Body Mass Index Is the Most Important Determining Factor for the Degree of Insulin Resistance in Non-obese Type 2 Diabetic Patients in Korea

Sang Ah Chang, Hee Seung Kim, Kun Ho Yoon, Seung Hyun Ko, Hyuk Sang Kwon, Sung Rae Kim, Won Chul Lee, Soon Jib Yoo, Hyun Sik Son, Bong Yun Cha, Kwang Woo Lee, Ho-Young Son, and Sung Koo Kang

With obesity, increased insulin secretion is needed to compensate for the additional demands and to maintain euglycemia. In contrast to Caucasians, the majority of type 2 diabetic patients belong to the non-obese category in Korea. There appears to be an ethnic difference underlying the pathogenesis in type 2 diabetes mellitus. However, there is only limited data on these subjects. The degree of insulin resistance in 267 Korean non-obese (body mass index [BMI] < 25 kg/m^2) ≥ patients with type 2 diabetes mellitus was analyzed, and the factors responsible for the insulin resistance were examined. The mean age and BMI of the patients were 50.8 ± 10.6 years and 22.6 ± 1.8 kg/m^2. Homeostasis model assessment-insulin resistance (HOMA-IR) ≥ 2.5 was defined as being insulin resistant according to our data (mean ± 1.5 SD of 1,917 normal subjects). There was no significant difference according to age, the duration of disease, and the glycosylated hemoglobin (HbA_{1c}) levels between the subjects with or without insulin resistance. The HOMA-IR values in the patients with insulin resistance and normal insulin sensitivity were 4.2 ± 1.4 and 1.5 ± 0.6, respectively. In the insulin-resistant group, the log-transformed triglyceride (TG) levels were higher and the high-density lipoprotein-cholesterol (HDL-C) levels were lower than those of the insulin-sensitive group (log-transformed TG: 5.2 ± .6 *v* 4.9 ± .7 and HDL-C: 1.13 ± 0.3 *v* 1.25 ± 0.3mmol/L). These differences were still observed after adjusting for BMI. The HOMA-IR value was independently predicted by BMI and HDL-C levels, which explained 7% and 3% in the variability of insulin resistance, respectively. However, the TG levels were not independently associated with the HOMA-IR. Logistic regression analysis showed that the significant factor associated with HOMA-IR was only BMI. These results suggest that the BMI is the most important determinant of insulin resistance, while TG and HDL-C levels might be good markers of insulin resistance in non-obese patients with type 2 diabetes mellitus in Korea.

TYPE 2 DIABETES mellitus occurs when there is inadequate insulin secretion to meet the insulin demands of the body.[1,2] With obesity, an increased β-cell mass is needed to compensate for the additional demands and to maintain euglycemia. In Korea, the majority of type 2 diabetic patients are non-obese, which is in contrast to Caucasians. There appears to be an ethnic difference underlying the pathogenesis in type 2 diabetes mellitus, because the degree of obesity is closely related with insulin resistance. It has been reported that there are racial differences in the degree of insulin resistance in type 2 diabetes. Haffner et al[3] recently reported that 92% of patients with type 2 diabetes mellitus were insulin resistant in the white population. However, a Japanese group showed that only 40% of the patients with type 2 diabetes mellitus might be subclassified into an insulin resistance group by using homeostasis model assessments. They also showed that the insulin resistance was independently associated with hypertriglyceridemia and the degree of obesity.[4,5]

In Korea, the prevalence of diabetes mellitus is 8% to 10%,[6] and over 95% of diabetic patients belong to the type 2 diabetes mellitus group. However, patients with a body mass index (BMI) greater than 25 kg/m^2 comprised only 35%.[7-9] This data suggests that the characteristics of Korean type 2 diabetic patients are quite different from those of western countries, but are similar to Japan and other Asian countries. However, there is only limited data regarding the insulin-sensitive and insulin-resistant variants ratio particularly in non-obese patients with type 2 diabetes mellitus and the factors that determine insulin resistance. In this report, 267 Korean non-obese (BMI < 25 kg/m^2) patients with type 2 diabetes mellitus were classified according to the degree of insulin resistance and the factors responsible for insulin resistance.

MATERIALS AND METHODS

Subjects

A total of 267 non-obese Korean type 2 diabetic patients who visited Kangnam St. Mary's hospital in Seoul, and St. Vincent's hospital in Suwon from November 1992 to December 1999 were enrolled in this study. Diabetes was diagnosed according to the American Diabetes Association (ADA) criteria.[10] The mean ± SD age of the patients was 50.8 ± 10.6 years (range, 30 to 79). The disease duration was 4.3 ± 6.2 years (range, 0 to 40 years). The BMI level was 22.6 ± 1.8 kg/m^2 (range, 17 to 24.9 kg/m^2)[11] (Table 1). Patients who were taking lipid-lowering drugs, such as statin or fibrate, were excluded. Patents who were taking antihypertensive agents that affected glucose tolerance and lipid metabolism (thiazide or beta blockers) were also excluded. None of the patients had significant renal, hepatic, or cardiovascular disease. All of the patients had been treated with diet and exercise only before the study. Most of the patients were followed up in the outpatient clinic of our hospital with relatively good glycemic control. Blood chemistry

From the Division of Endocrinology and Metabolism, Departments of Internal Medicine and Preventive MedicineColleges of Medicine and Nursing, The Catholic University of Korea, Seoul, Korea.

