Obesity and Risk Factors of Coronary Heart Disease in Healthy Thais : A Cross-Sectional Study

NITHI MAHANONDA, M.D.*,
WATTANA LEOWATTANA, M.D.**,
CHUNHAKASEM CHOTINAIWATTARAKUL, M.D.*,
MANOON SAMRANTHIN, M.D.*,
KIERTIJAI BHURIPANYO, M.D.*,
CHARUWAN KANGKAGATE, M.S. (Biostat)*,
SUWATCHAI PORNRATANARANGSI, M.D.***,

Abstract

Association between obesity and conventional risk factors for coronary artery disease is well established. Obesity is currently considered an independent risk for coronary artery disease. The relationship between body mass index (BMI) and fasting plasma lipids and glucose and blood pressures in non-obese subjects is not established. The authors studied relationships between BMI and lipids, and glucose, and blood pressure levels in healthy a population. The authors measured the weights and heights of 3,615 employees of a company during a routine yearly health examination.

There were 1,250 males aged 31.3 ± 6.6 and 2,365 females aged 29.3 ± 4.9 years old. The average BMI for males and females were 23.5 ± 3.6 and 20.1 ±3.0 respectively. The levels of total cholesterol (Chol), LDL-cholesterol, and triglyceride (TG), fasting plasma glucose (FPG) had a positive relationship with BMI ($r = 0.22, 0.26, 0.41, 0.20$; $p < 0.001$). HLD-cholesterol had a negative correlation with BMI ($r = -0.36$, $p<0.001$). Both systolic (SBP) and diastolic (DBP) blood pressures had a positive correlation with BMI. The association persisted after all values were adjusted by age and sex. BMI has a significant positive relationship with the conventional risk factors for coronary artery disease and a negative relationship with HDL-cholesterol.

Key word : Obesity, Risk Factors, Coronary Heart Disease

MAHANONDA N, BHURIPANYO K, LEOWATTANA W, et al
J Med Assoc Thai 2000; 83 (Suppl. 2); S35-S45

* Her Majesty Cardiac Center,
** Department of Clinical Pathology,
*** Department of Medicine, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700, Thailand.

The relationship between obesity and the risk of coronary heart disease remains controversial despite established association between an overweight person and unfavorable coronary risk-factors status. Obesity is a cause of diabetes mellitus, hypertension, and lipid abnormality[1-4]. Despite the biological plausibility of a strong link between adiposity and atherogenesis, epidemiologic studies have not consistently collaborated the association[5-7]. More recent reports[8-12] showed strong relationship of obesity and all causes of mortality, including cardiovascular disease, after adjustment of smoking status. These reports suggested the association between body weight as well as body mass index (BMI) and other factors including race, age and exercise status. These factors also have influence on coronary artery disease and its risk factors[13-16].

There are limited data about the influence of BMI on cardiovascular risk and its risk factors in Thai population. The objective of this study was to examine the association of BMI and conventional risk factors of coronary artery disease in healthy Thai population.

MATERIAL AND METHOD

Study population: The study subjects were 3,615 employees of Shinawatra Group who underwent a yearly routine physical check-up. They were 90 per cent of the total number of the company's employees. Participants were informed about the study, given a confidential questionnaire one week prior to their physical examination, and a blood sample was taken. The study was approved by the ethical committee of Siriraj Hospital. The questionnaire included information on demographic characteristics, personal history of acute and chronic diseases, and various aspects of behavior and life style.

All participants were examined by cardiologists. Their questionnaires were reviewed for completeness at the same time of the physical examination. Weight and height were measured by medical personnel using a standard balance scale. Weight was measured in light clothing and bare feet to the nearest 0.1 kg. Height was measured without shoes with participants standing with back square to the scale to the nearest 0.1 cm. Waist and hip circumference were measured by an Ohaus spring scale with subjects in standing position under light clothing at the level of smallest distance between the costal margin, iliac crest and the level of the greater trochanters to the nearest 0.1 cm. All measurements were done in duplicate during the same visit.

Blood pressure was measured in the right arm, with the subject in a relaxed, siting position. The average of at least two measurements to the nearest 2 mmHg with a mercury sphygmomanometer was used in all analyses. The third measurement was done if the first and second readings showed a difference greater than 10 mmHg.

Twenty milliliters of venous blood was collected in the morning after at least 12 hours fasting. It was immediately mixed with 1.5 mg of EDTA and centrifuged. Plasma for lipids and glucose analysis was stored at 4°C until being assayed. The analysis was performed within 2 hours. The rest of the blood sample was sealed and stored at -80°C for future analysis.

Laboratory Procedures: Plasma levels of total cholesterol (Chol) and triglyceride (TG) were measured by enzymatic procedures (Roche Diagnostics, Switzerland). High density lipoprotein (HDL) cholesterol was measured by dextran sulfate-magnesium precipitation followed by enzymatic determination of cholesterol[17-19]. The level of low density lipoprotein (LDL) cholesterol was derived from the Friedewald calculation. The plasma glucose (FPG) was determined by glucose oxidase method. The quality of these laboratory measurements were internally controlled by precinorm and precipath from Roche diagnostics and externally controlled with the Quality Assessment Program (QAP) with Roche Diagnostics with coefficients of variation for glucose, cholesterol, triglyceride and HDL-cholesterol of 2.18 per cent, 2.29 per cent, 3.09 per cent and 3.45 per cent respectively.

