Research Journal of Medical Sciences 4 (5): 298-304, 2010
ISSN: 1815-9346

Sex Differences in the Impact of Body Mass Index (BMI) and Waist/Hip (W/H) Ratio on Patients with Metabolic Risk Factors in Baghdad

Lujain Anwar Al-Khazrajy, Yossif Abdul Raheem and Yossra Khalaf Hanoon
Department of Community Medicine, Alkindy Medical College,
University of Baghdad, Baghdad, Iraq

Abstract: The aim of the study is to evaluate the impact of sex as variable in measuring waist/hip ratio as risk factor predictor in patients with metabolic disease. A longitudinal cross sectional study conducted on 234 patients with metabolic syndrome during 6 months duration, demographic data like Age and gender were recorded for each patient, other measures like waist circumference, hip circumference, height, weight, according to standards and body mass index and waist/hip ratio also calculated, blood tests including fasting blood sugar, lipid profile were also measured to the sample. Data were analyzed using descriptive statistics (frequencies and percentages) and analytic statistics (person correlation two ways (ANOVA) by SPSS, version 11. $p<0.05$ was considered statistically significant the mean age for male was 45.73(±7.83) years while for female was 46.92(±7.83) years. There was significant difference with W/H ratio 0.007 (-0.05 to -0.008) for both sexes (91.03%) of the total sample were having Diabetes mellitusand (63.25%) of the sample were having hypertension. Most of the participants (85.74%) had no physical activity. A positive correlation was obtained between W/H ratio and BMI, FBS, TG andHDL in male participants. the mean of W/H ratio in both gender as cross classified with Physical Exercise were the difference in mean is significantly associated WHR was significantly associated with the risk of incident CVD events. These simple measures of abdominal obesity should be incorporated into CVD risk assessments.

Key words: BMI (Body Mass Index), W/H ratio (Waist/Hip ratio), WC (Waist Circumference), HC (Hip Circumference) Metabolic syndrome, Iraq

INTRODUCTION

Metabolic syndrome is a combination of medical disorders that increase the risk of developing cardiovascular disease and diabetes. It affects one in five people and prevalence increases with age. The exact mechanisms of the complex pathways of metabolic syndrome are not yet completely known. The pathophysiology is extremely complex and has been only partially elucidated. Most patients are older, obese, sedentary and have a degree of insulin resistance. The most important factors in order are weight, genetics (Pollex and Hegele, 2006; Poulsen *et al*., 2001; Groop, 2000; Bouchard, 1995) aging and Sedentary lifestyle, i.e., low physical activity and excess caloric intake (Katzmarzyk *et al*., 2003).

Central adiposity is a key feature of the syndrome, reflecting the fact that the syndrome's prevalence is driven by the strong relationship between waist circumference and increasing adiposity. However, despite the importance of obesity, patients that are of normal weight may also be insulin-resistant and have the syndrome (Fauci, 2008). The metabolic syndrome has been associated with several obesity-related disorders including fatty liver disease, chronic renal disease, polycystic ovarian syndrome, obstructive sleep apnea and increase risk of cognitive decline and dementia (Grundy *et al*., 2004).

Physical inactivity is a predictor of CVD events and related mortality. Many components of the metabolic syndrome are associated with a sedentary lifestyle, including increased adipose tissue (predominantly central); reduced HDL cholesterol and a trend toward increased triglycerides, blood pressure and glucose in the genetically susceptible. Compared with individuals who watched television or videos or used their computer for >1 h daily, those that carried out these behaviors for >4 h daily have a twofold increased risk of the metabolic syndrome (Lara-Castro *et al*., 2007).

The metabolic syndrome affects 44% of the U.S. population older than age 50. A greater percentage of women older than age 50 have the syndrome than

Corresponding Author: Lujain Anwar Al-Khazrajy, Department of Community Medicine, Alkindy Medical College, University of Baghdad, Baghdad, Iraq

men. The age dependency of the syndrome's prevalence is seen in most populations around the world (Renaldi *et al*., 2009).

