Correlation of the Dysmetabolic Risk Factors with Different Anthropometric Measurements

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Abstract. Metabolic syndrome is a common disorder in Taiwan. For this study 431 subjects were randomly selected from visitors to the Department of Health Management. Blood pressure, blood glucose, lipid, uric acid levels and anthropometric measurements with immunoreactive insulin (IRI) and leptin levels were all correlated. We randomly selected 431 subjects who visited the Department of Health Management. Whole body three-dimensional (3-D) laser scanner scans were employed for the anthropometric measurements. The metabolic index (MI) was designed using anthropometric parameters. Of the 431 subjects, 50% had displayed a body mass index (BMI) equal to or exceeding 25 kg/m². Pearson correlation coefficient and multiple regression analysis revealed that MI constituted another index for correlating metabolic parameters by comparing MI with BMI and waist circumference to hip circumference ratio (WHR). Most data related to metabolic syndrome showed statistically significant differences between high and low IRI groups, comprising uric acid, total cholesterol, fasting plasma glucose, triglyceride, LDL, Chol/HDL ratio, and LDL/HDL ratio. Both IRI and leptin revealed statistical association with BMI, WHR, waist cross section area to hip cross section area ratio (WHAR), and MI in the study. Hypercholesterolemia appeared in 14.6% of the subjects. Elevated low-density lipoprotein (≥ 130 mg/dL) affected 36.9% of the subjects. In conclusion, MI calculated from 3-D body scanner correlated with many important metabolic risk factors and associated with clinical disorders like DM, hyperlipidemia, hyperuricemia and hypertension.

Key words: Body mass index, Waist/hip ratio, Metabolic index, Dyslipidemia, Leptin

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METABOLIC syndrome or syndrome X is defined as a clustering of cardiovascular disease risk factors comprising hyperlipidemia, hyperinsulinemia, hypertension, hyperuricemia, obesity, and glucose intolerance [1–5]. The clustering of these risk factors in syndrome X may reflect interrelations among risk variables or manifestation of a dominant underlying

common factor. The coexistence of cardiovascular risk variables related to syndrome X is well documented in epidemiological studies of adults and children [6–13]. Although the underlying pathologic mechanisms of the syndrome are not completely understood, the findings of prospective studies suggest that hyperinsulinemia or insulin resistance may play an important role in the development of syndrome X [5, 7, 9]. Obesity, with high waist circumference to hip circumference ratio (WHR), is deemed one of the important causes of syndrome X [10, 13].

The use, in recent work by the authors, of three-dimensional (3-D) scanning technology showed many

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advantages over the old system of anthropometric measurements [14] that employed tape measures, anthropometers (a special measuring ruler) [15, 16], and similar instruments. Laboratory data such as immuno-reactive insulin (IRI) and leptin were included in the analysis with anthropometric data to assess the detection possibility of metabolic syndrome in Taiwanese population.

Material and Methods

In this study, 431 subjects with a mean age of 52 ± 12 years were recruited from the Department of Health Management of Chang Gung Medical Center. There were 229 women (mean age, 51.0 ± 12.1 years) and 202 men (mean age, 55.0 ± 13.3 years). All subjects were Chinese residents of Taiwan and none had a history of systemic disease. Blood tests, body scanning, and evaluation of anthropometric measurements were performed on all subjects. To facilitate comparison with data from previous studies, a subject was considered obese when the body mass index (BMI) [weight (kg)/height (m)²] was greater than or equal to 30 kg/m^2 [17]. A subject was considered very obese when the BMI was greater than or equal to 45 kg/m^2 . Overweight was defined as BMI greater than 25. It is recognized that different cutoffs may be needed for certain groups [18]. Subjects were excluded from the study for obvious disease and body weight change greater than 10% during the last six months. The study was approved by the Institutional Review Board/Chang Gung Memorial Hospital.

Standardized protocols were used for all examinations, and the data were collected by trained staff. Subjects were instructed to fast for 12 to 14 hours prior to testing, and compliance regarding fasting was determined by interview on the morning of the examination. Measurements of height (to $\pm 0.1 \text{ cm}$) and weight (to $\pm 0.1 \text{ kg}$) were performed according to specified protocols [19, 20]. Blood pressure was taken on the right arm from seated, relaxed subjects. For both systolic and diastolic blood pressure, the average of three replicate mercury readings taken by two randomly assigned and trained nurses were used in analyses.

