

Comparison of the Associations of Body Mass Index, Percentage Body Fat, Waist Circumference and Waist/Hip Ratio with Hypertension and Other Cardiovascular Risk Factors

Tevfik Sabuncu Ender Ankan Ertuğrul Taşan Hüsrev Hatemi

İstanbul University, Cerrahpaşa Medical Faculty, Department of Endocrinology and Metabolism, İstanbul, Turkey

Body mass index (BMI) is the most frequently used method in the assessment of obesity, and its increment is assumed to be an important risk factor for the development of atherosclerosis. Recently, however, several investigators have recommended the use of percentage body fat (PBF), waist circumference (WC) and waist/hip ratio (WHR) instead of BMI. In this study we evaluated the correlations of BMI, PBF, WC and WHR with each others and their effects on the many atherosclerotic risk factors in 169 females (age 42.4 ± 13.4 [mean \pm SD] years). We observed that WC and WHR have a stronger correlation with sBP ($r = 0.49$, $p = 0.000$ and $r = 0.51$, $p = 0.000$, respectively), dBP ($r = 0.48$, $p = 0.000$ and $r = 0.48$, $p = 0.000$, respectively), the presence of hypertension ($r = 0.38$, $p = 0.000$ and $r = 0.39$, $p = 0.000$, respectively), plasma fasting insulin level ($r = 0.48$, $p = 0.000$ and $r = 0.44$, $p = 0.000$, respectively), second hour insulin level ($r = 0.40$, $p = 0.001$ and $r = 0.35$, $p = 0.004$, respectively), first hour glucose level ($r = 0.34$, $p = 0.001$ and $r = 0.29$, $p = 0.005$, respectively), and second hour glucose level ($r = 0.28$, $p = 0.007$ and $r = 0.25$, $p = 0.01$, respectively)(during OGTT), triglyceride level ($r = 0.37$, $p = 0.000$ and $r = 0.40$, $p = 0.000$, respectively), and total cholesterol level ($r = 0.23$, $p = 0.01$ and $r = 0.31$, $p = 0.01$, respectively) than BMI and PBF. These results suggest that WC and WHR have a stronger correlation with cardiovascular risk factors than BMI and PBF. Although PBF also has an important association with some cardiovascular risk factors, it is not a better predictor of hypertension, NIDDM, or plasma glucose, insulin and lipid abnormalities than BMI.

KEY WORDS Body mass index, percentage body fat, waist circumference, waist to hip ratio, cardiovascular risk factors

Introduction

The prevalence of obesity has been increasing steadily in many developed countries, and it greatly increases the risk of many serious and morbid conditions, including coronary artery disease, diabetes mellitus, hypertension, dyslipidemia, and

some cancers (1). Body fat and its relation to other body components can be quantitated in many ways (2). Although BMI is the most common method used to assess obesity, direct measurement of body fat and its distribution may be more important. Fat distribution is usually measured by the waist to hip circumference ratio (WHR). A high WHR seems to be a proxy measurement for an excess of intra-abdominal fat. Exact determinations can only be performed directly using expensive equipment, such as computed tomography (3).

The importance of body weight, body mass and other measures of adiposity in the prediction of

Correspondence address:

Tevfik SABUNCU
Haznedar Mah. Yeşilirmak Sok, No: 17, D: 11
Güngören/İSTANBUL
Tel: 0 212 642 38 27
E-mail: tsabuncu@turk.net

cardiovascular disease has been the subject of long-standing debate.(4) Since adiposity is rarely measured directly, it is important to examine the validity of indices used as surrogates. Any index derived from height and weight cannot distinguish the contribution to body weight of fat tissue and that of muscle, bone, and water (5). Debate over the value of BMI for the estimation of body fat has recently led some investigators to recommend the use of new technologies for the direct measurement of body fat levels in epidemiological research (6) It has been suggested that the "gold standard" for the measurement of body fat is the body density from which fat and fat-free body mass can be calculated. The new technique of bioelectric impedance analysis may substantially improve the estimation of total body fat (7). Recently several studies have indicated that WHR is associated with hypertension, diabetes mellitus, and dyslipidemia more than measures of general obesity (8-13). And some studies have reported that WC demonstrated the greatest correlations with the risk variables, especially in men (14,15). Therefore we aimed to compare the associations between BMI, PBF, WHR and WC and several cardiovascular risk factors.

