee.1999 World Health Organization-International Society of Hypertension Guidelines for the Management of Hypertension. J Hypertension 1999;17:151-83.
30. Expert Panel on Detection, Education and Treatment of High Blood Cholesterol in Adults, National Cholesterol Education Program. Second report of the Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adult (Adult Treatment Panel II). Circulation 1994;89:1329-445.

31. William SK. Essentials of nutrition and diet therapy. 7th ed. St. Louis: Mosby; 1999.394-423.

32. McKenzie JF, Pinger RR, Kotecki JE. An introduction to community health. Boston: Jones and Bartlett; 1999.95-122.

33. World Health Organization. The World Health Report 2002 " Reducing risk and promoting health life". Geneva, World Health Organization, 2002.

34. World Health Organization. Integrated management of cardiovascular risk: report of a WHO meeting, Geneva, 2002.

35. Poirierp, Eckel RH. The heart and obesity. In: Fuster V, Alexander RW, King S, editors. Hurst's the heart. New York: McGraw-Hill; 2000. p.2289-303.

36. Kannel WB, Dawber TR, Hagen A. Factors of risk in the development of coronary heart disease–six years follow–up experience: the Framingham Study. Ann Inter Med 1961;55:33-50.

37. Beirman EL, Chait A. Nutrition and diet in relation to hyperlipidemia and atherosclerosis. In: Shils ME, Young VR, editors. Modern nutrition in heath and disease. 7th ed. Philadelphia: Lea & Febiger;1988. p.1283-97.

38. Pearson TA, Fuster V. matching the intensity of risk factor management with the hazard of coronary disease events: executive summary. J Am Coll Cardiol 1996;27:961-3.

39. Grundy SM. Primary prevention of coronary heart disease: integrating risk assessment with intervention. Circulation 1999;100:988-98.

40. Wilson PWF, D'Agostino RB, Levy D. Prediction of coronary heart disease using risk factor categories. Circulation 1998;97:1837-47.

41. Grundy SM, Pasternak R, Greenlan P. AHA/ACC scientific statement: assessment of cardiovascular risk by use of multiple-risk-factor assessment equation: a statement for healthcare professionals from the American Heart Association and the American Collage of Cardiology. Circulation 1999;100:1481-92.

42. Daviglus MI, Stamler J. Major coronary risk factors and coronary heart disease: much has been achieved but crucial challenges remain. J Am Coll Cardiol 2001;38:1018-22.

43. Yusuf S, Reddy S, Ounpuu S. Global burden of cardiovascular disease. Part I: general considerations, the epidemiologic transition, risk factors, and impact of urbanization. Circulation 2001;104:2746-53.

44. Pearson TA, Blair SN, Daniels SR. AHA guidelines for primary prevention of cardiovascular disease and stroke:2002 update. Circulation 2002;106:388-91.

45. Kaplan NM, Systemic hypertension: mechanism and diagnosis. In: Braunwald E, Zipes Dp, Libby P, editors. Heart disease: a textbook of cardiovascular medicine, 6th ed. Philadelphia: Saunders;2001. p.941-71.

46. Black HR, Bakris GL, Elliott WJ. Hypertension: epidemiology, pathophysiology, diagnosis, and treatment. In: Fuster V, Alexander RW, O' Rourke RA, editors. Hurst' the heart. 10th ed. New York: McGraw-Hill; 2001. p.1533-604.

47. Law MR, Wald NJ, Thompson SG. Serum cholesterol reduction and health: by how much and how quickly is the risk of ischemic heart disease lowered. BMJ 1994;308:367-72.

48. Stamler J, Daviglus ML, Garside DB. Relationship of baseline serum cholesterol level in 3 large cohorts of younger men to long-term coronary, cardiovascular, and all-cause mortality and to longevity. JAMA 2000;284:311-8.

49. Assmann G, Schulte H, Eckardstein A, Huang Y. High- density lipoprotein cholesterol as a predictor of coronary heart disease risk. The PROCAM experience and pathophysiological implications for reverse cholesterol transport. Atherosclerosis 1996;124(2 Suppl.):S11-S20.

50. GottoAM Jr. Low high-density lipoprotein cholesterol as a risk factor in coronary heart disease: a working group report. Circulation 2001;103:2213-8.

51. Faergeman O. Hypertriglyceridemia and the fibrate trials. Curr Opin Lipidol 2000;11:609-14.

52. Cullen P. Evidence that triglycerides are an independent coronary heart disease risk factor. Am J Cordiol 2000;86:943-9.

53. Farkouh ME, Rayfield EJ, Fuster V. Diabetes and cardiovascular disease. In: Fuster V, Alexander RW, O' Rourke RA, editors. Hurst's the heart, 10th ed. New York: McGraw-Hill, 2001. p.2197-218.