Statistical Analysis

The authors analyzed men and women separately. Data entries were done separately by 2 officers and checked for error. Any discrepancies of both entries or extreme values of any variable were rechecked and corrected if possible. Otherwise, they were excluded from analysis. The completeness of all variables was more than 99.6 per cent.

Continuous variables were presented as mean ± SD. Categorical data were presented in percentage. All statistical tests were two-tailed. Pearson

correlation and regression analysis were performed to examine the relationship between BMI and other factors. Student's t-test, ANOVA and chi-square test were used to determine the difference between groups of various risk factors. The effect of BMI on lipid profiles and blood pressure were assessed by using the General Linear Model multivariate ANCOVA. A p-value less than 0.05 is considered as significant result.

RESULTS

There were 3615 people who participated in the survey. There were more female than male participants in the population. Other demographic data and completeness of each variable are shown in Table 1. The average age of females was less than that of males. Men are more likely to smoke cigarettes and have higher BMI than women. Average laboratory results are demonstrated in Table 2. Men had significantly higher mean levels of total cholesterol, LDL-cholesterol, triglyceride and fasting plasma glucose compared to those of women ($p<0.001$,<0.001,<0.001 and < 0.001). However, the mean level of HDL cholesterol in women was 61.7 ± 14.6 mg/dl, which is higher than that of men ($p=0.001$). Men also had 119.7 ± 12.7 and 80.2 ± 9.5 mmHg of average systolic (SBP) and diastolic (DBP) blood pressures higher than those in women ($p<0.001$, <0.001).

Obesity indices: Figures 1 and 2 showed that there were significant correlations of all three

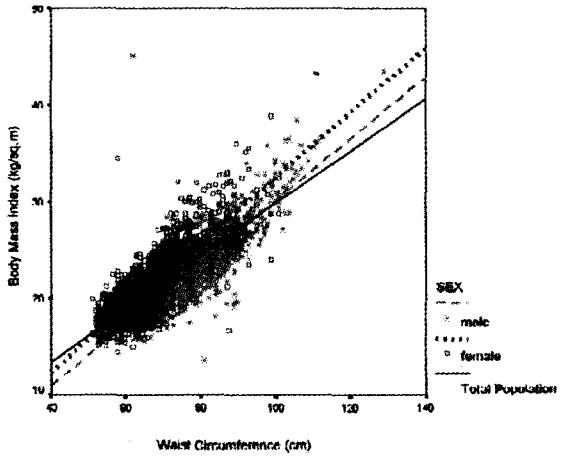
Table 1. Demographic information and history of illness.

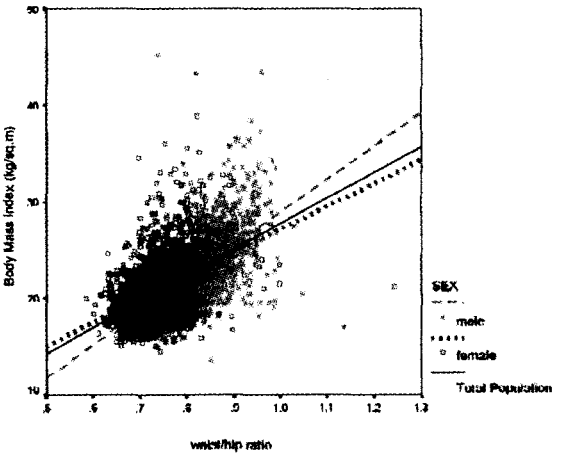
	Male	Female	p-value	Completeness of data %
N	1250	2365		
Mean age (y)	31.3 ± 6.6	29.3 ± 4.9	<0.001	99.9
Smoking	360 (30%)	118 (5.3%)	<0.001	95.3
Education:			<0.001	97.4
- below bachelor	421 (34.7%)	644 (5.3%)		
- bachelor or high	793 (65.3%)	1664 (72.1%)		
Income (baht)			<0.001	97.6
- < 20,000	539 (44%)	1148 (49.8%)		
- 20,000-50,000	406 (33.1)	720 (31.3%)		
- > 50,000	281 (22.9%)	435 (18.9%)		
Weight (kg)	65.5 ± 11.0	50.7 ± 7.9	<0.001	99.3
Height (cm)	168.5 ± 6.3	156.5 ± 5.3	<0.001	99.4
BMI (kg/m^2)	23.06 ± 3.57	20.71 ± 2.99	<0.001	99.3
Waist circumference (cm)	78.1 ± 9.4	64.9 + 7.2	<0.001	99.3
Waist-hip ratio	0.83 ± 0.06	0.74 ± 0.06	<0.001	99.3
History DM	11 (0.9%)	16 (0.7%)	0.488	98.4
History HTN	88 (7.2%)	78 (3.4%)	<0.001	98.1
History hyperlipidemia	140 (11.4%)	120 (5.2%)	<0.001	98.3
History of regular exercise	431 (35.5%)	383 (16.7%)	<0.001	96.9

Abbreviation: DM - diabetes mellitus, HTN – hypertension

Table 2. Mean levels of lipids, fasting plasma glucose and blood pressures.