It is estimated that the large majority (~75%) of patients with type 2 diabetes or Impaired Glucose Tolerance (IGT) have the metabolic syndrome. The presence of the metabolic syndrome in these populations is associated with a higher prevalence of CVD than found in patients with type 2 diabetes or IGT without the syndrome (Lara-Castro *et al*., 2007) Hypoadiponectinemia has been shown to increase insulin resistance (Lara-Castro *et al*., 2007) and is considered to be a risk factor for developing metabolic syndrome (WHO, 2000).

The approximate prevalence of the metabolic syndrome in patients with Coronary Heart Disease (CHD) is 50% with a prevalence of 37% in patients with premature coronary artery disease (age 45), particularly in women. With appropriate cardiac rehabilitation and changes in lifestyle (e.g., nutrition, physical activity, weight reduction and in some cases, Drugs), the prevalence of the syndrome can be reduced (Lara-Castro *et al*., 2007).

Lipo dystrophic disorders in general are associated with the metabolic syndrome. Both genetic (e.g., Berardinelli-Seip congenital lip dystrophy, Dunnigan familial partial lipodystrophy) and acquired (e.g., HIV-related lipodystrophy in patients treated with highly active antiretroviral therapy) forms of lipodystrophy may give rise to severe insulin resistance and many of the metabolic syndrome's components (Lara-Castro *et al*., 2007).

Body Mass Index (BMI) is an index widely used to define obesity. The World Health Organization (WHO) sets a BMI range of 18.5-24.99 kg m^{-2} as normal (WHO, 2000). Although, Asians constitute a large proportion of the world's population, the majority of Asians, including the Japanese are not clearly obese according to the WHO classification, (Yoshiike *et al*., 1998; De Onis and Habicht, 1996) despite rapid westernization of lifestyles and a corresponding increase in metabolic risks. BMI does not always accurately indicate the degree of fatness (Smalley *et al*., 1990).

An increasing number of papers indicate that the degree of central fat distribution may be more closely tied to metabolic risks than BMI (Blair *et al*., 2001; Kaplan, 1989; Depres, 1991) Measurement of the degree of central fat distribution thus appears to be important for the early detection of subsequent health risks, even among those of normal weight. (Hsieh and Yoshinaga, 1995a; Ruderman *et al*., 1998; Hsieh *et al*., 2000).

The criteria for waist circumference proposed by WHO (midpoint between the lower border of the rib cage and the iliac crest) were based on studies of Caucasians, who generally have a higher BMI than many other ethnic groups (WHO, 2000) Also stating that obese individuals whose waist circumference (umbilical level) was 85 cm (men) or 90 cm (women) faced a higher risk of visceral fat accumulation (Japan Society for the Study of Obesity, 2000).

Several reports from Asia indicate that waist to height ratio (W/Ht) corresponds better to metabolic risk than BMI, waist circumference, waist to hip ratio or skin fold measures (Hsieh and Yoshinaga, 1995b). There are also reports that the cutoff value for W/Ht (0.5) appears to offer a simple but effective index for identifying overweight individuals and those of normal weight who face higher risks (Hsieh and Yoshinaga, 1995c; Lee *et al*., 1995; Hsieh and Yoshinaga, 1996; Lin *et al*., 2002; Victor *et al*., 2004).

MATERIALS AND METHODS

This is a longitudinal cross sectional study conducted on 234 patients with metabolic syndrome, for the period from the 15 of November to 30 of April, 2010. Participants for the study group were recruited from The Specialized Center for Endocrinology and Diabetes (at Al-Rusafa sector) and The National Center for Treatment and Research of Diabetes in Al-Mustanseria College of Medicine (at Al-Karkh sector) Baghdad. These two centers are the referral points for diabetic patients in Baghdad.

Patients included were diagnosed to have metabolic syndrome by specialists in both centers. Age and gender were recorded for each patient; height was calculated from the anthropometric measurements standing height measurement (CMS weighing equipment LTD, England).