54. Turner RC, Millns H, Neil HA. Risk factor for coronary artery disease in noninsulin dependent diabetes mellitus (UKPD 23). BMJ 1998;316:823-8.

55. The Expert Committee on the Diagnosis and classification of Diabetes Mellitus. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Diabetic Care 1997;20:1183-97.

56. Peto R, Chen ZM, Boreham J. Tobacco-the growing epidemic. Nat Med 1999;5:15-7.

57. Peto R, Chen ZM, Boreham J. Mortality from tobacco in developed countries: indirect estimation from national vital statistic. Lancet 1992;339:1268-78.

58. Jackson G. Smoking cessation: a consensus statement with special reference to primary care. Int J Clin Pract 2001;55:358-92.

59. Betteridge DJ, Morrell JM. Clinicians Guide to lipids and coronary heart disease. Great Britain 1998.117-237.

60. Rigotti NS, Pasternak RC. Cigarette smoking and coronary heart disease: risk and management. Cordiol Clin 1996;14:51-68.

61. Fletcher GF, Balady G, Blaip SN. Statement on exercise. Benefits and recommendation for physical activity programs for all American. A statement for health professionals by the committee an Exercise and Cardiac Rehabilitation of the Council on Clinical Cardiology. American Heart Association. Circulation 1996;94:857-62.

62. Consensus Development Panel on Physical Activity and Cardiovascular Heart Physical activity and cardiovascular health. JAMA 1996;276:241-6.

63. Berg A, Halle M, Franz J, Keul J. Physical activity and lipoprotein metabolism epidemiological evidence and clinical trials. Eur J Med Res 1997;2:259-64.

64. Flecher GF, Balady G, Blair SN. Statement on exercise. Benefits and recommendation for physical activity programs for all Americans. A statement for health professionals by the committee on exercise and

cardiac rehabilitation of the council on clinical cardiology, American Heart Association. Circulation 1996;94:857-62.

65. National Institutes of Health. Clinical Guideline on the Identification, Evaluation, and Treatment of Overweight and Obesity in adults-The Evidence Report. Obes Res 1998; 6(2 Suppl):51S-210S.

66. Flegal KM, Carroll MD, Ogden CL. Prevalence and trends in obesity among US adults,1999-2000. JAMA 2002;288:1723-27.

67. Kuczmarshi RJ, Flegal KM. Criteria for definitions obesity for the United States. Am J Clin Nutr 2000;72;1074-81.

68. Devid H and Susan B. Body-composition analysis. In: Daniel HB, Robert K, editors. Evaluation and Management of obesity. 9th ed. Hanley and Belfus INC; 2002. p 23-8.

69. Seidell JC and Flegal KM. Assessing obesity: classification and epidemiology. Br Med Bull 1997;53:238-52.

70. US Department of Health and Human Services. Nutrition and overweight. In Health People 2010 (Conference edition). Washington DC: US Government Printing Office, 2000.

71. US Department of Agriculture and US Department of Health and Human Services. Nutrition and your health: dietary guidelines for Americans. 5th ed. Washington DC: US Government Printing Office, 2000.

72. Ko GTC, Chan JCN. Cockram CS, Woo J. Prediction of hypertension. Diabetes, dyslipidemia on albuminuria using simple anthropometric indexes in Hong Kong Chinese. Int J Obes 1999;23:1136-42.

73. Deurnberg-Yap M, Yian TB, Kaics, Derenberg P, Van Staveren WA. Manifestation of cardiovascular risk factors at low levels of body mass index and waist-to-hip ratio in Singaporean Chinese. Asia Pacific J Clin Nutr 1999;8:177-83.

74. Swinburn B, Ley SJ, Plank LD. Body size and composition in Polynesians. Int J Obes 1999;23;1178-83.

75. Ueshima. Mortality of cardiovascular disease in respect of BMI. Research report of the grant of Ministry of Health and Welfare. Japan, 1998.

76. Department of Health. Health Survey of England. London, The Stationery Office,2002.

77. Jebb SA. Aetiology of obesity. Br Med Bull 1997,53:264-85.

78. Dipietro L. Physical activity, body weight and adiposity: epidemiologic perspective. Exerc Sports Sci Rev 1995,23:275-303.

79. Coakley EH, Rimm EB, Colditz G. Predictors of weight change in men: Results from the Health Professionals Follow-Up Staly. Int J Obes 1998,22:89-96.

80. Whichelow MJ, Prevost AT. Dietary patterns and their association with demographic, lifestyle and health variables in a random sample of British adult. Br J Nutr 1996,76:17-30.

