

Measurement of Obesity by Anthropometry and Bioelectric Impedance Analysis: Correlation with Fasting Lipids and Insulin Resistance in an Asian Population

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Abstract

Obesity is associated with increased coronary artery disease risk. This is at least partially mediated by increased prevalence of other risk factors such as dyslipidaemia and insulin resistance. Various anthropometric indices have been used to quantify generalised and central obesity. Correlations between these measurements and risk factors are specific to the population and findings cannot be extrapolated to other ethnic groups. Bioelectric impedance analysis (BIA) is a simple means of estimating percentage body fat. We compared body mass index (BMI), waist circumference, waist-hip ratio (WHR) and percentage body fat measure by BIA as predictors of fasting lipid profiles and insulin resistance in 109 Singaporean Chinese. BMI was significantly correlated with insulin resistance and there appeared to be a threshold at 26 kg/m² above which the regression line became more steep. WHR best predicted fasting triglyceride in both men and women. In women, it was inversely correlated with high density lipoprotein cholesterol. A similar association was seen in men but this did not reach statistical significance. We conclude that measurement of BMI and WHR should be part of the assessment of cardiovascular risk in our population as they connote different aspects of risk. BIA was not found to be a useful tool for the prediction of coronary artery disease risk factors in our population.

Ann Acad Med Singapore 1999; 28:445-50

Key words: Body mass index, Chinese, Coronary artery disease risk, Waist circumference, Waist hip ratio

Introduction

Obesity is a risk factor for cardiovascular disease.¹ This risk is partially mediated by the correlation between obesity and other coronary artery disease risk factors.^{1,2} The distribution of fat is important as central obesity is thought to be a better guide to coronary artery disease risk through its association with insulin resistance, dyslipidaemia (high triglyceride, low high density lipoprotein cholesterol and predominance of small dense low density lipoprotein particles) and hypertension.^{3,4}

Several anthropometric indices have been developed to measure obesity and are associated with coronary artery disease.⁵⁻⁷ Perhaps the most widely used has been the body mass index (BMI). Others include skin fold thickness. These are chosen because other methods for assessing adiposity such as underwater weighing, total body potassium and dilution methods are time consuming and of limited application, particularly for population studies. Likewise, several indices of assessment of fat distribution have been used in the assessment of

coronary artery disease risk.^{2,6-9} Data on anthropometric measurements have been developed largely in Western populations. Relationships between anthropometric indices and lipoprotein parameters have been found to differ in different populations^{9,10} and cannot be generalised to other ethnic groups. It is therefore important that we find out the best predictors of risk in our own population.

Bioelectric impedance analysis (BIA) has been applied to the estimation of fat free mass or lean body mass with reasonable success. It is a simple and non-invasive technique, which allows a rapid assessment. It has been shown to be correlated with lipid parameters in other populations.¹¹

The aim of the study was to evaluate BIA compared to various anthropometric indices, namely BMI, waist circumference and waist-hip ratio (WHR), as a predictor of coronary artery disease risk factors. Relationships between the various measures of obesity and insulin resistance, blood pressure and fasting lipids were explored.

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Materials and Methods

We studied 109 normal volunteers with no past medical history and who were not on regular medication. We excluded individuals who had serious illness or surgery in the previous 3 months. Patients were seen after a 10-hour fast. They were advised to have no coffee for the 4 hours prior to the consultation and were asked to abstain from drinking excessive amounts of water and exercising in the 12 hours prior to attendance at the clinic.

Anthropometry

Height, weight, waist circumference and hip circumference were measured with the patient in light clothing and no shoes. Weight was measured to the nearest 0.1 kg on a digital scale (SECA) with the patient in light clothing. Height was measured with a wall-mounted stadiometer to the nearest 0.1 cm. BMI was calculated from the formula $\text{weight (kg)} / [\text{height (m)}]^2$. The investigation physician measured the waist and hip circumferences using a non-elastic tape measure. The waist was defined as the narrowest circumference between the costal margin and the iliac crests and the hips as the widest circumference between the waist and the thigh.^{4,11}

Blood pressure was measured in the left arm with the patient seated. Two measurements were made and the mean of the 2 values was used. Korotkov phase V was taken as the diastolic blood pressure. The mean arterial pressure was calculated as $2/3 (\text{systolic blood pressure} - \text{diastolic blood pressure}) + \text{diastolic blood pressure}$.