	Total	Male	Female	p-value
Chol (mg/dL)	200.5 ± 36.6	207.0 ± 39.5	197.1 ± 34.5	< 0.001
LDL (mg/dL)	124.1 ± 32.8	131.7 ± 35.9	120.1 ± 30.4	< 0.001
HDL(mg/dL)	58.1 ± 14.7	51.5 ± 12.3	61.7 ± 14.6	< 0.001
TG (mg/dL)	91.5 ± 60.0	119.0 ± 81.7	76.9 ± 37.0	< 0.001
FPG (mg/dL)	89.5 ± 12.0	92.9 ± 14.2	87.7 ± 10.3	< 0.001
SBP (mmHg)	113.4 ± 12.5	119.7 ± 12.7	110.1 ± 11.1	< 0.001
DBP (mmHg)	75.6 ± 9.4	80.2 ± 9.5	73.2 ± 8.3	< 0.001





	r	slope	equation
BMI & W	0.816*	0.27*	BMI = 0.27(W) + 2.68
BMI & W/H	0.554*	26.73*	BMI = 26.73(W/H) +0.99
W/H & W	0.856*	0.006*	W/H = 0.006(W) + 0.358

* p-value <0.001
Abbreviation : BMI - body mass index (kg/m^2), W - waist circumference (cm), W/H - waist to hip ratio, r = correlation coefficient

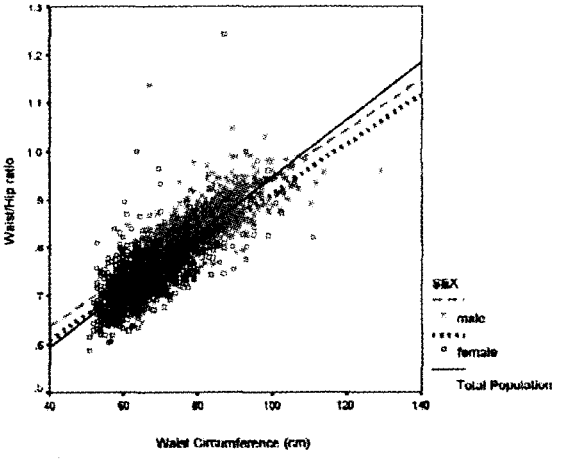


Fig. 1. Relationship of different parameters of obesity.

indices of obesity. Correlation between body mass index (BMI) and waist circumference (r=0.8, p< 0.001) was better than BMI and waist-hip (W/H) ratio (r=0.6, p<0.001). With the same waist circumference, females tended to have higher BMI than males. Figure 2 shows that BMI of both men and women varied with age (r=0.3, p<0.001). At the same age, men had higher BMI than women. BMI increased 1.6 kg/m^2 in every 10 years of life (p< 0.001). After adjustment with sex, BMI still varied significantly with age (p <0.001, Table 3). Average BMI in smokers was 22.92 ± 3.71 kg/m^2 and nonsmokers was 21.29 ± 3.29 (p< 0.001). Despite this association of smoking and BMI, the effect of smoking on other risk factors did not appear to be significant on a general linear model from multivariate of analysis of covariance model.

BMI and lipids levels: Figure 3 shows that BMI has a significant linear relationship with levels of total cholesterol (r=0.2), LDL cholesterol (r=0.3), triglyceride (r=0.4), and HDL cholesterol (r = -0.4) with a p-value less than 0.001. Every 1 kg/m^2 increment of BMI, levels of total cholesterol, LDL cholesterol, and triglyceride increased 0.9 mg/dl, 2.8 mg/dl, and 8.9 mg/dl respectively in males and 0.2 mg/dl 1.8 mg/dl and 3.6 mg/dl respectively in females (p=0.001). Table 4 shows that the percentage of subjects with abnormal lipid levels were increasing in higher BMI groups. After being