81. Prentice AM, Jebb SA. Fast food, energy density and obesity a possible mechanistic link. Obes Rev 2003,4:187-94.

82. Rolls BJ, Morrise EL, Roe LS. Portion size of food effects energy intake in normal weight and overweight men and women. Am J Clin Nutr 2002,76:1207-13.

83. Caggiula AW, Mustad VA. Effect of dietary fat on coronary artery disease risk and total and lipoprotein cholesterol concentration: epidemiological studies. Am J Clin Nutr 1997; 65 (2 Suppl): 1597S-1610S.

84. Pitt B, Mancini GBJ, Ellis GS. Pravastatin limitation of atherosclerosis in the coronary arteries (PLACI): reduction in atherosclerosis progression and clinical events. J Am Coll Cardiol 1995; 26:113-9.

85. Pasternak R, Gruandy SM, Leve D, Thomso P. Task Force 3: spectrum of risk factors for coronary heart disease. J Am Coll Cordiol 1996;27:978-90.

86. Grundy SM. Second Report of the Expert Panel on Detection, Evaluation and Treatment of High Blood cholesterol in adult (Adult Treatment Panel III), National Institutes of Health, NHLBI 1993. p. 93-5.

87. Yong-Hao PUA, Peck-Hoon ONG. Anthropometic indices as screening tools for cardiovascular risk factors in Singaporean women. Asia Pac J Clin Nutr 2005;14:74-9.

88. Timmreck TC. An introduction to Epidemilogy. Jones and Bartlett Publishers International, London. 1994.

89. Xiao-Hua Z, Nancy AO, Donna KM. Statistical Methods in Diagnostic Medicine. John Wiley&Sons. Inc. New York. 2002.

APPENDIX

APPENDIX A

QUESTIONNAIRE

แบบสอบถาม

เรื่อง จุดตัดของดัชนีมวลกายที่สะท้อนปัจจัยเสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือดในกลุ่มประชากรชายไทยที่อาศัยอยู่ในเขตเมือง

คำชี้แจง

1. แบบสอบถามชุดนี้จัดทำขึ้น มีวัตถุประสงค์เพื่อศึกษาถึงดัชนีมวลกายที่เหมาะสมสำหรับคนไทยในการบ่งชี้ภาวะโรคอ้วนซึ่งเป็นปัจจัยเสี่ยงสำคัญต่อการเกิดโรคหัวใจและหลอดเลือด เพื่อนำไปใช้ในการจัดทำวิทยานิพนธ์ประกอบการศึกษาปริญญาสาธารณสุขศาสตร์มหาบัณฑิต (โภชนวิทยา) คณะสาธารณสุขศาสตร์ มหาวิทยาลัยมหิดล
2. แบบสอบถามมีทั้งหมด 3 ส่วน คือ

ส่วนที่ 1 ข้อมูลทั่วไปของผู้ตอบแบบสอบถาม

ส่วนที่ 2 ข้อมูลเกี่ยวกับสุขภาพของผู้ตอบแบบสอบถาม

ส่วนที่ 3 การตรวจร่างกายโดยการชั่งน้ำหนัก วัดส่วนสูง วัดความดันโลหิต การตรวจเลือดเพื่อวัดระดับไขมันในเลือด

3. การตรวจเลือดเพื่อวัดระดับไขมันในเลือด เป็นการตรวจที่จัดขึ้นโดยสำนักงานบริการเทคโนโลยีสาธารณสุขและสิ่งแวดล้อม คณะสาธารณสุขศาสตร์ มหาวิทยาลัยมหิดล ในการตรวจสุขภาพเจ้าหน้าที่.....ประจำปี 2547 โดยผู้วิจัยขอความกรุณาจากท่านผู้ตอบแบบสอบถามโดยการขอผลการตรวจระดับไขมันในเลือด เพื่อนำมาใช้ในการศึกษาวิจัยในครั้งนี้
4. ข้อมูลจากแบบสอบถามทั้งหมด ผู้วิจัยจะเก็บเป็นความลับและจะไม่ปรากฏชื่อของผู้ตอบแบบสอบถามในวิทยานิพนธ์
5. ผู้ตอบแบบสอบถามสามารถงดเข้าร่วมการวิจัยนี้ได้ทุกเวลา และสามารถที่จะสงวนสิทธิ์ที่จะไม่ตอบคำถามได้โดยไม่มีผลกระทบใดๆต่อผู้ตอบแบบสอบถาม
6. ประโยชน์ที่คาดว่าจะได้รับจากการศึกษาในครั้งนี้ คือ สามารถหาจุดตัดของดัชนีมวลกายที่สามารถสะท้อนถึงปัจจัยเสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือดที่เหมาะสมต่อประชากรชายไทย