BIA

BIA was performed using a SEAC Bioimpedance meter (UniQuest Ltd), model SFB3 version 1.0 (for windows). This is a tetrapolar, multiple frequency bioimpedance meter. Bioimpedance was measured over a range of frequencies (4 to 1024 kHz). Fat-free mass was calculated according to the formula described by Lukaski et al.¹² Percentage body fat was calculated ($\text{weight-fat free mass} / \text{weight}$).

Biochemical Analysis

Serum for the lipid profile was collected in plain tubes. Serum for glucose was collected in fluoride oxalate tubes. The serum total cholesterol, triglyceride, high density lipoprotein cholesterol and glucose were assayed by dry chemistry method using the Kodak Ektachem Clinical Chemistry Slide and read on the Kodak Ektachem 700 analyser in the Biochemistry Department of the Singapore General Hospital.

The insulin assay was carried out using the Abbot IMX Insulin Assay, which is a Micro Particle Enzyme Immunoassay. Insulin resistance was then derived by homeostasis model assessment.¹³ This method was developed using a mathematical model to estimate insulin

resistance. It shows a high degree of correlation with fasting insulin, insulin mediated glucose disposal in clamp studies and insulin response to intravenous glucose challenge.”

Statistical Analysis

Statistical analysis was performed using the SPSS version 7.5 for Windows. Fasting triglyceride was logarithmically transformed to reduce skewness. Data were back-transformed to natural units for presentation in the Tables. The Student's *t*-test was used to compare means of parametric data. The Mann-Whitney *U* method was used to compare non-parametric data. Multivariate analysis was carried out using stepwise multiple regression analysis.

Subject Characteristics

Of the 109 subjects studied, 53 were females and 56 males. Their ages ranged from 21 to 61 years with a mean of 40.6 years. The range of BMI was from 18.67 to 45.11 kg/m² (mean 25.36 kg/m²). Means for the other measured parameters are shown in Table I where the breakdown by sex is also given.

Male subjects in this study were heavier but this was compensated for by greater height giving rise to similar BMI to the women. Despite the similar BMI, women generally had greater percentage body fat measured by BIA compared to men who were more centrally obese, as suggested by the greater waist circumference and waist hip ratio (WHR). The total and low density lipoprotein cholesterol did not differ significantly between the sexes. However, fasting triglyceride was higher and high density lipoprotein cholesterol lower in men than in women. Mean arterial pressure was slightly higher in men than in women.

Results

Since several of the anthropometric indices as well as the lipid parameters differed significantly between men and women, we carried out stepwise multiple regression with insulin resistance, total cholesterol, HDL cholesterol, LDL cholesterol and logTG as the dependent variables. Independent variables included the anthropometric index under consideration (BMI, waist circumference, WHR or percentage body fat), age, sex and the interaction term anthropometric index*sex (BMI*sex, waist circumference*sex, WHR*sex or percentage body fat*sex, respectively). With the exception of the association between BMI and insulin resistance, sex was a confounding variable or had a modifying effect on all associations studied. As such, the relationship between BMI and insulin resistance was studied for the entire group while univariate regression for other variables were stratified by gender for regression analysis.

BMI was a significant predictor of insulin resistance

($R^2 = 32.5\%$, $P \sim 0.001$). This relationship is depicted in Figure 1. To confirm our impression that the slope of the regression line appeared to increase at higher BMI, we compared pair-wise slopes above and below a BMI of 26 kg/m². These were significantly different ($P \sim 0.001$).

Tables II and III show the results of univariate linear regression analyses for men and women. It can be seen that all four anthropometric indices studied were significantly correlated with insulin resistance. However, BMI and waist circumference were the most significantly correlated with insulin resistance with the BMI explaining slightly more of the variation in insulin resistance in women. In contrast, the waist circumference showed the greatest correlation in men. The R^2 values

were very close for both measures of obesity. Waist hip ratio was the best predictor of fasting triglyceride in both men and women. It showed a significant inverse correlation with HDL cholesterol in women. In men, there was a trend towards an inverse correlation but this did not reach statistical significance ($P = 0.094$). None of the measures of obesity or fat distribution correlated with total or LDL cholesterol.

Percentage body fat by BIA correlated positively with fasting triglyceride in men and inversely with HDL cholesterol in women. However, the R^2 values were small and did not offer any advantage over anthropometry.