Blood pressure levels were classified according to 1999 World Health Organization-International Society of Hypertension guidelines [20]. Hypertension was defined as a systolic blood pressure (SBP) of 140 mmHg

or greater, and/or a diastolic blood pressure (DBP) of 90 mmHg or greater. High-normal blood pressure was defined as an SBP of 130–139 mmHg or a DBP of 85–89, while normal blood pressure was defined as an SBP of 120–129 mmHg and a DBP of 80–84 mmHg. Optimal blood pressure was SBP less than 120 mmHg and DBP of less than 80 mmHg. When SBP and DBP fell into different categories, the higher category was applied.

Venous blood was sampled after an overnight fast and centrifuged at 3,000 rpm for 30 minutes at 4°C. Cholesterol and triglyceride levels were measured using enzymatic procedures on an Abbott VP (Abbott Laboratories, North Chicago, IL) [21, 22]. Serum lipoprotein cholesterol levels were analyzed by a combination of heparin-calcium precipitation and agar-agarose gel electrophoresis procedures [22]. Plasma glucose levels were measured by an enzymatic method using the Beckman Instant Glucose Analyzer (Beckman Instruments, Palo Alto, CA). Human IRI and human leptin were assayed with Linco Research RIA kit (Linco Research, Inc., St. Charles, MO). Diabetes mellitus (DM) was defined as a fasting glucose greater than 125 mg/dL or a postprandial glucose greater than 200 mg/dL. Impaired glucose tolerance was defined as a fasting glucose between 110 and 125 mg/dL or a postprandial glucose between 140 mg/dL and 200 mg/dL. The formulas for the HOMA model for insulin resistance (HOMA IR) and β -cell function (HOMA β -cell) were as previously described [23].

The Whole Body 3-D Laser Scanner scans a cylindrical volume 1.9 meters high and 1.0 meter in diameter [14]. These dimensions accommodate the vast majority of human subjects. A platform structure supports the subject and provides alignment for the towers. The system is built to withstand shipping and repeated use without alignment or adjustment. A total of about 280 measurement results were calculated from the scanned data [24]. More than 30 measurements of the above results were new anthropometric factors which traditional measurements did not provide. The body scanner scans the human body from head to toe by laser in the co-horizontal plane around the whole body (Fig. 1). The computer processes 3-D data at the speed of 60 laser beams per second. For computation of a body height of 180 cm when the Gemini 10085 scanner is set up for a 4-mm vertical scanning resolution, it takes 7.5 seconds to scan the whole body. If the scanner is configured at a vertical scanning resolution of 2.5-mm, it

takes 12 seconds to scan the whole body. Subjects were also asked to provide demographic data such as age, ethnic group, gender, area of residence, education

level, and present occupation. Related hospital health and clinical records were obtained for each subject, if available.

Anthropometric measurements were performed to determine BMI and WHR. Data were coded into the computer. The results were correlated with data on IRI, leptin, blood pressure, blood glucose, lipid, and uric acid levels. Waist hip area ratio (WHAR) was defined as ratio of the waist cross section area and hip cross section area. The metabolic index (MI) was determined using the equation $MI = (\text{body weight} \times \text{waist area}) / (\text{body height}^2 \times \text{hip area})$. The MI combined the parameters of BMI and WHR. The above equation was proposed and revised after trial and error calculations to meet the best correlation with clinical biochemical data [14].

All data analyses were performed using SAS software [25]. Associations between individual pairs of risk variables were examined using partial Pearson's correlation coefficients and multiple regression analysis. All statistical analyses were carried out separately for men and women and for different age groups. Subjects taking antihypertensive medications were considered to have high blood pressure. Pearson's correlation coefficients were used to adjust for gender and age, between risk variables, and by age group.

Results

BMI was greater than or equal to 30 kg/m^2 in 7.4% of all subjects, including 7.4% of all males and 7.4% of all females. Among obese subjects, 2.1% were very obese. Among the 431 subjects, 216 (50.0%) had BMIs greater than 25 kg/m^2 and were therefore considered overweight. A higher percentage of males were overweight. Table 1 shows clinical and biochemical data including IRI, leptin in normal body weight, and overweight and MI obtained with the 3-D scanner data. In this analysis older subjects among 216 overweight patients presented with higher uric acid, higher total cholesterol, higher triglyceride, lower HDL level, higher VLDL, higher LDL, higher Chol/HDL ratio, higher LDL/HDL ratio, higher blood pressure, higher IRI, higher leptin, higher WHR, higher WHAR and higher MI. However, there was no difference in liver function, blood sugar, and TSH data.