โดยที่ประชากรชายไทยทั่วไปสามารถวัดและประมวลผลได้ด้วยตนเอง เพื่อเฝ้าระวังถึงภาวะสุขภาพของตนเอง และนอกจากนี้สามารถนำมาใช้ทางการแพทย์ในการคัดกรองผู้ป่วยโรคอ้วนที่เสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือด

ขอขอบคุณในความร่วมมือของท่านเป็นอย่างสูง

ขอแสดงความนับถือ
นางสาว ประภาพร แก้วบุญเรือง
ผู้วิจัย

หนังสือแสดงเจตนายินยอมเข้าร่วมโครงการวิจัย

วันที่.....เดือน.....พ.ศ.....

ข้าพเจ้า.....อายุ.....ปี สถานที่ทำงาน.....ขอแสดงเจตนายินยอมเข้าร่วมโครงการวิจัยเรื่อง จุดตัดของดัชนีมวลกายที่สะท้อนปัจจัยเสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือดในกลุ่มประชากรชายไทยที่อาศัยอยู่ในเขตเมือง โดย นางสาวประภาพร แก้วบุญเรือง โดยข้าพเจ้าได้รับทราบเกี่ยวกับรายละเอียดของงานวิจัยตามคำชี้แจง ดังต่อไปนี้ วัตถุประสงค์ของการวิจัย ประโยชน์ที่คาดว่าจะได้รับจากการวิจัย รายละเอียดและขั้นตอนที่ผู้ร่วมโครงการวิจัยจะต้องปฏิบัติ การติดต่อกับผู้วิจัยในกรณีที่มีคำถาม

ข้าพเจ้าทราบแล้วว่าข้าพเจ้ามีสิทธิที่จะงดการเข้าร่วมการวิจัยนี้ได้ทุกเวลา และสามารถที่จะสงวนสิทธิ์ที่จะไม่ตอบคำถามได้โดยไม่มีผลกระทบใดๆต่อตัวข้าพเจ้า

ข้าพเจ้าได้ทำการซักถามเกี่ยวกับการวิจัย และผู้วิจัยได้ตอบให้ข้าพเจ้าเข้าใจจนหมดข้อสงสัยแล้ว ข้าพเจ้าทราบว่าข้าพเจ้าสามารถถามคำถามอื่นที่จะมีเกี่ยวกับการวิจัยได้ตลอดระยะเวลาของการวิจัยนี้

ข้าพเจ้าทราบจากผู้วิจัยว่าจะไม่มีชื่อของข้าพเจ้าปรากฏบนข้อมูลการวิจัยที่ข้าพเจ้าตอบ และจะไม่เปิดเผยข้อมูลรายละเอียดส่วนบุคคล เป็นรายบุคคลในผลการวิจัย รวมทั้งจะไม่เปิดเผยข้อมูลที่เกี่ยวกับข้าพเจ้าแก่ผู้อื่นที่ไม่เกี่ยวข้องกับการวิจัยได้รับทราบ

ข้าพเจ้าได้รับทราบข้อมูลของโครงการข้างต้นแล้ว ข้าพเจ้ายินยอมที่จะเข้าร่วมในโครงการวิจัยครั้งนี้ จึงได้ลงลายมือชื่อไว้เป็นหลักฐาน

ลงชื่อ.....ผู้ให้ความยินยอม

(.....)

วันที่.....

ลงชื่อ.....พยาน

(.....)

ลงชื่อ.....พยาน

(.....)

แบบบันทึก

วันที่.....

ลำดับที่.....

จุดตัดของดัชนีมวลกายที่สะท้อนปัจจัยเสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือดในกลุ่มประชากรชายไทยที่อาศัยอยู่ในเขตเมือง

<u>ส่วนที่ 1</u> ข้อมูลทั่วไป

ชื่อ(นาย).....นามสกุล.....

สถานที่ทำงาน .....

วันเดือนปีที่เกิด วันที่..... เดือน.....พ.ศ.....อายุ.....ปี

เชื้อชาติ ☐ ไทย ☐ อื่นๆ (ระบุ).....

ศาสนา ☐ พุทธ ☐ คริสต์ ☐ อิสลาม ☐อื่นๆ (ระบุ).....