The patient stood shoeless with the heels and back in contact with the vertical column of the scale. Weight measurement was done by digitalweightscale (Seca, Australia). Before each measurement the digital scale was adjusted to zero, the patient was asked to take-off his or her shoes and jackets before weighing and the weight was taken to the nearest fraction of kg (to the closest 0.1 kg).

Body Mass Index (BMI) was calculated as weight (kg) divided by height squared (2 m) and was used as the criteria for diagnosis of overweight and obesity. Participants were divided into 3 groups: normal weight (BMI <25 kg m^{-2}), overweight (25 kg m^{-2} ≤BMI <30 kg m^{-2}) and obese (BMI≥30 kg m^{-2}) (Victor *et al*., 2004).

Standards used to collect patients indices: Waist circumference: measured on a horizontal plane 1cm above the iliac crest. The cutoff point is: >94 cm (male), 80 cm (female) (Hsieh and Yoshinaga, 1995a). Hip circumference: measure the widest circumference of the buttocks at the area of the greater trochanters.

The cutoff point of W/H ratio: >0.9 (male), >0.85 (female). The cutoff point of W/Ht ratio is: 0.5. (Hsieh *et al*., 2000; Lin *et al*., 2002; IDF, 2006). The cutoff point of BMI is 25-34.9.

Diabetes mellitus definition: Raised fasting plasma glucose :(FPG)>100 mg dL^{-1} (5.6 mmol L^{-1}). Or previously diagnosed type 2 diabetes. If FPG >5.6 mmol L^{-1} or 100 mg dL^{-1}, OGTT Glucose tolerance test is strongly recommended but is not necessary to define presence of the Syndrome (Chobanian *et al*., 2003).

Dyslipedemia: Raised triglycerides: >150 mg dL^{-1} (1.7 mmol L^{-1}) or specific treatment for this lipid abnormality. Reduced HDL cholesterol: <40 mg dL^{-1} (1.03 mmol L^{-1}) in males, <50 mg dL^{-1} (1.29 mmol L^{-1}) in females or specific treatment for this lipid abnormality (Chobanian *et al*., 2003).

Blood pressure: Was measured and evaluated using a mercury sphygmomanometer and a standard clinical protocol according to the Joint National Committee (JNC-VII) report. After 10 min of resting, two readings of the systolic and diastolic BP separated by 5 min were averaged to the nearest 2 mmHg from the top of the mercury meniscus.

Systolic BP was recorded at the first appearance of sounds and diastolic BP at phase V at the disappearance of sounds. Hypertension was defined as systolic BP = 140 mmHg and/or diastolic BP = 90 mmHg. The validity of the weight scales and sphygmomanometers was censured by calibration prior to their use (Benner *et al*., 2008).

Statistical analysis: Data were analyzed using descriptive statistics (frequencies and percentages) and analytic statistics (person correlation two ways ANOVA) by SPSS, version 11.p<0.05 was considered statistically significant (Zaadstra *et al*., 1993).

RESULTS AND DISCUSSION

The studied sample consist of 234 with 125 male participants and 109 female, the mean age for male was 45.73 (±7.83) years while for female was 46.92 (±7.83) years. The BMI mean was nearly the same for both sexes and showed no significant difference but this difference was significant with W/H ratio 0.007 (-0.05 to -0.008) as shown in Table1.

About (91.03%) of the total sample were having Diabetes mellitus (DM) with nearly similar percentage of male and female (63.25%) of the sample were having hypertension (66.40%) were male and (59.63%) were female, as for hyperlipedemia nearly (83%)of the sample were sulfuring from elevated serum cholesterol level ,2/3 (74.36%) were having elevated triglycerides as shown in Table 2. Most of the participants (85.74%) have no physical activity, while (49.15%) of them had family history of hypertension and diabetes mellitus.