For males, IRI was greatest in the patients with age over 80, leptin in 70 to 80 year-olds, while BMI, WHR,

Fig. 1. Anterior, lateral and posterior views of ideal, overweight, and obesity subjects in 3-D scanner. Obesity is BMI greater than or equal to 30 kg/m^2 . Overweight is defined as BMI greater than 25 and less than 30. Normal weight is between 17 and 25.

Table 1. The clinical and biochemistry factors of health clinics subjects by BMI

	BMI<25 kg/m ² (n = 216)	BMI≥25 kg/m ² (n = 216)	p value
Age (year)	52.8 ± 14.3	56.2 ± 10.9	0.0058
Total cholesterol (mg/dL)	196 ± 2.8	209 ± 2.7	0.0006
Triglyceride (mg/dL)	111 ± 4.9	157 ± 8.2	<0.0001
HDL (mg/dL)	57.5 ± 1.00	52.6 ± 0.94	0.0004
VLDL (mg/dL)	22.3 ± 1.01	30.3 ± 1.38	<0.0001
LDL (mg/dL)	116 ± 2.4	127 ± 2.4	0.0018
CHOL/HDL	3.61 ± 0.08	4.19 ± 0.08	<0.0001
LDL/HDL	2.17 ± 0.06	2.55 ± 0.06	<0.0001
*Glucose — AC (mg/dL)	106 ± 4.5	104 ± 2.4	0.6677
**Glucose — PC (mg/dL)	120 ± 6.6	115 ± 4.0	0.4333
Uric acid (mg/dL)	5.36 ± 0.09	6.16 ± 0.11	<0.0001
TSH (μIU/mL)	1.60 ± 0.09	3.19 ± 1.00	0.1151
Systolic blood pressure (mm-Hg)	117 ± 1.3	126 ± 1.3	<0.0001
Diastolic BP blood pressure (mm-Hg)	74.0 ± 0.80	79.1 ± 0.75	<0.0001
IRI (μIU/mL)	15.0 ± 0.48	19.8 ± 0.56	<0.0001
Leptin (ng/mL)	5.92 ± 0.35	9.75 ± 0.48	<0.0001
WHR	0.86 ± 0.01	0.93 ± 0.01	<0.0001
WHAR	0.76 ± 0.01	0.89 ± 0.01	<0.0001
MI	17.3 ± 0.25	24.9 ± 0.33	<0.0001

Metabolic syndrome index (MI) = (body weight × waist profile area)/(body height² × hip profile area)

*: Glucose — AC: fasting blood glucose

**Glucose — PC: 2 hours post-prandial blood glucose

Fig. 2. Correlation of immunoreactive insulin to other metabolic factors.

and MI were greatest in the 80 age group. Among female patients, however, the highest leptin, IRI, MI, WHR and WHAR occurred in the 71 to 80 year olds. BMI and WHR are known important indicators of metabolic syndrome. In this study, MI was analyzed to determine correlations with BMI and WHR. Fig. 2 demonstrates the correlation of IRI with age, BMI, WHR, WHAR and the MI. All these findings were sta-

tistically significant. The same results were found for leptin and their anthropometric measurement except for the age factor (Fig. 3).

Total cholesterol was normal in 50.1% of subjects. Total cholesterol in the 200 to 244 mg/dL range appeared in 35.3% of subjects; 11% of subjects had total cholesterol greater than or equal to 245 mg/dL. Percentage of men and women with total cholesterol

Fig. 3. Correlation of leptin to other metabolic factors.

Table 2. The correlation of serum immunoreactive insulin levels with other parameters of the metabolic syndrome

Factors	%	IRI		
		categories		
		abnormal	IRI<15.8 (μIU/mL)	IRI≥15.8 (μIU/mL)
Total cholesterol (≥200 mg/dL)		39.0%	59.9%	<0.0001
Triglyceride (>150 mg/dL)		15.5%	37.8%	<0.0001
HDL (<40 mg/dL)		9.0%	12.4%	0.2577
LDL (≥200 mg/dL)		33.0%	43.8%	0.0239
Cholesterol/HDL (1 × CHD risk factor)		14.0%	32.7%	<0.0001
LDL/HDL (1 × CHD risk factor)		8.5%	17.5%	0.0066
*Glucose — AC (≥105 mg/dL)		13.0%	27.7%	0.002
**Glucose — PC (≥140 mg/dL)		10.5%	14.3%	0.2424
Uric acid (≥8 mg/dL)		3.0%	10.6%	0.0023
Systolic blood pressure (≥140 mm-Hg)		13.7%	17.7%	0.2696
Diastolic blood pressure (≥90 mm-Hg)		15.3%	17.7%	0.5041