Material and Methods

This study was prospectively conducted in Cerrahpaşa medical faculty, endocrinology outpatient clinic and comprised 169 females aged 17-77 years. Individuals were excluded if they had a history of hepatic, renal and cardiac failure, or if endocrinological diseases, the use of a drug affecting lipid metabolisms, or pregnancy were present.

Body weight and height were measured in light clothing without shoes. The BMI was calculated as weight (in kilograms) divided by height (in meters) squared (kg/m^2). Waist circumference was measured at the narrowest diameter between the costal margin and the iliac crest, and the hip circumference was measured at the greatest diameter over the buttocks. Blood pressure (BP) was measured in the sitting position with a mercury sphygmomanometer after 10 min of rest. Two BP measurements were made (interval 1.5 min), and the mean was recorded. A subject was defined as having hypertension if systolic blood pressure (sBP) was > 140 mmHg or

diastolic blood pressure (dbp) was > 90 mmHg, or if the subject was receiving drug treatment for hypertension. A subject was defined as having NIDDM if fasting plasma glucose level (FPG) was > 140 mg/dl or second hour glucose level was > 200 mg/dl, or if the subject was receiving drug treatment for diabetes mellitus. There were no IDDM patients in the present study.

All BIA (Bioelectrical impedance analysis) measurements were performed using a single-frequency bioimpedance analyzer (model BIA 101/S: AKERN-RJL Systems, Detroit). A tetrapolar placement of electrodes was used, with current electrodes placed on the dorsal surfaces of the right hand and foot at the distal metacarpals and metatarsals, respectively, and the detector electrodes placed at the pisiform prominence of the right wrist and between the medial and lateral malleoli at the right ankle (16). BIA measurements were calculated using a computer program supplied by a commercial company. Percentage of body fat was used for the present study.

Blood samples were taken between 08:00 and 09:30 after a 12-h fast. A two hour OGTT with 75g glucose was performed, and baseline, first and second hour plasma glucose and insulin levels were measured. Plasma glucose was determined by the glucose oxidase method (Linear Chemicals, Badalona, Spain). Plasma insulin was determined from samples stored at -40°C by radioimmunoassay (RIA) using a commercial kit (DPC, Los Angeles, USA). Serum total cholesterol and high-density lipoprotein-cholesterol (HDL-C) concentrations were assayed using the Hitachi-717 autoanalyzer (DiaSys, Germany). Serum low-density lipoprotein cholesterol (LDL-C) concentration was calculated with Friedewald equation in patients with triglyceridemia < 400 mg/dl (17). Serum triglyceride concentration was determined using the Hitachi-717 autoanalyzer (BioSystem, Barcelona, Spain).

Statistics

Data analyses were performed with the SPSS for Windows release 6.1 program. Results were expressed as means \pm SD. Correlations between variables were tested by Spearman correlation coefficient. P values < 0.05 were considered statistically significant.

Results

Descriptive statistics for the study participants are presented in Table 1. There were 169 females, age 42.4 ± 13.4 years. In these subjects BMI was 28.4 ± 6.7 kg/m² (range:18 to 48), PBF was $29.3 \pm 6.2\%$ (range:15 to 45), WC was 87.9 ± 14.1 cm and WHR was 0.81 ± 0.08 (range: 0.6 to 1.08). The major independent variables in these analyses (BMI, PBF, WC and WHR) were inter-correlated. BMI was positively correlated with PBF ($r = 0.84$, $P = 0.000$), WC ($r = 0.89$, $P = 0.000$), and WHR ($r = 0.57$, $P = 0.000$), PBF was also positively correlated with WC ($r = 0.73$, $P = 0.000$), and WHR ($r = 0.45$, $P = 0.000$).