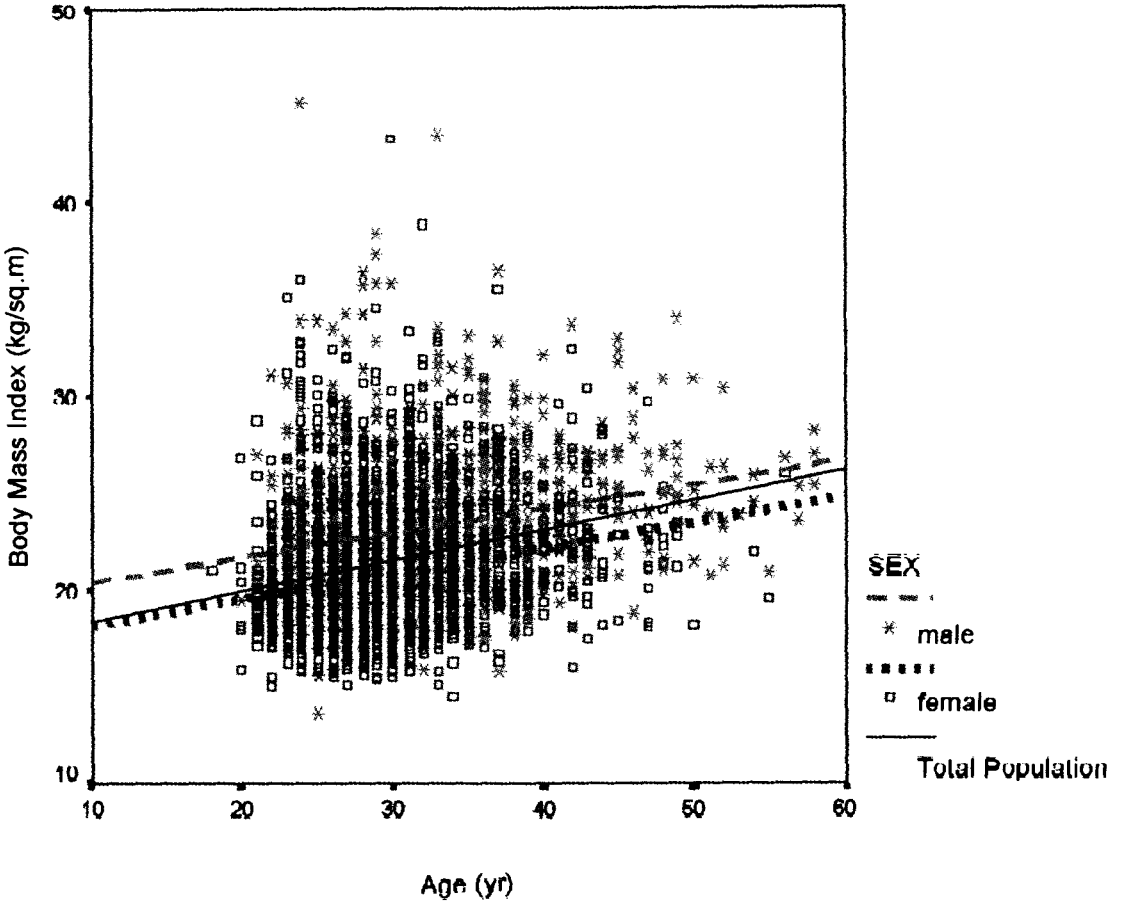


Fig. 2. Relationship between body mass index (BMI) and age. BMI = 16.8 + 0.16 (Age), r = 0.26 (p < 0.001).

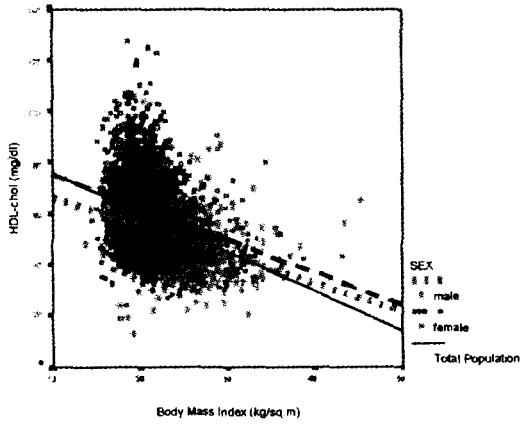
Table 3. Mean body mass index in different age groups.

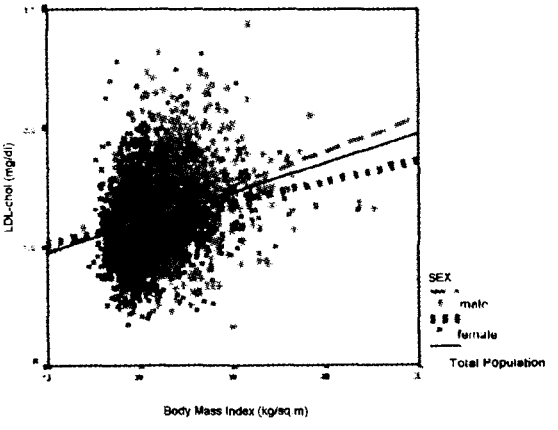
Age Group (yr)	BMI (kg/m^2)			BMI adjusted for sex
	Male	Female	Total	
< 25	22.20 ± 0.20	20.18 ± 0.13	20.82 ± 20.12	21.19 ± 0.12
25-29	22.65 ± 0.17	20.26 ± 0.11	20.95 ± 0.09	21.46 ± 0.10
30-34	22.95 ± 0.17	21.05 ± 0.12	21.67 ± 0.10	22.00 ± 0.10
35-39	23.76 ± 0.24	21.86 ± 0.22	22.75 ± 0.18	22.71 ± 0.16
40-44	24.37 ± 0.35	22.84 ± 0.40	23.70 ± 0.26	23.61 ± 0.27
≥ 45	25.46 ± 0.40	22.14 ± 0.64	24.54 ± 0.05	23.80 ± 0.38
p-value	-	-	<0.001	< 0.001

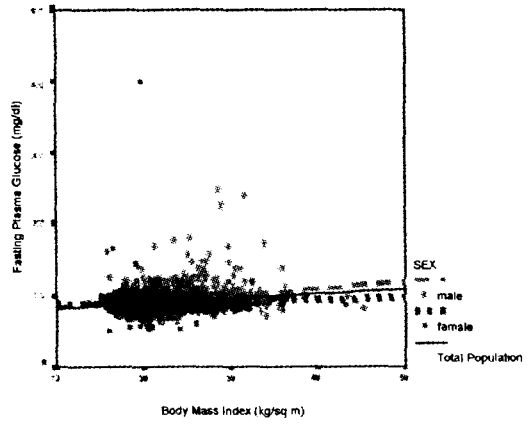
adjusted by age and sex (Table 5), the mean total cholesterol, LDL cholesterol and triglyceride levels still varied with BMI, while the mean HDL cholesterol had a reverse relationship with BMI. (p<0.001).