*: Glucose — AC: fasting blood glucose

**: Glucose — PC: 2 hours post-prandial blood glucose

greater or equal to 245 mg/dL were almost the same (15.3% vs. 14.0%). Elevated low-density lipoprotein (LDL>130 mg/dL) levels were found in 36.9% of subjects. Among them, 43.4% of the men and 31.8% of the women had LDL levels above the normal range. Low high-density lipoprotein (HDL<40 mg/dL) levels were found in 10.7% of subjects. Among them, 15.7% of men and 6.6% of women had HDL levels below the normal range. The percentage of subjects with abnormal total cholesterol, LDL and HDL is variable in different age groups. By age groups, subjects in the 51 to 60 year group had the highest abnormal levels of total cholesterol, with highest LDL among the 71 to 80 year

olds and lowest HDL among the 61 to 70 year olds. LDL levels were more markedly abnormal than total cholesterol and HDL levels for all age groups.

Table 2 reveals the different laboratory data for groups by fasting serum IRI levels. Most data related to metabolic syndrome show statistically significant difference between high and low IRI groups, including uric acid, total cholesterol, fasting plasma glucose, triglyceride, LDL, Chol/HDL ratio, and LDL/HDL ratio. Table 3 shows correlations of uric acid, total cholesterol, triglyceride, blood sugars, HDL, VLDL, LDL and ratio of total cholesterol with HDL, LDL/HDL ratio blood pressure, IRI, together with leptin levels with

Table 3. Correlation of blood pressure, glucose, TSH, uric acid, total cholesterol, triglyceride, HDL, VLDL, LDL and ratio of total cholesterol with HDL with other parameters including MI, BMI, WHR and WHAR

		MI	BMI	WHR	WHAR
Total cholesterol (mg/dL)	r	0.1249	0.1284	0.1088	0.1044
	<i>p</i> value	***	***	***	***
Triglyceride (mg/dL)	r	0.3053	0.2651	0.1253	0.2672
	<i>p</i> value	*	*	***	*
HDL (mg/dL)	r	-0.3196	-0.2614	-0.1881	-0.2965
	<i>p</i> value	*	*	**	*
VLDL (mg/dL)	r	0.3387	0.2713	0.2176	0.3119
	<i>p</i> value	*	*	*	*
LDL (mg/dL)	r	0.1026	0.1176	0.0924	0.0831
	<i>p</i> value	***	***	<i>ns</i>	<i>ns</i>
Cholesterol/HDL	r	0.3471	0.2923	0.2290	0.3181
	<i>p</i> value	*	*	*	*
LDL/HDL	r	0.2598	0.2266	0.1790	0.2409
	<i>p</i> value	*	*	**	*
#Glucose — AC (mg/dL)	r	0.1565	0.0499	0.1254	0.2102
	<i>p</i> value	***	<i>ns</i>	***	*
##Glucose — PC (mg/dL)	r	0.1730	0.0359	0.1523	0.2496
	<i>p</i> value	**	<i>ns</i>	***	*
Uric acid (mg/dL)	r	0.3436	0.3531	0.1178	0.2438
	<i>p</i> value	*	*	***	*
TSH (μIU/mL)	r	0.0160	0.0343	0.0331	0.0033
	<i>p</i> value	<i>ns</i>	<i>ns</i>	<i>ns</i>	<i>ns</i>
Systolic blood pressure (mm-Hg)	r	0.3576	0.2749	0.2444	0.3422
	<i>p</i> value	*	*	*	*
Diastolic blood pressure (mm-Hg)	r	0.2969	0.2805	0.1658	0.2435
	<i>p</i> value	*	*	**	*
IRI (μIU/mL)	r	0.0683	0.0811	0.0193	0.0407
	<i>p</i> value	<i>ns</i>	<i>ns</i>	<i>ns</i>	<i>ns</i>
Leptin (ng/mL)	r	0.3394	0.4081	0.1669	0.1955
	<i>p</i> value	*	*	**	*

#: Glucose — AC: fasting blood glucose; ##: Glucose — PC: 2 hours post-prandial blood glucose

*: $p < 0.0001$; **: $p < 0.001$; ***: $p < 0.05$; *ns*: no significant statistical difference

other parameters including BMI, WHR, WHAR, and MI. The results showed that leptin was better correlated with anthropometric measurement and MI than with IRI.