There was a positive correlation between BMI andage, WC, HC, W/H ratio, FBS, TG and HDL, while there was a negative correlation between BMI and cholesterol, diastolic andsystolic blood presser in male participants. Similar result was obtained with female participants in except for negative correlation of BMI with TG as shown in Table 3.

A positive correlation was obtained between W/H ratio and BMI, FBS, TG and HDL, while negative correlation was found with age, cholesterol, systolic and diastolic blood presser in male participants. Similar result was obtained for female participants except for the negative correlation of W/H ratio with FBS as shown in Table 4.

Regarding the mean of BMI in both gender as cross classified DM,HT, Cholestrol, TG, HDL, PE, FH(HT/DM). Two ways ANOVA only, reviled no statistical association as shown in Table 5.

Similar result was obtained regarding the mean of W/H ratio in both gender as cross classified with cholesterol, triglicerides, high density lipoprotein, family history hypertension/Diabetes Millitus) except for Physical activity were the difference in mean is significantly associated as shown in Table 6.

Relation of W/Hip ratio and mean age: The BMI mean was nearly the same for both sexes and showed no significant difference but this difference was significant with W/H ratio. In a sample of Dutch women, a lower WHR was associated with high fecundity and was a better predictor than other variables such as body mass index (Marlowe and Wetsman, 2001).

In a second study, without the weight categories and with frontal WHR ranging from 0.4-1.0, Hadza men preferred the highest ratios of 0.9 and 1.0 Wang *et al*. (2005). This may be because women with a larger waist appear heavier.

Table 1: Characteristics of the studied sample by sex and age

	Male (n = 125)		Female (n = 109)		Total (N = 234)		
Parameters	Mean	SD	Mean	SD	Mean	SD	p-value (95% CI)
Age (years)	45.73	7.83	48.29	8.59	46.92	8.28	0.02 (-4.67 to -0.44)
BMI (kg m^{-2})	29.45	4.55	30.17	4.97	29.78	4.75	0.25 (-1.95 to 0.51)
WC (CM)	110.63	11.75	117.32	12.70	114.02	12.20	0.0001 (-9.84 to -3.54)
HC (CM)	101.50	9.12	104.75	9.02	103.09	9.07	0.006 (-5.59 to -0.91)
W/H ratio (%)	1.09	0.08	1.12	0.09	1.11	0.08	0.007 (-0.05 to -0.008)
FBS	10.80	5.04	10.74	5.36	10.39	5.19	0.333 (-0.00-0.68)
TG	3.59	3.75	4.42	8.10	3.98	6.17	0.305 (-2.42-0.76)
Chole	3.86	1.57	3.78	1.48	3.86	1.57	0.69 (-0.31-0.47)
HDL	1.41	1.10	1.40	1.13	1.41	1.11	0.95 (-0.28-0.30)
SPB (mmHg)	140.58	17.73	139.03	17.80	139.85	17.74	0.50 (-3.03-6.15)
DPB (mmHg)	91.64	12.01	90.54	11.34	90.66	11.72	0.47 (-1.92-4.12)

BMI: Body Mass Index, WC: Waist Circumference, HC: Hip Circumference, W/H Waist Hip ratio, FBS: Fasting Blood Sugar, TG: Triglycerides, Chol. Cholesterol, HDL: High Density Lipoprotein, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure

Table 2: Associated condition (determinants) by sex

	Male (n = 125)		Female (n = 109)		Total (N = 234)		
Parameters	No	%	No	%	No	%	p-value
DM							
Yes	114	90.02	99	90.83	213	91.03	0.92
No	11	8.20	10	9.17	21	8.97	
HT							
yes	83	66.40	65	59.63	148	63.25	0.284
no	42	33.60	44	40.37	86	36.75	
↑Chol							
Yes	100	80.00	94	86.24	194	82.91	0.219
No	25	20.00	15	13.76	40	17.09	
↑TG							
Yes	90	72.00	84	77.06	174	74.36	0.207
No	35	28.00	25	22.94	60	25.64	
↑HDL							
Yes	69	55.20	64	58.72	133	56.84	0.588
No	56	44.80	45	41.28	101	43.16	
Phy Ex							
Yes	17	13.60	17	15.60	34	14.53	0.666
No	108	86.40	92	84.40	200	85.74	
FH (HT/DM)							
Yes	62	49.60	53	48.62	115	49.15	0.882
No	63	50.40	56	51.38	119	50.85	