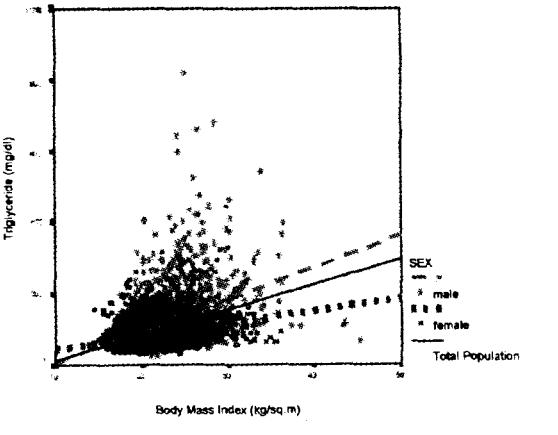
BMI and fasting plasma glucose: Similar to the total cholesterol level, fasting plasma glucose increases as BMI increases in both male and female groups (Figure 3). The relationship was a

linear fashion. Fasting plasma glucose increased 0.9 mg/dl in males and 0.2 mg/dl in females in every increment of 1 kg/m^2 of BMI (p= < 0.001, < 0.001). The percentage of people in each BMI subgroup with abnormal fasting plasma glucose (110-125 mg/dl and > 125 mg/dl) increased in subgroups of higher BMI (p<0.001) (Table 5). The mean fasting plasma glucose levels both before and









Relationship between BMI and fasting lipids, BMI and fasting plasma glucose.

	r	slope	equation
Chol	0.22	2.41	chol = 2.41(BMI) + 148.7
TG	0.41	7.22	TG = 7.22(BMI) - 63.97
HDL	-0.36	-1.56	HDL = -0.36(BMI) + 91.68
LDL	0.26	2.52	LDL = 0.26(BMI) + 69.75
FPG	0.20	0.71	FPG = 0.20(BMI) + 74.34

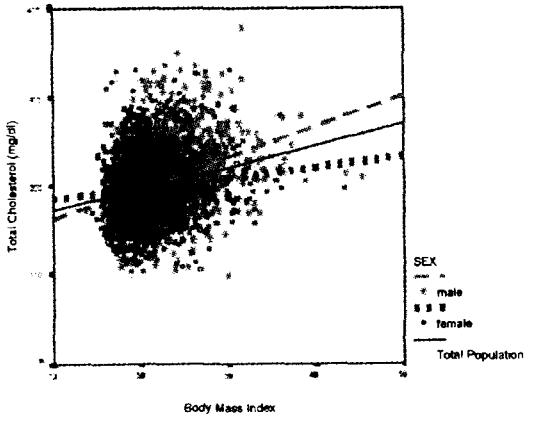


Fig. 3. BMI and fasting plasma lipid.

Table 4. Number (%) of subjects with abnormal blood test and hypertension in each groups of BMI.

	Body Mass Index (kg/m^2)									p-value
	< 18.5	18.5-20.4	20.5-21.9	20.0-23.4	23.5-24.9	25.0-26.4	26.5-27.9	28.0-29.9	≥ 30.0	
Chol	52 (9.0%)	104 (10.2%)	73 (10.8%)	83 (17.5%)	61 (20.1%)	43 (21.7%)	29 (22.8%)	25 (30.5%)	26 (30.2%)	< 0.001
LDL	38 (6.6%)	84 (8.2%)	70 (10.4%)	91 (19.2%)	68 (22.4%)	41 (20.7%)	32 (25.2%)	19 (23.2%)	25 (29.1%)	<0.001
HDL (6.0%)	35	58 (5.7%)	46 (6.8%)	38 (8.0%)	39 (12.9%)	32 (16.2%)	24 (18.9%)	18 (22.0%)	27 (31.4%)	< 0.001
TG	2 (0.3%)	11 (1.1%)	14 (2.1%)	23 (4.8%)	27 (8.9%)	31 (15.7%)	25 (19.7%)	15 (18.3%)	22 (25.6%)	<0.001
FPG	7 (1.2%)	10 (1.0%)	13 (1.9%)	10 (2.1%)	12 (3.9%)	11 (5.6%)	9 (7.1%)	9 (10.8%)	10 (11.6%)	< 0.001
HTN	9 (1.6%)	26 (2.5%)	31 (4.6%)	39 (8.2%)	44 (14.4%)	35 (17.6%)	27 (21.1%)	22 (26.2%)	29 (33.3%)	< 0.001

Chol- total cholesterol > 240 mg/dl, LDL - LDL cholesterol > 160 mg/dl, HDL- HDL cholesterol < 35 mg/dl
TG- triglyceride > 200 mg/dl, FPG - impaired fasting plasma glucose ≥ 110 mg/dl
HTN- hypertension : systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥90 mmHg.

after being adjusted by age and sex of people in high BMI subgroups were higher than the levels of those in lower BMI subgroups (p<0.001 Table 5).