Table 1. Details of the study participants

Variable	Mean	SD
Age (years)	42.4	13.4
BMI (kg/m ²)	28.4	6.7
PBF (%)	29.3	6.2
WC (cm)	87.9	14.1
WHR	0.81	0.08
sBP (mmHg)	132.8	23.4
dBP (mmHg)	86.5	12.2
FPG (mg/dl)	88.3	29.1
First hour glucose (mg/dl)	153.9	53.5
Second hour glucose (mg/dl)	112.8	44.7
Fasting insulin (mg/dl)	15.2	16.8
First hour insulin (mg/dl)	101.7	89.9
Second hour insulin (mg/dl)	71.2	59.9
Triglyceride (mg/dl)	135.8	68.3
Total cholesterol (mg/dl)	225.2	43.4
LDL-C (mg/dl)	146.7	38.3
HDL-C(mg/dl)	51.6	11.5

Systolic BP, diastolic BP and the existence of hypertension were positively correlated with all BMI, PBF, WC and WHR, but this correlation was the most significant with WHR and WC. Fasting, first and second hours plasma insulin levels were also positively correlated with all BMI, PBF, WC and WHR, but this correlation was the most significant with WC. The existence of NIDDM was positively correlated only with WHR ($P = 0.01$), and marginally correlated with WC ($P = 0.06$). Although plasma first hour glucose level was positively correlated with all BMI, PBF, WC and WHR, second hour glucose level was positively correlated with BMI, WC and WHR (not with PBF), and fasting glucose level was

marginally correlated only with WHR ($P = 0.06$). Plasma triglyceride level was positively correlated with BMI, WC and WHR ($r = 0.25$, $r = 0.37$, $r = 0.40$, respectively). Although plasma total cholesterol level was significantly positively correlated with WC and WHR, LDL-C level was positively correlated with WC and marginally correlated with WHR, while HDL-C level did not significantly correlate with any indices. All results are shown in Table 2.

Table 2. Correlation coefficients of variables with BMI, PBF, WC and WHR

Variable	BMI	PBF	WC	WHR
sBP	0.37 ^c	0.35 ^c	0.49 ^c	0.51 ^c
dBP	0.40 ^c	0.29 ^b	0.48 ^c	0.48 ^c
Age	0.32 ^b	0.15	0.14	0.25 ^b
Hypertension	0.26 ^b	0.22 ^a	0.38 ^c	0.39 ^c
NIDDM	0.12	0.08	0.17	0.22 ^a
FPG	0.04	0.05	0.11	0.18
First hour glucose	0.26 ^a	0.20 ^a	0.34 ^b	0.29 ^b
Second hour glucose	0.22 ^a	0.08	0.28 ^b	0.25 ^a
Fasting insulin	0.43 ^c	0.38 ^c	0.48 ^c	0.44 ^c
First hour insulin	0.44 ^c	0.42 ^c	0.45 ^c	0.34 ^b
Second hour insulin	0.30 ^b	0.21	0.40 ^c	0.35 ^b
Triglyceride	0.25 ^a	0.13	0.37 ^c	0.40 ^c
Total cholesterol	0.14	0.11	0.23 ^a	0.31 ^b
LDL-C	0.11	0.02	0.20 ^a	0.18
HDL-C	-0.02	0.17	-0.04	0.001

a $P < 0.05$; b $P < 0.01$; c $P < 0.001$

Discussion

Obesity is becoming an increasingly important medical problem. It is associated with a greatly increased likelihood of diabetes, hypertension, hyperlipidemia, and cardiovascular disease (18). Although BMI is widely used as an epidemiological measure of obesity, some investigators have suggested that BMI is an imprecise measurement of fatness, compared with BIA, and therefore more direct measures of body fat should be used instead of it (18-25). However, we found that BMI was well correlated with percentage of body fat. Furthermore BMI had a stronger positive correlation with hypertension, fasting plasma triglyceride and cholesterol levels, and all plasma glucose and insulin levels during OGTT (except FPG level) compared to PBF. Results are shown in Table 2.