DM: Diabetes Mellitus; HT: Hypertension, Chol. Cholesterol, TG: Triglycerides; HDL: High Density Lipoprotein, Physical activity, FH: Family History

Table 3: Corralation of BMI ratio

	BMI					
	Male		Female		Total	
Parameters	r	p	r	p	r	p
Age (years)	0.118	0.19	0.114	0.239	0.126	0.091
WC	0.707	0.00	0.653	0.00	0.681	0.000
HC	0.740	0.00	0.708	0.00	0.725	0.000
W/H ratio(%)	0.438	0.002	0.473	0.002	0.495	0.000
FBS	0.264	0.003	0.195	0.004	0.224	0.001
TG	0.146	0.104	-0.002	0.982	0.049	0.456
Chole	-0.140	0.119	-0.750	0.439	-0.102	0.119
HDL	0.106	0.238	0.052	0.588	0.079	0.228
SBP(mm Hg)	-0.056	0.538	-0.021	0.827	-0.042	0.523
DPB (mm Hg)	-0.040	0.654	-0.056	0.565	-0.054	0.411

WC: Waist Circumference, HC: Hip Circumference, W/H: Waist Hip ratio, FBS: Fasting Blood Sugar, TG: Triglycerides, Chol. Cholesterol, HDL: High Density Llipoprotein, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure

Table 4: Correlation of W/H R

	W/HR					
	Male		Female		Total	
Parameters	r	p	r	p	r	p
Age (year)	-0.090	0.318	-0.003	0.976	24.000	0.520
BMI	0.438	0.002	0.473	0.002	0.495	0.000
FBS	0.115	0.203	-0.112	0.246	0.001	0.983
TG	0.045	0.616	0.061	0.530	0.054	0.410
Chole	-0.089	0.321	-0.062	0.525	-0.074	0.260
HDL	0.113	0.208	0.120	0.214	0.117	0.075
SBP	-0.071	0.432	-0.015	0.876	-0.044	0.501
DBP	-0.093	0.310	-0.051	0.597	-0.074	0.258

BMI: Body Mass Index, FBS: Fasting Blood Sugar, TG: Triglycerides, Chol. Cholesterol, HDL: High Density Lipoprotein, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure

BMI and W/Hip ratio correlation: A positive correlation was obtained between W/H ratio and BMI, FBS, TG and HDL. Compared with Body Mass Index (BMI), anthropometric measures of abdominal obesity (e.g., Waist Circumference (WC) Waist to Hip Ratio (WHR), sagittal abdominal diameter) appear to be more strongly associated with metabolic risk factors (Despres and Lemieux, 2006) incident CVD events and death. The cardio-metabolic risk associated with abdominal obesity is attributed to the presence of Visceral Adipose Tissue (VAT) which promotes insulin resistance, dyslipidaemia and hypertension (Blair *et al*., 2001).

Physical activity: With Physical activity, the difference in mean was significantly associated. Two recent reviews have evaluated the relation between physical activity and CVD/cancer incidence and mortality (Haapanen-Niemi *et al*., 2000; Stevens *et al*., 2002). They conclude that individuals who report regular physical activity are less likely than sedentary individuals to die from coronary heart disease, stroke, CVD, certain cancers and all causes. Several studies have assessed the independent and combined effects of fattiness and

Table 5: Association of BMI (2 way ANOVA)