BMI and blood pressure: Both systolic (SBP) and diastolic (DBP) blood pressures also increased as BMI increased in both males and females (Figure 3). At the same BMI level, men had higher systolic and diastolic blood pressures than women. With 1 kg/m^2 increment of BMI, there would be 1.1 mmHg and 1.0 mmHg higher systolic and diastolic blood pressures in males and 1.0 mmHg and 0.5 mmHg higher systolic and diastolic blood pressures in females (p< 0.001).

Table 4 shows that the percentage of people with hypertension (SBP ≥ 140 or DBP ≥ 90) was higher in subgroups of people with high BMI, compared to those in the low BMI subgroups. The mean blood pressure before and after adjustment by age and sex were also higher in higher BMI subgroups compared to low BMI subgroups, p < 0.001 (Table 5).

DISCUSSION

In this study, we demonstrated the crossectional relationship between body mass index (BMI) and conventional risk factors of coronary artery disease in a healthy Thai population. Because of good correlations between BMI and waist circumference, and waist/hip ratio, we used only BMI as our index of obesity. We observed a strong relationship of BMI and fasting plasma lipids, and of glucose and blood pressure levels.

We also found that a prevalence of abnormal fasting plasma lipids and glucose levels and hypertension were higher in people with high BMI compared to those with low BMI. These findings were consistent with the findings and reports of several large cohort studies[20-30]. These relationships of risk factors and BMI were presented in both men and women. However, we found that fasting plasma lipids, glucose and blood pressures in men seemed to be more sensitive to the change of BMI than those in women. This may be a result of older males than females in our study population. We observed crossectional association of age and BMI in both males and females. This association has been reported previously[31].

In our study, those who smoked had higher BMI than nonsmokers. This finding was different to what has been previously reported[8]. However, that report recruited only women to the study. Another potential explanation of the difference in this finding is the presence of an older male population than a female one. In the male population, there was

Table 5. Mean (values of lipids, glucose and blood pressure of each BMI groups before and after adjustment with age and sex.

		BMI (kg/m^2)									
		< 18.5	18.5 - 20.4	20.5 - 21.9	22.0 - 23.4	23.5-24.9	25.0-26.4	26.5-27.9	28.0-29.9	≥ 30	p-value
Chol	a)	190.8 ± 1.4	195.9 ± 1.0	196.8 ± 1.3	206.7 ± 1.6	206.5 ± 2.3	213.1 ± 2.8	215.8 ± 3.9	221.3 ± 4.3	223.4 ± 4.4	<0.001
	b)	189.6 ± 2.1	195.7 ± 1.4	197.0 ± 1.4	206.0 ± 1.6	203.1 ± 2.1	208 ± 2.6	210.4 ± 3.2	218.3 ± 3.9	219.1 ± 3.8	<0.001
LDL	a)	113.7 ± 1.2	118.0 ± 0.9	121.5 ± 1.2	131.7 ± 1.4	132.1 ± 2.0	136.4 ± 2.5	139.8 ± 3.5	143.2 ± 3.8	145.6 ± 3.6	<0.001
	b)	113.8 ± 1.9	118.9 ± 1.2	122.3 ± 1.3	131.4 ± 1.4	129.5 ± 1.8	133.0 ± 2.3	136.1 ± 2.9	140.8 ± 3.5	143.1 ± 3.4	<0.001
HDL	a)	63.1 ± 0.6	62.9 ± 0.5	58.9 ± 0.6	55.6 ± 0.6	51.8 ± 0.7	48.9 ± 0.8	47.9 ± 1.0	47.9 ± 1.4	44.7 ± 1.1	<0.001
	b)	60.8 ± 0.8	60.5 ± 0.5	57.9 ± 0.5	55.2 ± 0.6	52.2 ± 0.8	49.6 ± 1.0	48.4 ± 1.2	48.4 ± 1.5	45.1 ± 1.5	<0.001
TG	a)	69.5 ± 1.2	75.0 ± 1.1	81.8 ± 1.6	95.6 ± 2.4	113.7 ± 4.4	139.0 ± 7.2	141.8 ± 7.6	150.7 ± 10.6	165.1 ±10.4	<0.001
	b)	74.8 ± 3.1	82.0 ± 2.0	84.1 ± 2.1	95.9 ± 2.4	107.2 ± 3.0	126.9 ± 3.9	130.4 ± 4.8	145.0 ± 5.8	154.1 ± 5.7	<0.001
FPG	a)	87.3 ± 0.4	88.0 ± 0.4	89.9 ± 0.4	89.5 ± 0.4	91.2 ± 0.6	94.0 ± 0.9	94.6 ± 1.2	97.3 ± 2.9	97.3 ± 2.4	<0.001
	b)	88.9 ± 0.7	89.0 ± 0.5	89.3 ± 0.5	89.4 ± 0.5	90.1 ± 0.7	92.0 ± 0.9	92.4 ± 1.1	95.7 ± 1.3	95.5 ± 1.3	<0.001
SBP	a)	108.2 ± 0.4	110.2 ± 0.3	112.2 ± 0.4	115.4 ± 0.6	119.3 ± 0.7	120.4 ± 0.9	122.0 ± 1.2	124.0 ± 1.4	124.0 ± 1.4	<0.001
	b)	111.0 ± 0.6	112.3 ± 0.4	113.4 ± 0.4	115.4 ± 0.5	117.9 ± 0.6	118.2 ± 0.8	120.0 ± 1.0	122.5 ± 1.2	122.6 ± 1.2	<0.001
DBP	a)	72.3 ± 0.3	73.5 ± 0.3	74.6 ± 0.3	76.3 ± 0.4	79.3 ± 0.5	80.6 ± 0.7	81.6 ± 0.9	82.9 ± 1.3	84.4 ± 1.2	<0.001
	b)	73.8 ± 0.5	45.2 ± 0.3	75.5 ± 0.3	76.3 ± 0.4	78.3 ± 0.5	78.8 ± 0.6	80.1 ± 0.8	81.8 ± 0.9	83.3 ± 0.9	<0.001