Several studies have suggested that besides the overall quantity of excess fat, the pattern of body-fat distribution may have important effects on the risk of CVD (26,27). The deposition of fat predominantly in the abdomen and upper body has frequently been found to be associated with abnormalities of blood pressure, glucose tolerance, and serum lipid levels (26). The WHR is considered as a measure of abdominal obesity and a surrogate measure for visceral fat deposition (19). Central obesity is generally regarded as a more important predictor of CVD than is generalized obesity (28,29). Aging, sex hormones, genetic, and dietary factors and physical inactivity may induce visceral fat accumulation. Visceral fat is characterized by its high lipogenic activity as well as its accelerated lipolytic activity. High levels of portal free fatty acids (FFAs) may eventually result in an enhancement of hepatic triglyceride synthesis, causing hyperlipidemia. High portal FFA levels would also induce insulin resistance, thereby causing glucose intolerance, hypertension, and finally atherosclerosis (30,31). In the present study, we also determined that WHR and WC have a stronger positive correlation with hypertension, diabetes mellitus, fasting plasma triglyceride, total cholesterol and LDL-C levels, and plasma glucose and insulin levels during OGTT compared to both BMI and PBF. There was no significant correlation between all indices of obesity and HDL-C, although some investigators reported that WHR is inversely related to HDL-C (32). This may be due to a genetic characteristic of the Turkish population, because it has been demonstrated that the Turkish population has lower HDL-C levels than those of other western populations (33).

Although some previous studies (34-36) have reported that BMI, WHR and PBF are increased with age, we found that both BMI and WHR were strongly correlated with age, but PBF and WC were weakly correlated with age.

It is reported that hypertension directly predisposes to all of the major atherosclerotic cardiovascular disease outcomes, including coronary artery disease, stroke, cardiac failure, and peripheral artery disease (37). The association between hypertension and obesity is well documented. Cross-sectional studies have shown that those persons who are 20% or

more overweight (\sim BMI > 27 kg/m²) have a greater risk of high blood pressure than lean persons (38,39). A possible cause is a decreased renal filtration surface, which may lead to renal sodium retention (40). Obesity also is known to lead to insulin resistance with consequent hyperinsulinemia, and insulin enhances tubular reabsorption of sodium. Enhanced catecholamine activity may also be involved. Plasma renin activity has also been reported to be elevated in obese persons with hypertension (20-22).

It has been recognized, for a long time, that obesity is a significant risk factor for NIDDM (41,42). The association between hyperinsulinemia and atherogenic risk factors has been well studied in both obese and lean individuals. The atherosclerosis Risk in Communities (ARIC) Study has demonstrated that individuals with hyperinsulinemia had more atherogenic levels of most risk factors than those with normoinsulinemia (23). In our population WHR and WC had a stronger correlation with NIDDM and all plasma insulin and glucose levels (except FPG) than other indices of obesity.

It has been reported that another important cardiovascular risk factor in obese persons is dyslipidemia (36,43). In several studies, HDL-C, a higher level of which has been clearly implicated in decreased risk for coronary heart disease, has been established to be lower in obese persons (44-47). Total and LDL-C, however, have been found in cross-sectional studies to be normal or elevated in obese compared with lean persons (48,49). Because HDL-C is low and LDL-C is normal to high, the ratio of LDL to HDL-C is generally high, leading to greater atherogenic risk. Elevated triglyceride levels have been described with weight gain (43,47,49). Increased free fatty acid availability from enhanced lipolytic activity and hyperinsulinemia enhances the formation of VLDL in the liver(50). Also, because lipoprotein lipase activity is decreased, a decreased clearance of triglycerides occurs (51,52). Some studies (53-55) have observed a stronger association between general obesity, rather than abdominal obesity, others (8,56) found, on the contrary, that fat distribution was more important than overall fatness. In our study, WHR and WC had a stronger

correlation with plasma triglyceride, total and LDL cholesterol levels than BMI and PBF.

These data suggest that percentage of body fat, compared with BMI, is not a better predictor of hypertension, NIDDM, or plasma glucose, insulin and lipid abnormalities. Both WHR and waist circumference have a stronger correlation with all these cardiovascular risk factors than overall fatness.