	BMI						
	Male (n = 125)		Female (n = 125)		Total (n = 125)		
Parameters	No	Mean (SD)	No	Mean (SD)	No	Mean	p values
DM							
Yes	114	30.48 (4.65)	99	30.27 (4.98)	213	30.44 (4.81)	0.678
No	11	28.42 (3.53)	10	29.15 (5.03)	21	28.72 (4.19)	
HT							
yas	83	29.38 (4.40)	65	30.43 (4.57)	148	29.90 (4.42)	0.687
No	42	29.58 (4.89)	44	29.77 (5.55)	86	29.67 (4.82)	
Chol							
Yes	100	31.98 (4.72)	94	31.34 (4.89)	194	31.66 (4.84)	0.724
No	25	28.28 (4.31)	15	29.98 (4.98)	40	29.13 (4.74)	
TG							
Yes	90	29.62 (4.59)	84	30.54 (4.68)	174	30.08 (4.60)	0.168
No	35	29.00 (4.48)	25	29.46 (4.72)	60	29.24 (4.58)	
HDL							
Yes	69	29.60 (4.19)	64	30.64 (4.62)	133	30.12 (4.48)	0.214
No	56	29.35 (5.16)	45	29.38 (4.34)	101	29.36 (4.86)	
PE							
Yes	17	27.89 (5.14)	17	29.93 (4.90)	34	28.91 (5.02)	0.189
No	108	30.54 (5.28)	92	31.34 (5.30)	200	30.94 (5.70)	
FH (HT/DM)							
Yes	62	29.78 (4.96)	53	30.21 (4.88)	115	29.99 (4.90)	0.386
No	63	29.12 (4.13)	56	30.09 (4.72)	119	29.60 (4.58)	

DM: Diabetes Mellitus; HT: Hypertension, Chol. Cholesterol; TG: Triglycerides HDL: High Density lipoprotein; Physical activity, FH: Family History

Table 6: Association of W/HR (2 way ANOVA)

	BMI						
	Male (n = 125)		Female (n = 125)		Total (n = 125)		
Parameters	No	Mean (SD)	No	Mean (SD)	No	Mean	p values
DM							
Yes	114	1.13 (0.07)	99	1.14 (0.08)	213	1.13 (0.08)	0.205
No	11	1.05 (0.12)	10	1.10 (0.07)	21	1.08 (0.09)	
HT							
yes	83	1.10 (0.07)	65	1.12 (0.08)	148	1.11 (0.07)	0.312
No	42	1.09 (0.08)	44	1.11 (0.08)	86	1.10 (0.08)	
Chol							
Yes	100	1.14 (0.07)	94	1.16 (0.07)	194	1.15 (0.07)	0.07
No	25	1.04 (0.09)	15	1.08 (0.08)	40	1.07 (0.08)	
TG							
yes	90	1.11 (0.07)	84	1.13 (0.07)	174	1.12 (0.07)	0.205
No	35	1.07 (0.08)	25	1.11 (0.08)	60	1.09 (0.08)	
HDL							
Yes	69	1.09 (0.08)	64	1.11 (0.08)	133	1.10 (0.08)	0.204
No	56	1.10 (0.07)	45	1.13 (0.07)	101	1.11 (0.07)	
PE							
Yes	17	0.98 (0.09)	17	1.02 (0.07)	34	1.01 (0.08)	0.030
No	108	1.20 (0.07)	92	1.22 (0.08)	200	1.21 (0.07)	
FH (HT/DM)							
Yes	62	1.11 (0.08)	53	1.13 (0.08)	115	1.12 (0.08)	0.126
No	63	1.08 (0.06)	56	1.11 (0.07)	119	1.10 (0.07)	

DM: Diabetes Mellitus; HT: Hypertension, Chol. Cholesterol; TG: Triglycerides; HDL: High Density Lipoprotein; Physical activity; FH: Family History

physical fitness on mortality (Wannamethee and Shaper, 2001; Haapanen-Niemi *et al*., 2000). Moderate or high level of cardio respiratory fitness may be protective against the excess mortality among overweight and obese individuals.

CONCLUSION

WHR was significantly associated with the risk of incident CVD events. These simple measures of abdominal obesity should be incorporated into CVD risk assessments in metabolic syndrome.