Table IV shows the Pearson correlation between the

TABLE I: ANTHROPOMETRIC DATA, BLOOD PRESSURE, BLOOD LIPIDS AND INSULIN RESISTANCE FOR MALES AND FEMALES IN THE STUDY POPULATION

	Overall	Males	Females	<i>P</i> for difference between sexes
Weight (kg)*	68.6 (12.5)	74.2 (10.4)	62.69 (12.0)	<0.0001
Height (m)	165.4 (149.7 – 181.0)	170.45 (157.9 – 181.0)	157.2 (149.7 – 170.0)	<0.0001
Waist circumference (cm)*	82.6 (9.95)	86.6 (1.14)	78.3 (1.31)	<0.0001
Hip circumference (cm)	98 (84 – 135.5)	98.5 (87.0 – 118.0)	98.0 (84.0 – 135.5)	0.88
Body mass index (kg/m ²)	25.0 (18.6 – 45.1)	25.7 (19.68 – 34.92)	24.2 (18.67 – 45.11)	0.30
Waist-hip ratio*	0.83 (0.07)	0.88 (0.05)	0.79 (0.05)	<0.0001
Lean body mass (kg)	49.0 (32.0 – 71.0)	55.9 (42.3 – 71.04)	39.6 (32.0 – 61.7)	<0.0001
Fat mass (kg)	19.5 (4.5 – 52.7)	16.7 (4.5 – 39.0)	20.4 (10.4 – 52.7)	0.0016
Percentage body fat (BIA)*	28.9 (8.26)	23.4 (5.4)	34.7 (6.7)	<0.0001
Total cholesterol (mmol/l)	5.84 (4.36 – 10.35)	5.99 (4.74 – 10.35)	5.78 (4.36 – 8.72)	0.13
HDL cholesterol (mmol/l)	1.27 (0.7 – 2.29)	1.11 (0.70 – 2.29)	1.38 (0.78 – 2.20)	<0.0001
LDL cholesterol (mmol/l)	3.74 (2.38 – 6.83)	3.86 (2.38 – 6.44)	3.6 (2.58 – 6.83)	0.19
Triglyceride (mmol/l)*	1.83 (1.02)	2.25 (1.12)	1.38 (0.67)	<0.0001
Fasting insulin	8.2 (0.5 – 37.4)	8.9 (0.5 – 24.5)	7.4 (0.5 – 37.4)	0.10
HOMA insulin resistance	1.91 (0.1 – 10.1)	2.01 (0.10 – 5.88)	1.60 (0.10 – 10.14)	0.14
Mean arterial pressure (mmHg)	103.7 (81.3 – 127.3)	107.0 (82.7 – 127.3)	100.0 (81.3 – 122.0)	0.002

BIA: bioelectric impedance analysis

HDL: high density lipoprotein

LDL: low density lipoprotein

HOMA: homeostasis model assessment

* Arithmetic mean (standard deviation), compared using Student's *t*-test. All other values represent geometric mean (minimum-maximum) and are compared using the Mann-Whitney *U* method.

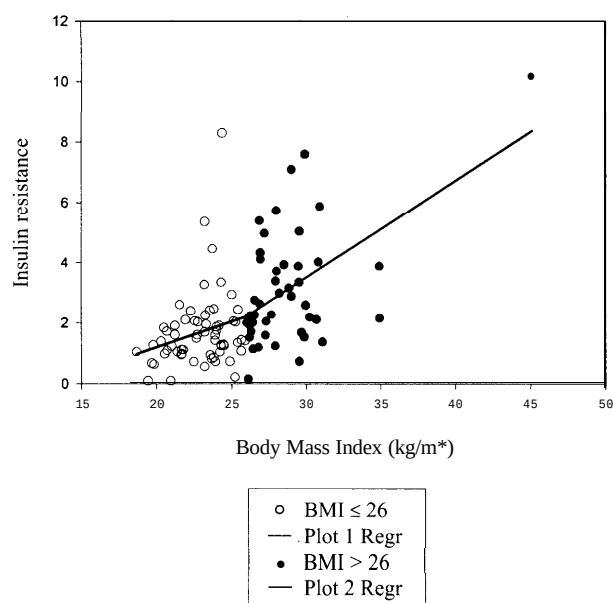


Fig. 1. Correlation between BMI and insulin resistance showing threshold effect (difference between slopes $P < 0.001$).

four anthropometric indices studied. All four indices correlated significantly with one another.