References

- Solomon CG, Manson JE. Obesity and mortality: a review of the epidemiologic data. *Am J Clin Nutr* **66** (suppl): 1044S-1050S, 1997.
- Bray GA. Overweight is risking fate. Definition, classification, prevalence, and risks. *Ann NY Acad Sci* **499**: 14-28, 1987.
- Ashwell M. Obesity in men and women. *Int J Obes Relat Metab Disord* **18** (suppl. 1): S1-S7, 1994.
- 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* **67**: 968-977, 1983.
- Manson JE, Stampfer MJ, Hennekens CH, Willett WC. Body weight and longevity. A reassessment. *JAMA* **257**: 353-358, 1987.
- Roubenoff R, Dallal GE, Wilson PW. Predicting body fatness: the body mass index vs estimation by bioelectrical impedance. *Am J Public Health* **85**: 726-728, 1995.
- Bray GA. Obesity: basic considerations and clinical approaches. *Dis Mon* **35**: 449-537, 1989.
- Seidell JC, Cigolini M, Charzewska J, Ellsinger BM, Di Biase G, Bjorntorp P, Hautvast JG, Contaldo F, Szostak V, Scuro LA. Indicators of fat distribution, serum lipids, and blood pressure in European women born in 1948--the European Fat Distribution Study. *Am J Epidemiol* **130**: 53-65, 1989.
- Kissebah AH, Vydelingum N, Murray R, Evans DJ, Hartz AJ, Kalkhoff RK, Adams PW. Relation of body fat distribution to metabolic complications of obesity. *J Clin Endocrinol Metab* **54**: 254-260, 1982.
- Soler JT, Folsom AR, Kaye SA, Prineas RJ. Associations of abdominal adiposity, fasting insulin, sex hormone binding globulin, and estrone with lipids and lipoproteins in post-menopausal women. *Atherosclerosis* **79**: 21-27, 1989.
- Haffner SM, Stern MP, Hazuda HP, Pugh J, Patterson JK. Do upper-body and centralized adiposity measure different aspects of regional body-fat distribution? Relationship to non-insulin-dependent diabetes mellitus, lipids, and lipoproteins. *Diabetes* **36**: 43-51, 1987.
- Ostlund RE Jr, Staten M, Kohrt WM, Schultz J, Malley M. The ratio of waist-to-hip circumference, plasma insulin level, and glucose intolerance as independent predictors of the HDL2 cholesterol level in older adults. *N Engl J Med* **322**: 229-234, 1990.
- Hartz AJ, Rupley DC, Rimm AA. The association of girth measurements with disease in 32,856 women. *Am J Epidemiol* **119**: 71-80, 1984.
- Reeder BA, Senthilselvan A, Despres JP, Angel A, Liu L, Wang H, Rabkin SW. The association of cardiovascular disease risk factors with abdominal obesity in Canada. Canadian Heart Health Surveys Research Group. *CMAJ* **157** (Suppl 1): S39-S45, 1997.
- Seidell JC, Cigolini M, Charzewska J, Ellsinger BM, Deslypere JP, Cruz A. Fat distribution in European men: a comparison of anthropometric measurements in relation to cardiovascular risk factors. *Int J Obes Relat Metab Disord* **16**: 17-22, 1992.
- Lukaski HC, Bolonchuk WW, Hall CB, Siders WA. Validation of tetrapolar bioelectrical impedance method to assess human body composition. *J Appl Physiol* **60**: 1327-1332, 1986.
- Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* **18**: 499-502, 1972.
- Jack DB. Fighting obesity the Franco-British way. *Lancet* **347**: 1756, 1996.
- Kvist H, Chowdhury B, Grangard U, Tylen U, Sjostrom L. Total and visceral adipose-tissue volumes derived from measurements with computed tomography in adult men and women: predictive equations. *Am J Clin Nut* **48**: 1351-1361, 1988.
- DeFronzo RA, Cooke CR, Andres R, Faloona GR, Davis PJ. The effect of insulin on renal handling of sodium, potassium, calcium, and phosphate in man. *J Clin Invest* **55**: 845-855, 1975.
- Sowers JR, Whitfield LA, Catania RA, Stern N, Tuck ML, Dornfeld L, Maxwell M. Role of the sympathetic nervous system in blood pressure maintenance in obesity. *J Clin Endocrinol Metab* **54**: 1181-1186, 1982.
- Dornfeld LP, Maxwell MH, Waks A, Tuck M. Mechanisms of hypertension in obesity. *Kidney Int* (Suppl. 22): S254-S258, 1987.
- Nabulsi AA, Folsom AR, Heiss G, Weir SS, Chambless LE, Watson RL, Eckfeldt JH. Fasting hyperinsulinemia and cardiovascular disease risk factors in nondiabetic adults: stronger associations in lean versus obese subjects. Atherosclerosis Risk in Communities Study Investigators. *Metabolism* **44**: 914-922, 1995.
- Landsberg L. Obesity and the insulin resistance syndrome. *Hypertens Res* **19** (suppl. 1): S51-S55, 1996.
- DeFronzo RA. Insulin resistance: a multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidaemia and atherosclerosis. *Neth J Med* **50**: 191-197, 1997.
- Kissebah AH, Vydelingum N, Murray R, Evans DJ, Hartz AJ, Kalkhoff RK, Adams PW. Relation of body fat distribution to metabolic complications of obesity. *J Clin Endocrinol Metab* **54**: 254-260, 1982.