REFERENCES

Benner, J.S., L. Erhardt, M. Flammer, R.A. Moller and N. Rajicic *et al*., 2008. A novel programme to evaluate and communicate 10-year risk of CHD reduces predicted risk and improves patients modifiable risk factor profile. Int. J. Clin. Pract., 62: 1484-1498.

Blair, S.N., Y. Cheng and J.S. Holder, 2001. Is physical activity or physical fitness more important in defining health benefits. Med. Sci. Sports Exerc., 33: S379-S399.

Bouchard, G., 1995. Genetics and the metabolic syndrome. Int. J. Obes. Relat. Metab. Disord., 19: S52-S59.

Chobanian, A.V., G.L. Bakris, H.R. Black, W.C. Cushman and L.A. Green *et al.*, 2003. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. The JNC 7 Report. J. Am. Medical Assoc., 289: 2560-2572.

De Onis, M. and J.P. Habicht, 1996. Anthropometric reference data for international use: recommendations from a World Health Organization Expert Committee. Am. J. Clin. Nutr., 64: 650-658.

Depres, J.P., 1991. Lipoprotein metabolism in visceral obesity. Int. J. Obes., 15: 45-52.

Despres, J.P. and I. Lemieux, 2006. Abdominal obesity and the metabolic syndrome. Nature, 444: 881-887.

Fauci, A.S., 2008. Harrison's principles of internal medicine. 17th Edn., McGraw-Hill Medical Publishing, New York, pp: 2958.

Groop, L., 2000. Genetics of the metabolic syndrome. Br. J. Nutr., 83: S39-S48.

Grundy, S.M., B. Hansen, S.C. Jr. Smith, J.I. Cleeman and R.A. Kahn *et al.*, 2004. Clinical management of metabolic syndrome: Report of the American Heart Association/National Heart, Lung and Blood Institute/American Diabetes Association conference on scientific issues related to management. Circulation, 109: 551-556.

Haapanen-Niemi, N., S. Miilunpalo, M. Pasanen, I. Vuori, P. Oja and J. Malmberg *et al.*, 2000. Body mass index, physical inactivity and low level of physical fitness as determinants of all-cause and cardiovascular disease mortality- 16 years follow-up of middle-aged and elderly men and women. Int. J. Obes. Relat. Metab. Disord., 24: 1465-1474.

Hsieh, S.D. and H. Yoshinaga, 1995a. Is there any difference in coronary heart disease risk factors and prevalence of fatty liver in subjects with normal body mass index having different physiques. Tohoku. J. Exp. Med., 177: 223-231.

Hsieh, S.D. and H. Yoshinaga, 1995b. Abdominal fat distribution and coronary heart disease risk factors in men waist/height ratio as a simple and useful predictor. Int. J. Obes. Relat. Metab. Disord., 19: 585-589.

Hsieh, S.D. and H. Yoshinaga, 1995c. Waist/height ratio as a simple and useful predictor of coronary heart disease risk factors in women. Intern. Med., 34: 1147-1152.

Hsieh, S.D. and H. Yoshinaga, 1996. The relationships between various obesity indices and coronary heart risk factors. The comparisons of waist/height ratio to other obesity indices. J. Jpn. Human Dry. Dock., 11: 130-133.

Hsieh, S.D., H. Yoshinaga, T. Muto, Y. Sakurai and K. Kosaka, 2000. Health risks among Japanese men with moderate body mass index. Int. J. Obes., 24: 358-362.

IDF, 2006. The IDF consensus worldwide definition of the metabolic syndrome. International Diabete Federation. http://www.idf.org/webdata/docs/MetSyndrome_FINAL.pdf.

Japan Society for the Study of Obesity, 2000. New criteria for the diagnosis of obesity. Himankenkyu, 6: 18-28, (In Japanese).

Kaplan, N.M., 1989. The deadly quartet. Upper-body obesity, glucose intolerance, hypertriglyceridemia and hypertension. Arch. Intern. Med., 149: 1514-1520.