สถานภาพสมรส ☐ โสด ☐ คู่ ☐ หม้าย ☐ แยกกันอยู่

การศึกษา ☐ มัธยมศึกษา ☐อนุปริญญา ☐ปริญญาตรี ☐ ปริญญาโทหรือสูงกว่า

<u>ส่วนที่ 2</u> ข้อมูลเกี่ยวกับสุขภาพ

1. ขณะนี้ท่านป่วยหรือได้รับการวินิจฉัยว่าเป็นโรคต่อไปนี้หรือไม่

<u>โรคอ้วน</u> ☐ ไม่ใช่ ☐ ใช่ ระยะเวลาที่ป่วย.....ปี.....เดือน

การรักษา ☐ ไม่เคยรักษา

☐ เคยรักษาแต่ขณะนี้ไม่ได้รักษาแล้ว เป็นระยะเวลา.....ปี.....เดือน

☐ กำลังทำการรักษาอยู่ (ตอบได้มากกว่า 1 ข้อ)

☐ควบคุมอาหาร

☐ออกกำลังกาย

☐รับประทานยา(ระบุ).....

☐อื่นๆ(ระบุ).....

<u>โรคความดันโลหิตสูง</u> ☐ ไม่ใช่ ☐ ใช่ ระยะเวลาที่ป่วย.....ปี.....เดือน

การรักษา ☐ ไม่เคยรักษา

☐ เคยรักษาแต่ขณะนี้ไม่ได้รักษาแล้วเป็นระยะเวลา.....ปี.....เดือน

☐ กำลังทำการรักษาอยู่ (ตอบได้มากกว่า 1 ข้อ)

☐ควบคุมอาหาร

☐ออกกำลังกาย

☐รับประทานยา(ระบุ).....

☐อื่นๆ(ระบุ).....

โรคเบาหวาน ☐ ไม่ใช่ ☐ ใช่ ระยะเวลาที่ป่วย……ปี……เดือน

การรักษา ☐ ไม่เคยรักษา

☐ เคยรักษาแต่ขณะนี้ไม่ได้รักษาแล้วเป็นระยะเวลา……..ปี……..เดือน

☐ กำลังทำการรักษาอยู่ (ตอบได้มากกว่า 1 ข้อ)

☐ควบคุมอาหาร

☐ออกกำลังกาย

☐รับประทานยา(ระบุ)…….

☐อื่นๆ(ระบุ)……

โรคหัวใจและหลอดเลือด ☐ ไม่ใช่ ☐ ใช่ ระยะเวลาที่ป่วย……ปี……เดือน

การรักษา ☐ ไม่เคยรักษา

☐ เคยรักษาแต่ขณะนี้ไม่ได้รักษาแล้วเป็นระยะเวลา……..ปี……..เดือน

☐ กำลังทำการรักษาอยู่ (ตอบได้มากกว่า 1 ข้อ)

☐ควบคุมอาหาร

☐ออกกำลังกาย

☐รับประทานยา(ระบุ)…….

☐อื่นๆ(ระบุ)……

โรคเก๊าท์ ☐ ไม่ใช่ ☐ ใช่ ระยะเวลาที่ป่วย……ปี……เดือน

การรักษา ☐ ไม่เคยรักษา

☐ เคยรักษาแต่ขณะนี้ไม่ได้รักษาแล้วเป็นระยะเวลา……..ปี……..เดือน

☐ กำลังทำการรักษาอยู่ (ตอบได้มากกว่า 1 ข้อ)

☐ควบคุมอาหาร

☐รับประทานยา(ระบุ)…….

☐อื่นๆ(ระบุ)……

โรคตับ ☐ ไม่ใช่ ☐ ใช่ ระยะเวลาที่ป่วย……ปี……เดือน

การรักษา ☐ ไม่เคยรักษา

☐ เคยรักษาแต่ขณะนี้ไม่ได้รักษาแล้ว เป็นระยะเวลา……..ปี……..เดือน

☐ กำลังทำการรักษาอยู่ (ตอบได้มากกว่า 1 ข้อ)

☐ควบคุมอาหาร

☐รับประทานยา(ระบุ)……

☐อื่นๆ(ระบุ)……..

<u>โรคไต</u> ☐ ไม่ใช่ ☐ ใช่ ระยะเวลาที่ป่วย……ปี……เดือน

การรักษา ☐ ไม่เคยรักษา

☐ เคยรักษาแต่ขณะนี้ไม่ได้รักษาแล้ว เป็นระยะเวลา……..ปี……..เดือน

☐ กำลังทำการรักษาอยู่ (ตอบได้มากกว่า 1 ข้อ)

☐ควบคุมอาหาร

☐รับประทานยา(ระบุ)……

☐อื่นๆ(ระบุ)……

<u>โรคของต่อมไทรอยด์</u> ☐ ไม่ใช่ ☐ ใช่ ชนิด…….. ระยะเวลาที่ป่วย……ปี……เดือน

การรักษา ☐ ไม่เคยรักษา

☐ เคยรักษาแต่ขณะนี้ไม่ได้รักษาแล้ว เป็นระยะเวลา……..ปี……..เดือน

☐ กำลังทำการรักษาอยู่ (ตอบได้มากกว่า 1 ข้อ)

☐รับประทานยา(ระบุ)……..