27. Krotkiewski M, Bjorntorp P, Sjostrom L, Smith U. Impact of obesity on metabolism in men and women. Importance of regional adipose tissue distribution. *J Clin Invest* **72**: 1150-1162, 1983.
28. Lapidus L, Bengtsson C, Larsson B, Pennert K, Rybo E, Sjostrom L. Distribution of adipose tissue and risk of cardiovascular disease and death: a 12 year follow up of participants. *Br Med J (Clin Res Ed)* **289**: 1257-1261, 1984.
29. Bjorntorp P: Regional patterns of fat distribution. *Ann Intern Med* **103(6 Pt 2)**: 994-995, 1985.
30. Yamashita S, Nakamura T, Shimomura I, Nishida M, Yoshida S, Kotani K, Kameda-Takemura K, Tokunaga K, Matsuzawa Y. Insulin resistance and body fat distribution. *Diabetes Care* **19**: 287-291, 1996.
31. Tokunaga K, Matsuzawa Y. Obesity and insulin resistance syndrome. *Nippon Rinsho* **54**: 2679-2686, 1996.
32. Ward KD, Sparrow D, Landsberg L, Young JB, Vokonas PS, Weiss ST. The relationship of epinephrine excretion to serum lipid levels: the Normative Aging Study. *Metabolism* **43**: 509-513, 1994.
33. Mahley RW, Palaoglu KE, Atak Z, Dawson-Pepin J, Langlois AM, Cheung V, Onat H, Fulks P, Mahley LL, Vakar F, et al. Turkish Heart Study: lipids, lipoproteins, and apolipoproteins. *J Lipid Res* **36**: 839-859, 1995.
34. Gallagher D, Visser M, Sepulveda D, Pierson RN, Harris T, Heymsfield SB. How useful is body mass index for comparison of body fatness across age, sex, and ethnic groups? *Am J Epidemiol* **143**: 228-239, 1996.
35. Manson JE, Colditz GA, Stampfer MJ, Willett WC, Rosner B, Monson RR, Speizer FE, Hennekens CH. A prospective study of obesity and risk of coronary heart disease in women. *N Engl J Med* **322**: 882-889, 1990.
36. Despres JP. Dyslipidaemia and obesity. *Baillieres Clin Endocrinol Metab* **8**: 629-660, 1994.
37. Kannel WB. Cardioprotection and antihypertensive therapy: the key importance of addressing the associated coronary risk factors (the Framingham experience). *Am J Cardiol* **77**: 6B-11B, 1996.
38. Havlik RJ, Hubert HB, Fabsitz RR, Feinleib M. Weight and hypertension. *Ann Intern Med* **98(5 Pt 2)**: 855-859, 1983.
39. Stamler R, Stamler J, Riedlinger WF, Algera G, Roberts RH. Weight and blood pressure. Findings in hypertension screening of 1 million Americans. *JAMA* **240**: 1607-1610, 1978.
40. Brenner BM, Garcia DL, Anderson S. Glomeruli and blood pressure. Less of one, more the other? *Am J Hypertens* **1(4 Pt 1)**: 335-347, 1988.
41. Colditz GA, Willett WC, Stampfer MJ, Manson JE, Hennekens CH, Arky RA, Speizer FE: Weight as a risk factor for clinical diabetes in women. *Am J Epidemiol* **132**: 501-513, 1990.
42. Lundgren H, Bengtsson C, Blohme G, Lapidus L, Sjostrom L: Adiposity and adipose tissue distribution in relation to incidence of diabetes in women: results from a prospective population study in Gothenburg, Sweden. *Int J Obes* **13**: 413-423, 1989.