The study showed that the total cholesterol level was highest in the 51 to 60 year olds which differed from triglyceride levels in the 61 to 70 year old group. The mean fasting glucose level was less than 105 mg/dL and the postprandial sugar less than 140 mg/dl in 77.5% of the 431 subjects, ranging from 105 to 126 mg/dL in 10.9% but greater than 126 mg/dL in 9.3%. A higher percentage of men than women had diabetes mellitus (DM) (fasting glucose > 125 mg/dL; 11.3% vs 7.5%). Postprandial plasma glucose was less than 140 mg/dL in 87.5% of the 431 subjects, between 140 and 200 mg/dL in 4.6%, and greater than or equal to 200 mg/dL in 7.9%. A higher percentage of men

than women had postprandial glucose greater than 200 mg/dL (8.4% vs 7.5%). Among 431 subjects, 56 (13.0%) subjects meet the criteria of DM. Table 4 demonstrates multiple regression analysis of the MI, BMI, WHR, and WHAR. In the analyses, MI associated positively with uric acid, postprandial sugar, VLDL, systolic blood pressure, IRI, and leptin but associated negatively with HDL. WHAR seemed to be more closely associated with metabolic risk factors better than WHR.

Table 5 shows percentages of subjects diagnosed with parameters of metabolic syndrome at different ages, BMI, anthropometric measurements, HOMA insulin resistance, beta cell function index, and leptin levels. By contrast data analysis of the HOMA insulin resistance index is a better predictor than leptin levels for metabolic syndrome. To further investigate the

Table 4. Blood pressure, glucose, TSH, uric acid, total cholesterol, triglyceride, HDL, VLDL, LDL and ratio of total cholesterol with HDL compared with other parameters including MI, BMI, WHR and WHAR by multiple regression

		MI	BMI	WHR	WHAR
Total cholesterol (mg/dL)	R-square	0.6291	0.6170	0.3529	0.5503
	t	2.1645	0.0803	-0.0182	0.0710
	p value	***	ns	ns	ns
Triglyceride (mg/dL)	t	0.0437	0.0369	-0.0588	0.0582
	p value	ns	ns	ns	ns
HDL (mg/dL)	t	-5.6051	-2.0083	-0.0507	-4.4493
	p value	*	***	ns	*
VLDL (mg/dL)	t	0.0600	0.0252	0.0399	0.0872
	p value	ns	ns	ns	ns
LDL (mg/dL)	t	-0.0600	0.0473	-0.0239	0.0381
	p value	ns	ns	ns	ns
Cholesterol/HDL	t	0.0049	2.1940	3.5540	0.0698
	p value	ns	***	***	ns
LDL/HDL	t	-0.0602	-0.0214	-0.0384	0.0360
	p value	ns	ns	ns	ns
#Glucose — AC (mg/dL)	t	-0.0189	0.0093	0.0654	-0.0764
	p value	ns	ns	ns	ns
##Glucose — PC (mg/dL)	t	2.3966	-0.0115	0.0932	4.4083
	p value	***	ns	ns	*
Uric acid (mg/dL)	t	4.8560	5.5816	0.0090	2.6388
	p value	*	*	ns	***
TSH (μIU/mL)	t	-0.0097	0.0215	0.0068	-0.0062
	p value	ns	ns	ns	ns
Systolic blood pressure (mm-Hg)	t	5.7436	0.0380	3.9894	5.6266
	p value	*	ns	**	*
Diastolic blood pressure (mm-Hg)	t	0.0454	4.6195	-0.0306	-0.0151
	p value	ns	*	ns	ns
IRI (μIU/mL)	t	3.7898	3.0059	2.0205	3.2630
	p value	**	***	***	***
Leptin (ng/mL)	t	6.0643	7.9693	2.3264	2.6927
	p value	*	*	***	*

#: Glucose — AC: fasting blood glucose; ##: Glucose — PC: 2 hours post-prandial blood glucose; *: $p < 0.0001$; **: $p < 0.001$; ***: $p < 0.05$; ns: no significant statistical difference

Table 5. Parameters of dysmetabolic syndrome with correlation of anthropometric measurements, HOMA and health index