☐อื่นๆ(ระบุ)…….

2. ท่านเคยผ่าตัดเกี่ยวกับกระดูกโดยการใส่เหล็กดามกระดูกไว้หรือไม่

☐ ไม่เคย ☐เคย ขณะนี้ท่าน……☐เอาเหล็กออกแล้ว ☐ยังคงมีเหล็กอยู่ในร่างกาย

3. การสูบบุหรี่ ท่านสูบบุหรี่หรือไม่

☐ไม่สูบ

☐สูบ สูบวันละประมาณ……มวน เริ่มสูบบุหรี่เมื่ออายุ……ปี

☐เคยสูบแต่ปัจจุบันเลิกสูบแล้ว ระยะเวลาที่เลิกสูบ……ปี……เดือน

4. การดื่มอัลกอฮอล์ ปัจจุบันท่านดื่มสุรา,เบียร์,ไวน์ ,ยาดองเหล้าหรือไม่

☐ไม่ดื่ม ☐ ดื่ม… ☐ทุกวัน

☐…….. ครั้ง/สัปดาห์

☐1 ครั้ง/เดือนหรือนานกว่า

ท่านดื่มเครื่องดื่มประเภทอัลกอฮอล์ภายใน 24 ชั่วโมงก่อนที่จะมารับการตรวจร่างกายในวันนี้ หรือไม่

☐ ไม่ดื่ม ☐ ดื่ม

5. ประวัติการออกกำลังกาย ปัจจุบันท่านออกกำลังกายหรือไม่

☐ ไม่ออกกำลังกาย

☐ออกกำลังกาย ชนิดของกีฬา (ตอบได้มากกว่า 1 อย่าง)……

☐ < 3 ครั้ง/สัปดาห์และครั้งละ < 30 นาที

☐ ≥ 3 ครั้ง/สัปดาห์และครั้งละ ≥ 30 นาที

วันที่…….
ลำดับที่……..

<u>ส่วนที่ 3</u> แบบบันทึกน้ำหนัก ส่วนสูง ความดันโลหิตและผลการตรวจเลือด

น้ำหนัก …….. ก.ก.

ส่วนสูง ……... ซ.ม.

BMI ……. ก.ก./ $ม^2$

ความดันโลหิต ……. มม.ปรอท.

ผลการตรวจเลือด

TC ……มก. / ดล.

TG…….มก./ ดล.

LDL-C …….มก./ ดล.

HDL-C …….มก./ ดล.

APPENDIX B

EPIDEMIOLOGICAL METHODS

Sensitivity and specificity: Tests of validity

Sensitivity is the ability of a test to correctly identify those who have the disease; the percentage of those who have the disease and are proven to have the disease as demonstrated by a test. Sensitivity shows the proportion of truly diseased persons in a population who underwent screening and who are correctly identified as being diseased by the screening test (88).

Specificity is the ability of a test to correctly identify the percentage of those who do not have the disease; those who do not have the disease and are proven to not have the disease as demonstrated by a test. Specificity shows the proportion of non-diseased persons in a population who underwent screening and who are correctly identified as not being diseased by the screening test (88).

Table 18. A 2 × 2 probability table

Screening Test	Disease		Total
	+	-	
+	a (True positive)	b (False positive)	a + b (Total test positive)
-	c (False negative)	d (True negative)	c + d (Total test negative)
Total	a + c (Total disease)	b+ d (Total non-disease)	a + b + c + d (Grand total)

True positive is when the test says the person has a disease and the person indeed has the disease.

False positive is when a screening test indicates that the individual has a disease but the person does not actually have the disease. The test was wrong in indicating that the person had the disease when the person was in fact well and not diseased. The test wrongly said yes to being diseased, labeling a healthy person as diseased.

False negative is when a screening test indicates that the individual not have a disease but the person in fact does have a disease. The test was wrong in indicating that the person is well when the person will get sick or is diseased. The test wrongly said no to being diseased, labeling a diseased person as health.

True negative is when the test says the person is healthy and has no disease and the person in fact is healthy and disease free.

$$\text{Sensitivity} = \Pr(\text{Test+} \mid \text{Dx+}) = \frac{a}{a+c}$$

$$\text{Specificity} = \Pr(\text{Test-} \mid \text{Dx-}) = \frac{d}{b+d}$$

$$\text{Accuracy} = \frac{a+d}{a+b+c+d}$$

Standardization of tests is the term used to show that a test has been used over a long period of time, has had widespread use, the cut-off levels/ values have been well established, and the test has a proven track record over time with normative data. Screening programs should use standardized tests as it is important to have test with high predictability, reliability, validity, and long term use. This usually means that the test has been refined and retested in order to make it as effective and accurate as possible.