a) mean ± SE before adjustment, b) after adjustment by age and sex.

a higher prevalence of smoking, and they also had higher average BMI compared to the female population.

After being adjusted by age and sex, we still found significant relationships between BMI and lipids, and glucose and blood pressure levels. Adjustment with smoking status did not have any effect on the levels of the risk factors. These findings were consistent with previous reports[8,26-30]. The increase in the prevalence of risk factors was also associated with higher mortality in a longitudinal study[8].

In conclusion, increasing BMI is strongly associated with an increasing risk of having abnormal plasma lipids and impaired fasting glucose levels. Moreover, men and women with higher BMI are more likely to have higher blood pressure. Multivariate analysis indicates that although relationship of BMI and these risk factors is attributable to the influence of age and sex on BMI, a moderate relationship persists after adjustment by age and sex. Other factors, such as physical activity and dietary intake, may explain the relationship and should be further studied.

ACKNOWLEDGEMENT

The authors would like to thank cardiologists, cardiac fellows, and nursing and secretariat staff from Her Majesty Cardiac Center, and technicians from the Department of Clinical Pathology, Siriraj Hospital for their help, surveys and meticulous data records. This study was partly sponsored by a grant from Mahidol University, and Shinawatra Group. Support from the company's Human Resource Department, and co-operation from all employees are sincerely appreciated.

(Received for publication on September 22, 2000)

REFERENCE

1. Van Itallie TB. Obesity: adverse effects on health and longevity. Am J Clin Nutr 1979;32:Suppl: 2723-33.
2. Bierman EL, Hirsch J. Obesity. In: Williams RH, ed. Textbook of endocrinology. 6th ed. Philadelphia: W.B. Saunders. 1981:907-21.
3. Mann GV. The influence of obesity on health. N Engl J Med 1974;291:178-85.
4. Lew EA, Garfinkel L. Variations in mortality by weight among 750,000 men and women. J Chronic Dis 1979;32:563-76.
5. Keys A. Is overweight a risk factor for coronary heart disease? Cardiovasc Med 1979;4:1233-43.
6. Andres R. Effect of obesity on total mortality. Int J Obes 1980;4:381-6.
7. Bradley PJ. Is obesity an advantageous adaptation? Int J Obes 1982;6:43-52.
8. Allison DB, Gallagher D, Heo M, Pi-Sunyer FX, Heymsfiedl SB. Body mass index and all-cause mortality among people age 70 and over: the Longitudinal Study of Aging. Int J Obes Relat Metab Disord 1997;21:424-31.
9. Cornoni-Huntley JC, Harris TB, Everett DF, et al. An overview of body weight of older persons, including the impact on mortality. J Clin Epidemiol 1991;44:743-53.
10. Diehr P, Bild DE, Harris TB, Duxbury A, Siscovick D, Rossi M. Body mass index and mortality in nonsmoking older adults: the Cardiovascular Health Study. Am J Public Health 1998;88:623-9.
11. Durazo-Arvizu R, Cooper RS, Luke A, Prewitt TE, Liao Y, McGee DL. Relative weight and mortality in U.S. blacks and whites: whites: findings from representative national population samples. Ann Epidemiol 1997;7:383-95.
12. Folsom AR, Kaye Sa, Sellers TA, et al. Body fat distribution and 5-year risk of death in older women. JAMA 1993;269:483-7. [Erratum, JAMA 1993;269:1254].
13. Wood PD, Stefanick ML, Dreon DM, et al. Changes in plasma lipids and lipoproteins in overweight men during weight loss though dieting as compared with exercise. N Engl J Med 1988; 319:1173-9.
14. Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity as an independent risk factor for cardiovascular disease : a 26-year follow-up of participants in the Framingham Heart Study. Circulation 1983;67:968-77.
15. Manson JE, Colditz GA, Stampfer MJ, et al. A prospective study of obesity and risk of coronary heart disease in women. N Engl J Med 1990;322: 882-9.
16. Larsson B, Svardsudd K, Welin L, Wilhelmsen L, Bjorntorp P, Tibblin G. Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow-up of participants in the study of men born in 1913.Br Med J 1984;288:1401-4.