	Metabolic syndrome				
	DM	Hyperlipidemia	Hyperuricemia (uric acid >8 mg/dL)	Hypertension (140/90 mmHg)	Overweight (BMI>25)
Age	*	**	ns	ns	**
BMI	ns	**	*	**	*
Waist/Hip ratio	*	*	*	*	*
Waist/Hip area	*	*	*	*	*
Metabolic index	*	*	*	*	*
HOMA insulin resistance	*	*	*	ns	**
HOMA β cell function index	*	ns	ns	ns	**
Leptin	ns	ns	ns	ns	*
Waist circumference	**	*	*	*	*
Hip circumference	ns	*	**	ns	*
Waist width	ns	**	**	**	*
Waist profile	**	*	*	*	*

*: $p < 0.0001$; **: $p < 0.005$. ns: no significant statistical difference

roles of IRI and leptin relationship with DM, these data were compared in three groups of normal, IGT, and DM. Only IRI demonstrated higher statistical significance than normal subjects in IGT and DM patients.

Discussion

This study focused on the biochemical data, results of radioimmunoassay, and mechanical studies with 3-D dimensional detection of the body surface to investigate of metabolic syndrome. Obesity and body fat distribution is an important predictor of metabolic abnormalities including hyperinsulinemia, hypertension, hyperuricemia, glucose intolerance, DM and hyperlipidemia [26, 27]. An increment in leptin seems to be one of the important factors in the laboratory data and anthropometric measurements of metabolic syndrome. The increment of leptin was correlated significantly with BMI, WHR and the MI. Abdominal obesity has an important relationship with these chronic metabolic disorders [10, 13]. Although, computerized tomography is the most accurate method for the assessment of body fat, it is costly and time consuming. This investigation used a 3-D laser body scanner to determine distribution of body fat and then compared this to the calculated MI and traditional WHR and BMI. The results demonstrated that 3-D scanning provides another rapid and accurate way to obtain anthropometric measurement, while computerized tomography remains the best method for the assessment of body fat. Statistical analysis demonstrated that MI was another choice alongside WHR and BMI for predicting metabolic disorders. The MI calculated from the data of the 3-D body scanner was better than the health index in our previous studies (data not shown in the results) [14, 24].

Hyperinsulinemia was known as an important indicator of metabolic syndrome [2, 3, 28]. In this correlation study, subjects with higher serum insulin level correlated with higher ratios of abnormal laboratory data in uric acid, total cholesterol, fasting glucose, triglyceride, and LDL. Blood pressure and postprandial glucose showed higher abnormality rates in subjects with higher insulin levels. However, the differences were not statistically significant. Factors like age and gender may have interfered with these analyses. In contrast to correlation studies of IRI with laboratory data, IRI revealed poor correlations with BMI, WHR,

and MI. These anthropometric results had better correlations with biochemical data, blood pressure and leptin level. Insulin resistance and abdominal obesity are associated with low soluble leptin receptor concentration and low bound-free ratio of leptin independent of fat mass [29, 30]. More information is needed to determine that serum leptin level is one of the important factors in metabolic syndrome.

The standard BMI cut-off points for obesity in Europe and the USA are not appropriate for the Taiwanese population. However, until now there has been no long-term longitudinal reference defining obesity risk for Taiwanese. Further long-term longitudinal studies in this area are required to characterize population norms for obesity in Taiwan. In this study, 7.4% of subjects were obese and 2.1% were very obese. These rates are similar to results in a study from France and the UK but are lower than in studies from the USA (15%) [18]. The percentage of overweight subjects in this study was lower than in a previous study of Chinese women from Hong Kong [31]. Although BMI can be used as a marker for obesity, WHR is a better indicator of metabolic disorders. This study revealed that IRI in men increased with the age group, whereas leptin was highest in the 71 to 80 year olds. Among women, the highest IRI and leptin levels were among the 71 to 80 year olds. These results may have been due to estrogen effects or perhaps the over 80 sample was too small. MI and WHR values were significantly related to laboratory markers for metabolic disorders, findings which further demonstrate that MI is a useful tool in predicting metabolic disorders. The causes of the differences in fat distribution and metabolic abnormalities in both genders need further investigation. Long-term follow-up study is needed to determine the association of MI with the risk of cardiovascular diseases.

A high prevalence of dyslipidemia was found in this study, including hypercholesterolemia in 49.9% of subjects, hypertriglyceridemia in 26.7%, abnormally low HDL in 10.7%, and elevated LDL in 38.3%. Men had a higher prevalence in all metabolic abnormalities except for low LDL, which had higher prevalence among women, although these values were lower than in a recent Swiss study [32]. However, the present data were similar to findings from the study in the Seychelles [32]. Prevalence of hypertension and hyperlipidemia was much higher in the present study than in a previous one from Anqing, China [33]. The

Anqing study documented a higher prevalence of DM and impaired glucose tolerance than in a recent study on Kinmen, Taiwan, a rural island near China [34]. These data based on epidemiological results demonstrated the effects of differences in lifestyle and population between urban and rural areas. Although the prevalence of hyperuricemia in males in the present study was high (12.8%), this result was lower than in a recent study from Kinmen (25.8%) [35].