Katzmarzyk, P.T., A.S. Leon, J.H. Wilmore, J.S. Skinner, D.C. Rao, T. Rankinen and C. Bouchard, 2003. Targeting the metabolic syndrome with exercise: Evidence from the heritage family study. Med. Sci. Sports Exerc., 35: 1703-1709.

Lara-Castro, C., Y. Fu, B.H. Chung and W.T. Garvey, 2007. Adiponectin and the metabolic syndrome: mechanisms mediating risk for metabolic and cardiovascular disease. Curr. Opin. Lipidol., 18: 263-270.

Lee, J.S., K. Aoki, K. Kawakubo and A. Gunji, 1995. A study on indices of body fat distribution for screening for obesity. J. Occup. Health, 37: 9-18.

Lin, W.Y., L.T. Lee, C.Y. Chen, H. Lo and H.H. Hsia, 2002. Optimal cut-off values for obesity: Using simple anthropometric indices to predict cardiovascular risk factors in Taiwan. Int. J. Obes., 26: 1232-1238.

Marlowe, F. and A. Wetsman, 2001. Preferred waist-to-hip ratio and ecology. Personality Individual Differences, 30: 481-489.

Pollex, R.L. and R.A. Hegele, 2006. Genetic determinants of the metabolic syndrome. Nat. Clin. Pract. Cardiovasc. Med., 3: 482-489.

Poulsen, P., A. Vaag, K. Kyvik and H. Beck-Nielsen, 2001. Genetic versus environmental aetiology of the metabolic syndrome among male and female twins. Diabetologia, 44: 537-543.

Renaldi, O., B. Pramono, H. Sinorita, L.B. Purnomo, R.H. Asdie and A.H. Asdie, 2009. Hypoadiponectinemia: A risk factor for metabolic syndrome. Acta. Med. Indones., 41: 20-24.

Ruderman, N., D. Chisholm, X. Pi-Sunyer and S. Schneider, 1998. The metabolically obese, normal-weight individual revisited. Diabetes, 47: 699-713.

Smalley, K.J., A.N. Knerr, Z.V. Kendrick, J.A. Colliver and O.E. Owen, 1990. Reassessment of body mass indices. Am. J. Clin. Nutr., 52: 405-408.

Stevens, J., J. Cai, K.R. Evenson and R. Thomas *et al.*, 2002. Fitness and fatness as predictors of mortality from all causes and from cardiovascular disease in men and women in the lipid research clinics study. Am. J. Epidemiol., 156: 832-841.

Victor, R.G., R.W. Haley, D.L. Willett, R.M. Peshock and P.C. Vaeth*et al.*, 2004. The Dallas heart study: A population-based probability sample for the multidisciplinary study of ethnic differences in cardiovascular health. Am. J. Cardiol., 93: 1473-1480.

WHO, 2000. Obesity: Preventing and managing the global epidemic. Report of a WHO Consultation. WHO. Tech. Rep. Ser., 894: 1-253.

Wang, Y., E.B. Rimm, M.J. Stampfer, W.C. Willett and F.B. Hu, 2005. Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. Am. J. Clin. Nutr., 81: 555-563.

Wannamethee, S.G. and A.G. Shaper, 2001. Physical activity in the prevention of cardiovascular disease: An epidemiological perspective. Sports Med., 31: 101-114.

Yoshiike, Y., M. Matsumura, M.M. Zaman and M. Yamaguchi, 1998. Descriptive epidemiology of body mass index in Japanese adults in a representative sample from the National Nutrition Survey 1990-1994. Int. J. Obes. Relat. Metab. Disord., 22: 684-687.

Zaadstra, B.M., J.C. Seidell, P.A.H. van Noord, E.R. te Velde, J.D.F. Habbema, B. Vrieswijk and J. Karbaat, 1993. Fat and female fecundit Prospective study of effect of body fat distribution on conception rates. Br. Med. J., 306: 484-487.