Discussion

The correlation between percentage body fat and coronary artery disease risk has not been extensively studied. Furthermore, the relevance of other anthropometric measures of obesity and body fat distribution has not been studied in a Chinese population. This study seeks to clarify some of these issues.

The authors appreciate that this is a small sample and may not represent the general population in Singapore. The study subjects were generally more obese and had higher lipid levels than the general population (1992 Singapore National Health Survey-unpublished). However, the study population does include a wide range of BMI and lipid levels and we believe that the data can be extrapolated to the general population. The results of this study should be validated in a larger cross sectional survey of the population.

TABLEII: REGRESSION OF INSULIN RESISTANCE, FASTING TRIGLYCERIDE AND HDL CHOLESTEROL AGAINST ANTHROPOMETRIC INDICES AND BIA FOR FEMALES

	BMI		Waist circumference		WHR		BIA	
	R ²	P	R ²	P	R ²	P	R ²	P
HOMA IR	35.9	co.00 1	32.6	co.00 1	17.1	0.002	10.0	0.02 1
TC	4.8	0.113	3.6	0.172	0.5	0.622	1.2	0.435
LDL	0.8	0.519	0.5	0.605	0.1	0.862	0.0	0.897
Log _e (TG)	2.0	0.885	3.7	0.167	9.8	0.023	2.6	0.25
HDL-C	15.1	0.003	17.0	0.002	24.4	co.00 1	10.3	0.019

BMI : body mass index

WHR : waist hip ratio

BIA : percentage body fat as measure by bioelectric impedance analysis

HOMA IR : insulin resistance (homeostasis model assessment)

TC : total cholesterol

TG : triglyceride

HDL-C : high density lipoprotein cholesterol

TABLEIII: REGRESSION OF INSULIN RESISTANCE, FASTING TRIGLYCERIDE AND HDL CHOLESTEROL AGAINST ANTHROPOMETRIC INDICES AND BIA FOR MALES

	BMI		Waist circumference		WHR		BIA	
	R ²	P	R ²	P	R ²	P	R ²	P
HOMA IR	27.0	co.00 1	28.8	co.00 1	22.8	co.00 1	12.6	0.008
TC	0.1	0.856	0.3	0.713	0.1	0.777	0.0	0.89
LDL	0.7	0.553	0.4	0.645	0.2	0.777	3.1	0.195
Log _e (TG)	0.0	0.885	2.2	0.279	8.7	0.027	7.9	0.037
HDL-C	1.5	0.378	3.4	0.175	5.1	0.094	0.9	0.494

BMI : body mass index

WHR : waist hip ratio

BIA : percentage body fat as measure by bioelectric impedance analysis

HOMA IR : insulin resistance (homeostasis model assessment)

TC : total cholesterol

TG : triglyceride

HDL-C : high density lipoprotein cholesterol

TABLEIV: PEARSON CORRELATION COEFFICIENT BETWEEN BMI, WAIST CIRCUMFERENCE, WAIST-HIP RATIO AND PERCENTAGE BODY FAT IN MEN AND WOMEN

Females	BMI	Waist circumference	WHR	Percentage body fat
BMI		0.912 ($P < 0.01$)	0.418 ($P < 0.01$)	0.708 ($P < 0.01$)
Waist circumference	0.912 ($P < 0.01$)		0.668 ($P < 0.01$)	0.656 ($P < 0.01$)
WHR	0.418 ($P < 0.01$)	0.668 ($P < 0.01$)		0.344 ($P = 0.012$)
Percentage body fat	0.708 ($P < 0.01$)	0.344 ($P < 0.001$)	0.344 ($P = 0.012$)	

* Correlation is significant at $P = 0.012$ (2-tailed)

Males	BMI	Waist circumference	WHR	Percentage body fat
BMI		0.929	0.635	0.655
Waist circumference	0.929		0.787	0.681
WHR	0.635	0.787		0.411*
Percentage body fat	0.655	0.681	0.411*	

* Correlation is significant at $P = 0.002$ (2-tailed)

Insulin resistance is a risk factor for coronary artery disease.¹⁴ Obesity has been associated with higher insulin resistance or reduced insulin sensitivity.^{4,15-19} All four indices of obesity were significantly correlated with insulin resistance and this is not surprising since they were also significantly correlated with each other (Table IV). BMI emerged as a good predictor of insulin resistance independent of gender. Few studies have correlated the degree of adiposity with insulin resistance but those that did showed conflicting results. Two showed a linear correlation with obesity¹⁷⁻¹⁹ while others found that the effect of increasing obesity on insulin resistance reached a plateau at a body fat content of 28% to 30%.¹⁵⁻¹⁸ In contrast, we found that the slope of the regression line increased with BMI above 26 kg/m². The other studies were smaller samples and the strength of our data is the number of subjects recruited.