Understanding the results of screening, sensitivity, and specificity

From each screening program, individuals are classified as either negative (those without the disease) or positive (those with the disease). However, because the sensitivity and specificity of a test is often less than 100%, false negatives and false

positive occur. Thus, the epidemiologist classifies the individual participants into four categories.

False Negative (FN) **True Negatives (TN)**

False Positive (FP) **True Positive (TP)**

These four categories are used to understand and evaluate the results of screening programs. These categories are also used to assess test results and for data analysis of a study population. To more easily understand the role of the four categories, it is helpful to use a graphic presentation of the screening process and where each of the four possible outcomes of a test fits.

Sensitivity and specificity are proportionate and therefore expressed in percentages. Ideally, the epidemiologist would like to see a test that works so well that both sensitivity and specificity are at 100%.

To help in the analysis, some observations about sensitivity and specificity will be helpful. As the percentage of true negative (TN) and true positives (TP) goes up, sensitivity and specificity go down. In summary, sensitivity is the ability to identify correctly those with the disease. Specificity is the ability is the ability to identify correctly those without the disease.

An inverse relationship exists between true positives and false positives. Conversely an inverse relationship exists between false negatives and true negatives.

The percentage of false negatives is the complement of sensitivity. Conversely, the percentage of false positives is the complement of specificity. The epidemiologist wants a sensitive test so that the test will identify a high proportion of those who have the disease and a test that will generate few false negatives. Conversely the epidemiologist wants the test to be specific enough to detect the disease, so that responses are limited to the study group who are truly diseased and few false positive are produced. Once a screening process is complete, a diagnosis is needed to establish the disease in those who are suspected of having it and rule out those persons screened who are suspected of being diseased but are not (88).

The receiver operating characteristic (ROC) curve

In 1971, Lusted described how a method used often in psychophysics could be adopted for medical decision making. This method overcomes the limitation of a

single sensitivity and specificity pair and the summary measures associated with single sensitivity and specificity pair and the summary measures associated with single sensitivity and specificity pairs by including all of the decision thresholds. A receiver operating characteristic (ROC) curve is a method of describing the intrinsic accuracy of a test apart from the decision thresholds. Since the 1970s, it has been the most valuable tool for describing and comparing diagnostic tests.

An ROC curve is a plot of a test's sensitivity (plotted on the y axis) versus its false positive fraction (FPF), or (100 – specificity and plotted on the x axis). Each point on the graph is generated by a different decision threshold.

Diagnostic test with ROC curves above the chance diagonal have at least some ability to discriminate between patients with and without the condition. The closer the curve is to the (0, 100) point (left upper corner) the better the test (89).

The ROC curve area has several interpretations:

1. The average value of sensitivity for all possible values of specificity.

2. The average value of specificity for all possible values of sensitivity.

3. The probability that a randomly selected patient with the condition has a test result indicating greater suspicion than that of a randomly chosen patient without the condition.

Table 19. The sensitivity and specificity of the samples by the variables to identify those with BMI of 25 kg/m^2

	BMI		Sensitivity	Specificity
	< 25	**≥ 25**		
SBP				
Normal	155	44	0.43	0.78
High-Risk	123	91		
DBP				
Normal	233	79	0.55	0.75
High-Risk	45	56		
TC				
Normal	78	24	0.36	0.76
High-Risk	200	111		
TG				
Normal	230	77	0.55	0.75
High-Risk	48	58		
LDL-C				
Normal	112	48	0.34	0.70
High-Risk	166	87		
HDL-C				
Normal	262	110	0.61	0.70
High-Risk	16	25		

Table 20. The sensitivity and specificity of the samples by the variables to identify those with BMI of 24.5 kg/m^2

	BMI		**Sensitivity**	**Specificity**
	< 24.5	**≥ 24.5**		
SBP				
Normal	135	64	0.50	0.68
High-Risk	107	107		
DBP				
Normal	209	106	0.64	0.66
High-Risk	36	65		
TC				
Normal	72	30	0.45	0.71
High-Risk	107	141		
TG				
Normal	203	104	0.63	0.66
High-Risk	39	67		
LDL-C				
Normal	99	61	0.43	0.62
High-Risk	143	110		
HDL-C				
Normal	227	145	0.63	0.61
High-Risk	15	26		

Table 21. The sensitivity and specificity of the samples by the variables to identify those with BMI of 24 kg/m^2