In conclusion, MI calculated from the data of 3-D body scanning was significantly associated with metabolic disorders including hyperinsulinemia, hyperglycemia, dyslipidemia, hyperuricemia, and hypertension. This study determined the relationships between 3-D body scanning data and metabolic disorders, and com-

pared these relationships with those obtained by conventional anthropometric measurements. Prospective long-term follow-up study is needed to determine the relationships of 3-D body scanning data and MI to cardiovascular diseases.

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References

1. Garaulet M, Perex-Llamas F, Fuente T, Zamora S, Tebar FJ (2000) Anthropometric, computed tomography and fat cell data in an obese population: relationship with insulin, leptin, tumor necrosis factor- α , sex hormone-binding globulin and sex hormones. *Eur J Endocrinol* 143: 657–666.
2. Modan M, Halkin H (1991) Hyperinsulinemia or increased sympathetic drive as links for obesity and hypertension. *Diabetes Care* 14: 470–487.
3. Reaven GM, Lithell H, Landsberg L (1996) Hypertension and associated metabolic abnormalities: the role of insulin resistance and the sympathoadrenal system. *N Eng J Med* 334: 374–381.
4. Bays H, Mandarino L, DeFronzo RA (2004) Mechanism of endocrine disease: role of the adipocyte, free fatty acids, and ectopic fat in pathogenesis of type 2 diabetes mellitus: peroxisomal proliferator-activated receptor agonists provide a rational therapeutic approach. *J Clin Endocrinol Metab* 89: 463–478.
5. Haffner SM, Valdez RA, Hazuda HP, Mitchell BD, Morales PA, Stern MP (1992) Prospective analysis of the insulin-resistance syndrome (Syndrome X). *Diabetes* 41: 715–722.
6. Bao W, Srinivasan SR, Wattifney WA, Berenson GS (1994) Persistence of multiple cardiovascular risk clustering related to Syndrome X from childhood to young adulthood: The Bogalusa Heart Study. *Arch Intern Med* 154: 1842–1847.
7. Chen W, Bao W, Begum S, Elkasabany A, Srinivasan SR, Berenson GS (2000) Age-related patterns of the clustering of cardiovascular risk variables of syndrome X from childhood to young adulthood in population made up of black and white subjects: the Bogalusa Heart Study. *Diabetes* 49: 1042–1048.
8. Jeppesen J, Hein HO, Suadicani P, Gyntelberg F (2000) High triglycerides and low HDL cholesterol and blood pressure and risk of ischemic heart disease. *Hypertension* 36: 226–232.
9. Haffner SM, Ferrannini E, Hazuda HP, Stern MP (1992) Clustering of cardiovascular risk factors in confirmed prehypertensive individuals. *Hypertension* 20: 38–45.
10. Srinivasan SR, Bao W, Berenson GS (1993) Coexistence of increased levels of adiposity, insulin, and blood pressure in a young adult cohort with elevated very-low-density lipoprotein cholesterol: The Bogalusa Heart Study. *Metabolism* 42: 170–176.
11. Meigs JB, D'Agostino RB, Wilson PW, Cupples LA, Nathan DM, Singer DE (1997) Risk variable clustering in the insulin resistance syndrome: The Framingham Offspring Study. *Diabetes* 46: 1594–1600.
12. Mykkanen L, Haffner SM, Ronnema T, Bergman RN, Laakso M (1997) Low insulin sensitivity is associated with clustering of cardiovascular disease risk factors. *Am J Epidemiol* 146: 315–321.
13. Bacha F, Saad R, Gungor Janosky J, Arslanian SA (2003) Obesity, regional fat distribution, and syndrome X in obese black versus white adolescents: race differential in diabetogenic and atherogenic risk factors. *J Clin Endocrinol Metab* 88: 2534–2540.
14. Lin JD, Chiou WK, Weng HF, Tsai YH, Liu TH (2002) Comparison of three-dimensional anthropometric body surface scanning with waist hip ratio and body mass index in correlation of metabolic risk factors. *J Clin Epidemiol* 55: 757–766.
15. Coombes AM, Moss JP, Linney AD, Richards R, James DR (1991) A mathematical method for the comparison of 3D changes in the facial surface. *Eur J*