43. Rabkin SW, Chen Y, Leiter L, Liu L, Reeder BA. Risk factor correlates of body mass index. Canadian Heart Health Surveys Research Group. *CMAJ* **157(Suppl. 1)**: S26-S31, 1997.
44. Glueck CJ, Taylor HL, Jacobs D, Morrison JA, Beaglehole R, Williams OD. Plasma high-density lipoprotein cholesterol: association with measurements of body mass. The Lipid Research Clinics Program Prevalence Study. *Circulation* **62(4 Pt 2)**: IV-IV629, 1980.
45. Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR. High density lipoprotein as a protective factor against coronary heart disease. The Framingham Study. *Am J Med* **62**: 707-714, 1977.
46. Shelley E, Daly L, Kilcoyne D, Graham I. Obesity: a public health problem in Ireland? *Ir J Med Sci* **160 (Suppl. 9)**: 29-34, 1991.
47. Ferrannini E, Muscelli E, Stern MP, Haffner SM. Differential impact of insulin and obesity on cardiovascular risk factors in non-diabetic subjects. *Int J Obes Relat Metab Disord* **20**: 7-14, 1996.
48. Reeder BA, Angel A, Ledoux M, Rabkin SW, Young TK, Sweet LE: Obesity and its relation to cardiovascular disease risk factors in Canadian adults. Canadian Heart Health Surveys Research Group. *Med Assoc J* **146**: 2009-2019, 1992.
49. Lamon-Fava S, Wilson PW, Schaefer EJ. Impact of body mass index on coronary heart disease risk factors in men and women. The Framingham Offspring Study. *Arterioscler Thromb Vasc Biol* **16**: 1509-1515, 1996.
50. Bjorntorp P. "Portal" adipose tissue as a generator of risk factors for cardiovascular disease and diabetes. *Arteriosclerosis* **10**: 493-496, 1990.
51. Pi-Sunyer FX. Medical hazards of obesity. *Ann Intern Med* **119(7 Pt 2)**: 655-660, 1993.
52. Eckel RH, Yost TJ. Weight reduction increases adipose tissue lipoprotein lipase responsiveness in obese women. *J Clin Invest* **80**: 992-997, 1987.
53. Folsom AR, Prineas RJ, Kaye SA, Soler JT. Body fat distribution and self-reported prevalence of hypertension, heart attack, and other heart disease in older women. *Int J Epidemiol* **18**: 361-367, 1989.
54. Bonora E, Zenere M, Branzi P, Bagnani M, Maggiulli L, Tosi F, Travia D, Cacciatori V, Querena M, Moghetti P, et al. Influence of body fat and its regional localization on risk factors for atherosclerosis in young men. *Am J Epidemiol* **135**: 1271-1278, 1992.
55. Mykkanen L, Laakso M, Pyorala K. Association of obesity and distribution of obesity with glucose tolerance and cardiovascular risk factors in the elderly. *Int J Obes Relat Metab Disord* **16**: 695-704, 1992.
56. Reichley KB, Mueller WH, Hanis CL, Joos SK, Tulloch BR, Barton S, Schull WJ. Centralized obesity and cardiovascular disease risk in Mexican Americans. *Am J Epidemiol* **125**: 373-386, 1987.