It is important to note that the means of measuring insulin resistance differed in previous studies as they made use of insulin clamp techniques, which are likely to be a better reflection of insulin resistance than the model we have used in this study. However, homeostasis model assessment is a simple means of assessing insulin resistance and has been found to connote a 3-fold increased risk of developing diabetes mellitus in a prospective study.²⁰ Our results are thus in keeping with the finding that BMI is a better predictor of the development of diabetes mellitus than waist circumference and WHR.²¹ The same study found that the relative risk of developing diabetes mellitus increased sharply when the BMI was greater than 25 kg/m², even after correction for age, smoking status, family history and WHR. This is consistent with our finding that at high BMI, the increment of insulin resistance as measured by homeostasis model assessment is greater for the same increment in BMI. The relevance of this finding in the Singapore population is great given the rising prevalence of diabetes mellitus

and its attendant complications.

In contrast to the BMI, a measure of central adiposity, the WHR best predicted fasting triglyceride in both men and women. WHR was also inversely correlated with high density lipoprotein cholesterol levels in women. In men there was a trend towards a similar association but the analysis did not reach statistical significance. The relationships appeared to be linear with no level at which the correlation reached a plateau. Low levels of high density lipoprotein cholesterol have long been recognised as an important risk factor for coronary artery disease.²² Fasting triglyceride has more recently been recognised as an independent risk factor for coronary artery disease.²³ This may relate to an association with higher levels of atherogenic lipoproteins such as remnant particles and intermediate density lipoproteins. High levels also predict predominance of the small dense low density lipoprotein subfractions, which is associated with a 3- to 7-fold increased risk of coronary artery disease.^{24,25} Some authors have suggested that the waist circumference is a better indicator of dyslipidaemia than the WHR²⁶ but this was not demonstrated in our population.

In contrast to the findings in the Manitoba Heart Health Survey where BMI tended to be the stronger predictor for most metabolic variables studied,²⁷ our data showed that BMI was a better predictor of insulin resistance and WHR a better predictor of triglyceride and high density lipoprotein cholesterol. Thus, BMI and WHR seem to connote different aspects of coronary artery disease risk and this may explain the findings of previous authors that BMI and WHR are independent predictors of coronary artery disease.^{2,8}

BIA was disappointing as a tool for predicting coronary artery disease risk factors. This may relate to the fact that BIA was originally devised to measure total body water and has been modified to measure fat free

mass or lean body mass. It is therefore dependant on a number of variables other than adipose tissue mass which includes the state of hydration of the subject at the time of testing.

Conclusion

In a Chinese population, BMI correlated significantly with insulin resistance while WHR was the best marker for fasting triglyceride and high density lipoprotein cholesterol levels. As such, both these parameters are an important aspect of assessing overall cardiovascular risk in our population. BIA was not found to be useful in predicting levels of coronary artery disease risk factors.