- Orthodontic* 13: 95–110.
16. Jones PRM, Baker AJ, Hardy CJ, Mowat AP (1994) The measurement of body surface area in children with liver disease by a novel 3D body scanning device. *Eur J Appl Physiol* 68: 514–518.
 17. Pi-Sunyer XF (1998) Clinical guidelines on the identification, evaluation and treatment of overweight and obesity in adults -the evidence report. *Obes Res* 6: 51S–210S.
 18. Laurier D, Guiguet M, Chau NP, Wells JA, Valleron AJ (1992) Prevalence of obesity: a comparative survey in France, the United Kingdom and the United States. *Int J Obes Relat Metab Disord* 16: 565–572.
 19. Berenson GS, McMahan CA, Voors AW (1980) Cardiovascular risk factors in children: the early natural history of atherosclerosis and essential hypertension. New York, NY: Oxford University.
 20. Guidelines Subcommittee (1999) World Health Organization-International Society of Hypertension guidelines for the management of hypertension. *J Hypertension* 17: 151–183.
 21. Allain CC, Poon LS, Chan CS, Richmond W, Fu PC (1974) Enzymatic determination of total serum cholesterol. *Clin Chem* 20: 470–475.
 22. Bucolo G, David H (1973) Quantitative determination of serum triglycerides by the use of enzymes. *Clin Chem* 19: 476–482.
 23. Haffner SM, Miettinen H, Stern MP (1997) The homeostasis model in the San Antonio Heart Study. *Diabetes Care* 20: 1087–1092.
 24. Liu TH, Ciou WK, Lin JD, Yu CY (2001) Implementation of whole body scanner for determining somatotype index at Chang Gung Memorial Hospital. *Chang Gung Med J* 24: 697–707.
 25. SAS Institute, Inc. (1990) SAS/STAT user's guide, version 6.4th ed. Cary, NC: SAS Institute.
 26. Kissebah A, Vydelingum N, Murray R, Evans DJ, Hartz AJ, Kalkhoff PK, Adams PW (1982) Relationship of body fat distribution to metabolic complications of obesity. *J Clin Endocrinol Metab* 54: 254–260.
 27. Larsson B, Svärdsudd K, Welin L, Wilhelmsen L, Björntorp P, Tibblin G (1984) Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and health: 13 year follow up of participants in the Study of Men Born in 1913. *Br Med J* 288: 1401–1404.
 28. Reaven GM (2003) Insulin resistance/compensatory hyperinsulinemia, essential hypertension, and cardiovascular disease. *J Clin Endocrinol Metab* 88: 2399–2403.
 29. Sandhofer A, Laimer M, Ebenbichler CF, Kaser S, Paulweber B, Patsch JR (2003) Soluble leptin receptor and soluble receptor-bound fraction of leptin in the metabolic syndrome. *Obes Res* 11: 760–768.
 30. Goldstone AP, Brynes AE, Thomas EL, Bell JD, Frost G, Holland A, Ghatei MA, Bloom SR (2002) Resting metabolic rate, plasma leptin concentrations, leptin receptor expression, and adipose tissue measured by whole-body magnetic resonance imaging in women with Prader-Willi syndrome. *Am J Clin Nutr* 75: 468–475.
 31. He M, Li ETS, Kung AWC (1999) Dual-energy X-ray absorptiometry for body composition estimation in Chinese women. *Eur J Clin Nutr* 53: 933–937.
 32. Paccaud F, Schlüter-Fasmeyer V, Wietlisbach V, Bovet P (2000) Dyslipidemia and abdominal obesity: an assessment in three general populations. *J Clin Epidemiol* 53: 393–340.
 33. Hu FB, Wang B, Chen C, Jin Y, Yang J, Stampfer MJ, Xu X (2000) Body mass index and cardiovascular risk factors in rural Chinese. *Am J Epidemiol* 151: 88–97.
 34. Tsai ST, Li CL, Chen CH, Chou P (2000) Community-based epidemiological study of glucose tolerance in Kin-Chen, Kinmen: support for a new intermediate classification. *J Clin Epidemiol* 53: 505–510.
 35. Lin KC, Lin HY, Chou P (2000) The interaction between uric acid level and other risk factors on the development of gout among asymptomatic hyperuricemia men in a prospective study. *J Rheumatol* 27: 1501–1505.