17. Allain CC, Poon LS, Chan CS, Richmond W, Fu PC. Enzymatic determination of total serum cholesterol. Clin Chem 1974;20:470-5.
18. Sampson EJ. Demers LM, Krieg AF. Faster enzymatic procedure for serum triglycerides. Clin Chem 1975;21:1983-5.
19. Warnick GR, Benderson J, Albers JJ. Dextran sulfate-Mg^{2+} precipitation procedure for quantitation of high-density-lipoprotein cholesterol. Clin Chem 1982;28:1379-88.
20. Ernst ND, Obarzanek E, Clark MB, Briefel RR, Brown CD, Donato K. Cardiovascular health risks related to overweight. J Am Diet Assoc 1997;97 (suppl 7):S47-S51.
21. Hanson RL, Narayan KMV, McCance DR, et al. Rate of weigh gain, weight fluctuation and incidence of NIDDM. Diabetes. 1995;261-6.
22. Colditz GA, Willett WC, Rotnitsky A, Manson JE. Weight gain as a risk factor for clinical diabetes mellitus in women. Ann Intern Med 1995;122: 481-6.
23. Huang Z, Willett WC, Manson JE, Colditz GA, Body weight, weight change, an hypertension in women. Ann Intern Med 1998;128:81-8.
24. Lapidus L, Bengtsson C, Larsson B, Pennert K, Rybo E, Sjostrom L. Distribution of adipose tissue and risk of cardiovascular disease and death: a12-year follow-up of participants in the population study of women in Gothenburg, Sweden. Br Med J 1984;289:1257-61.
25. Willett WC, Manson JE, Stampfer MJ, et al. Weight, weight change, and coronary heart disease in women: risk within the "normal: weight range. JAMA. 1995;273:464-5.
26. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. JAMA 1999;282:1523-9.
27. Grundy -*SM, Bazzarre T, Cleeman, J. Prevention conference V beyond secondary prevention: Identifying the high-risk patient for primary prevention medical office assessment. Circulation 2000: 101:1-9.
28. Carretero OA, Oparil S. Essential hypertension Part II: Treatment. Circulation 2000; 101:446-53.
29. Carretero OA, Oparil S. Essential hypertension part I: Definition and Etiology. Circulation 2000;101: 329-35.
30. Wei M, Kampert JB, Barlow CE, Nichaman MZ, Gibbons LW, Paffenbarger RS, Blair SN. Relationship between low cardiorespiratory fitness and mortality in normal-weight, overweight and obese men. JAMA 1999;282:1547-53.
31. Stevens J, Jianwen C, Pamuk ER, Willamson DF, Thun MJ, Woods JL. The effect of age on the association between body-mass index and mortality. N Engl J Med 1998;338:1-7.

ความสัมพันธ์ของความอ้วนกับปัจจัยเสี่ยงอื่นๆ ของโรคหลอดเลือดหัวใจในประชากรไทยสุขภาพดี

นิธิ มหานนท์, พ.บ., เกียรติชัย ภูริปัญโญ, พ.บ.*, วัฒนา เลี้ยววัฒนา, พ.บ.**, จารุวรรณ คังคะเกตุ, วท.ม. (ชีวสถิติ)*, ชุณหเกษม โชตินัยวัตรกุล, พ.บ.*, สุวัจชัย พรรัตนรังสี, พ.บ.***, มนูญ สำราญถิ่น, พ.บ.**

ความอ้วนเป็นปัจจัยเสี่ยงที่สำคัญของโรคหลอดเลือดหัวใจ และยังเป็นสาเหตุของปัจจัยเสี่ยงอื่น ๆ ของโรคหลอดเลือดหัวใจ เช่น ความดันโลหิตสูง, เบาหวาน และไขมันในเลือดสูง การศึกษานี้ ศึกษาประชากร 3615 ราย เมื่อดูความสัมพันธ์ของ cross-section ของความอ้วน และปัจจัยเสี่ยงต่าง ๆ ของโรคหลอดเลือดหัวใจ โดยพบว่าดัชนีมวลกาย ซึ่งเป็นค่าที่วัดดูความอ้วนนั้น มีความสัมพันธ์กับระดับไขมัน, น้ำตาล และความดันโลหิต ซึ่งค่านี้ยังคงมีความสัมพันธ์อย่างมีนัยสำคัญทางสถิติอยู่ ถึงแม้จะปรับด้านผลจากปัจจัยเรื่องอายุ และเพศ แล้ว

คำสำคัญ : ความอ้วน, ปัจจัยเสี่ยง, โรคหลอดเลือดหัวใจโคโรนารีย์

นิธิ มหานนท์, เกียรติชัย ภูริปัญโญ, วัฒนา เลี้ยววัฒนา และคณะ
จดหมายเหตุทางแพทย์ ฯ 2543; 83 (ฉบับพิเศษ 2): S35-S45

* สำนักงานศูนย์โรคหัวใจสมเด็จพระบรมราชินีนาถ,
** ภาควิชาพยาธิวิทยาคลินิก,
*** ภาควิชาอายุรศาสตร์, คณะแพทยศาสตร์ศิริราชพยาบาล, มหาวิทยาลัยมหิดล, กรุงเทพ ฯ 10700