REFERENCES

- Hubert H B, Feinleib M, McNamara P M, Castelli W P. 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.
- Larsson B, Svardssudd K, Welin L, Wilhelmsen L, Bjornthorp I, 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. *BMJ* 1984; 289:1401-4.
- Kissebah AH, Vydelingum N, Murray R, Evans D J, Hartz A J, Kalkhoff R K, et al. Relation of body fat distribution to metabolic complications of obesity. *J Clin Endocrinol Metab* 1984; 54:254-60.
- Lindahl B, Asplund K, Hallmans G. High serum insulin, insulin resistance and their associations with cardiovascular risk factors. The Northern Sweden MONICA Population Study. *J Inter Med* 1993; 234:263-70.
- Kissebah A H, Freedman D S, Peiris A N. Health risks of obesity. *Med Clin North Am* 1989; 73:111-38.
- Kahn H S, Austin H, Williamson D F, Arensberg D. Simple anthropometric indices associated with ischemic heart disease. *J Clin Epidemiol* 1996; 49:1017-24.
- Kannel W B, Cupples L A, Raaswami R, Stokes J, Kreger B E, Higgins M. Regional adiposity and risk of cardiovascular disease; The Framingham Study. *J Clin Epidemiol* 1991; 44:183-90.
- Lapidus L, Bengtsson C, Larsson B. Distribution of adipose tissue and risk of cardiovascular disease and death: a 12-year follow-up of participants in the population study of women in Gothenburg, Sweden. *BMJ* 1984; 289:1257-61.
- Karter A J, Mayer Davis E J, Selby J V, D'Agostino R B J, Haffner S M, Sholinsky P, et al. Insulin sensitivity and abdominal obesity in African-American, Hispanic, and non-Hispanic white men and women. The insulin resistance and atherosclerosis study. *Diabetes* 1996; 45:1547-55.
- Seidell J C, Cigolini M, Charzewska J, Ellsinger B M, Di Biase G, Bjornthorp P, et al. Indicators of fat distribution, serum lipids, and blood pressure in European women born in 1948. *Am J Epidemiol* 1989; 130:53-65.
- Campos H, Bailey S M, Gussak L S, Siles X, Orvados M, Schaefer E J. Relations of body habitus, fitness level, and cardiovascular risk factors including lipoproteins and apolipoproteins in a rural and urban Costa Rican population. *Arterioscler Thromb Vasc Biol* 1991; 11:1077-88.
- Lukaski H C, Johnson P E, Bolonchuk W W, Lykken G I. Assessment of fat-free mass using bioelectric impedance measurement of the human body. *Am J Clin Nutr* 1985; 41:410-4.
- Matthews D R, Hosker J P, Rudenski A S, Naylor B A, Treacher D F, Turner R C. Homeostasis model assessment: insulin resistance and B-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985; 28:412-9.
- Reaven G. Role of insulin resistance in human disease. *Diabetes* 1988; 37:1595-607.
- Abate N, Garg A, Peshock R M, Stray-Gundersen J, Grundy S M. Relationship of generalized and regional adiposity to insulin sensitivity in men. *J Clin Invest* 1995; 96:88-98.
- Bonadonna R C, Groop L, Kraemer N, Ferrenini E, Del Prato S, Defrozo R A. Obesity and insulin resistance in humans: a dose-response study. *Metabolism* 1990; 39:452-9.
- Yki-Jarvinen H, Koivisto V A. Effects of body composition on insulin sensitivity. *Diabetes* 1983; 32:965-9.
- Bogardus C, Lilloja S, Mott D M, Hollenbeck C, Reaven G. Relationship between degree of obesity and in vivo insulin action in man. *Am J Physiol* 1985; 248:286-91.
- Nagulesparan M, Savage P J, Unger R H, Bennet P H. A simplified method using somatostatin to assess in vivo insulin resistance over a range of obesity. *Diabetes* 1979; 28:980-3.
- Haffner S M, Kennedy E, Gonzalez C, Stern M P, Miettinen H. A prospective analysis of the HOMA model. The Mexico City Diabetes Study. *Diabetes Care* 1996; 19:1138-41.
- Chan J M, Rimm E B, Colditz G A, Stampfer M J, Willett W C. Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. *Diabetes Care* 1994; 17:961-9.
- Barter P J, Rye K A. High density lipoproteins and coronary heart disease. *Atherosclerosis* 1996; 121:1-12.
- Austin M A. Plasma triglyceride and coronary heart disease. *Arterioscler Thromb Vasc Biol* 1991; 11:2.
- Austin M A, Breslow J L, Hennekens C H, Buring J E, Willett W C, Krauss R M. Low density lipoprotein subclass and risk of myocardial infarction. *JAMA* 1988; 260:1917-21.
- Griffin B, Freeman D, Tait G, Thomson J, Caslake M, Packard C, et al. Role of plasma triglyceride in the regulation of plasma low density lipoprotein (LDL) subfractions: Relative contribution of small, dense LDL to coronary artery disease. *Atherosclerosis* 1994; 106:241-53.
- Pouliot M C, Despres J P, Lemieux S, Moorjani S, Bouchard C, Tremblay A, et al. Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. *Am J Cardiol* 1994; 73:460-8.
- Young T K, Gelskey DE. Is noncentral obesity metabolically benign? Implications for prevention from a population survey. *JAMA* 1995; 274:1939-41.