	BMI		Sensitivity	Specificity
	< 24	**≥ 24**		
SBP				
Normal	127	72	0.57	0.64
High-Risk	93	121		
DBP				
Normal	188	124	0.68	0.60
High-Risk	32	69		
TC				
Normal	68	34	0.51	0.67
High-Risk	152	159		
TG				
Normal	186	121	0.68	0.61
High-Risk	34	72		
LDL-C				
Normal	91	68	0.49	0.57
High-Risk	129	125		
HDL-C				
Normal	209	162	0.74	0.56
High-Risk	11	31		

Table 22. The sensitivity and specificity of the samples by the variables to identify those with BMI of 23.5 kg/m^2

	BMI		**Sensitivity**	**Specificity**
	< 23.5	**≥ 23.5**		
SBP				
Normal	111	88	0.63	0.56
High-Risk	80	134		
DBP				
Normal	163	149	0.72	0.56
High-Risk	28	73		
TC				
Normal	65	37	0.59	0.64
High-Risk	126	185		
TG				
Normal	162	145	0.73	0.53
High-Risk	29	77		
LDL-C				
Normal	87	73	0.39	0.54
High-Risk	104	149		
HDL-C				
Normal	181	191	0.76	0.49
High-Risk	10	31		

Table 23. The sensitivity and specificity of the samples by the variables to identify those with BMI of 23 kg/m^2.

	BMI		Sensitivity	Specificity
	< 23	**≥ 23**		
SBP				
Normal	102	97	0.66	0.51
High-Risk	72	142		
DBP				
Normal	149	163	0.75	0.48
High-Risk	25	76		
TC				
Normal	61	42	0.64	0.60
High-Risk	113	198		
TG				
Normal	149	158	0.76	0.49
High-Risk	25	81		
LDL-C				
Normal	82	78	0.64	0.51
High-Risk	92	161		
HDL-C				
Normal	166	206	0.80	0.45
High-Risk	8	33		

Table 24. The sensitivity and specificity of the samples by the variables to identify those with BMI of 22.5 kg/m^2

	BMI		**Sensitivity**	**Specificity**
	< 22.5	**≥ 22.5**		
SBP				
Normal	87	112	0.75	0.44
High-Risk	54	160		
DBP				
Normal	122	190	0.81	0.39
High-Risk	19	82		
TC				
Normal	56	46	0.73	0.55
High-Risk	85	226		
TG				
Normal	124	183	0.84	0.40
High-Risk	17	89		
LDL-C				
Normal	73	87	0.72	0.46
High-Risk	68	185		
HDL-C				
Normal	134	238	0.83	0.36
High-Risk	7	34		

Table 25. The sensitivity and specificity of the samples by the variables to identify those with BMI of 22 kg/m^2

	BMI		**Sensitivity**	**Specificity**
	< 22	**≥ 22**		
SBP				
Normal	75	124	0.86	0.36
High-Risk	38	176		
DBP				
Normal	101	211	0.88	0.32
High-Risk	12	89		
TC				
Normal	52	50	0.80	0.51
High-Risk	61	250		
TG				
Normal	100	207	0.88	0.33
High-Risk	13	93		
LDL-C				
Normal	64	96	0.81	0.40
High-Risk	49	204		
HDL-C				
Normal	107	265	0.85	0.29
High-Risk	6	35		

Table 26. The sensitivity and specificity of the samples by the variables to identify those with BMI of 21.5 kg/m^2

	BMI		Sensitivity	Specificity
	< 21.5	**≥ 21.5**		
SBP				
Normal	61	138	0.85	0.31
High-Risk	32	182		
DBP				
Normal	84	228	0.91	0.27
High-Risk	9	92		
TC				
Normal	43	59	0.84	0.42
High-Risk	50	261		
TG				
Normal	80	227	0.88	0.26
High-Risk	13	93		
LDL-C				
Normal	55	105	0.82	0.34
High-Risk	38	215		
HDL-C				
Normal	89	283	0.90	0.24
High-Risk	4	37		

Table 27. The sensitivity and specificity of the samples by the variables to identify those with BMI of 21 kg/m^2.

	BMI		Sensitivity	Specificity
	< 21	**≥ 21**		
SBP				
Normal	52	147	0.91	0.26
High-Risk	19	195		
DBP				
Normal	68	244	0.97	0.22
High-Risk	3	98		
TC				
Normal	38	64	0.89	0.37
High-Risk	33	278		
TG				
Normal	66	241	0.95	0.21
High-Risk	5	101		
LDL-C				
Normal	45	115	0.90	0.28
High-Risk	26	227		
HDL-C				
Normal	67	305	0.90	0.18
High-Risk	4	37		

BIOGRAPHY

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