Submitted October 12, 2002; accepted June 27, 2003.

Supported by a grant from the Korea Health 21 R & D Project, Ministry of Health and Welfare (00-PJ3-PG6-GN07-001) and from the Basic Research Program of the Korea Science and Engineering Foundation (R04-2002-000-20024-0), Republic of Korea.

Address reprint requests to Ho-Young Son, MD, Division of Endocrinology and Metabolism, Department of Internal Medicine, The Catholic University of Korea, Kangnam St. Mary's Hospital, #505, Banpo-Dong, Seocho-Ku, Seoul, Korea, 137-701.

0026-0495/04/5302-0040$30.00/0

doi:10.1016/S0026-0495(03)00314-7

Table 1. Characteristics of Korean Non-obese Type 2 Diabetic Patients

Characteristics	Mean ± SD (range)
Age (yr)	50.8 ± 10.6 (30-79)
Duration of disease (yr)	4.3 ± 6.2 (0-40)
BMI (kg/m^2)	22.6 ± 1.8 (17-24.9)
HbA_{Ic} (4-5.5)* (%)	7.9 ± 2.1 (4.2-18.9)
Fasting glucose (mmol/L)	8.8 ± 2.9 (11-347)
Fasting insulin (pmol/L)	36.6 ± 27 (0.47-192)
HOMA-IR	2.1 ± 1.5 (.02-8.19)
HOMA–β cell	27.9 ± 46.2 (−420-196)

NOTE. n = 267.
*Reference value.

and serum insulin concentrations were determined on a regular basis. Data were also corrected from the patients who transferred from primary clinics for evaluation of glycemic control status.

Methods

Blood was drawn in the morning after a 12-hour fast. The plasma glucose levels were determined by the glucose oxidase method (Beckman Glucose Analyzer II, Beckman Instruments, Fullerton, CA) using venous blood. The serum insulin levels were measured using a radioimmunoassay kit (Dainabot, Tokyo, Japan). The glycosylated hemoglobin (HbA_{1c}) levels were measured by high-performance liquid chromatography (Variant II HbA_{Ic} analyzer, Bio-Rad, Montreal, Canada). The triglyceride (TG), total cholesterol, and high-density lipoprotein-cholesterol (HDL-C) were also measured using an automatic blood analyzer (Hitachi 736-40, Tokyo, Japan). The low-density lipoprotein-cholesterol (LDL-C) levels were calculated using the Friedewald formula.[12] The homeostasis model assessment of insulin resistance (HOMA-IR) was calculated using the following formula: fasting serum insulin (μU/mL) × fasting plasma glucose (mmol/L)/22.5.

For determining for insulin resistance by HOMA in the Korean population, we examined 1,901 normal subjects who had a normal range of fating blood glucose (3.6 to 6.4 mmol/L), blood pressure (<140/90 mm Hg), lipid levels (total cholesterol < 5.69 mol/L, TG 2.6 mmol/L), and renal function (creatinine <1.3 mg/dL) aged 25 to 80 years. The subjects visited Kangnam St. Mary's hospital for a general check up from March to September 1996. Their mean age was 44.2 years and the mean BMI was 23.3 ± 0.1 kg/m^2. The fasting insulin was measured using a radioimmunoassay kit (Dainabot) and blood glucose levels were measured by hexokinase method. The HOMA-IR value (mean ± SD) of 1,901 normal subjects in Korea was 1.3 ± 0.8. Therefore, values greater than 2.5 (mean ± 1.5 SD) were defined as being insulin resistant. The HOMA–β-cell function was calculated using the following formula: (20 × fasting serum insulin [μU/mL])/(fasting plasma glucose [mmol/L] − 3.5).

Statistical Analysis

The data are presented as a mean ± SD or a median (range). Statistical analyses were performed using SAS System, version 6.12 (SAS, Cary, NC). The means of the insulin- resistant and insulin-sensitive diabetes groups were compared with either Student's *t* test or a Wilcoxon rank sum test. The analysis of the covariance (ANCOVA) was used to compare the 2 groups after adjusting for BMI and fasting glucose. Pearson correlation coefficients between the HOMA-IR values and the measured variables were calculated, and a stepwise multiple regression analysis was then used to evaluate the independent association of these variables with the HOMA-IR. Logistic regression was

Table 2. General Characteristics and Clinical Profile of Insulin-Resistant and Insulin-Sensitive Diabetic Patients

	Insulin-Resistant (n = 63)	Insulin-Sensitive (n = 204)	t	*P*
Male	28 (19.4)	116 (80.6)		
Female	35 (28.5)	88 (71.5)		
Age (yr)	51.9 ± 10.7	50.4 ± 10.5	0.98	.33
Duration of disease (yr)	2 (0.1-23.0)	2 (0.1-40.0)		.57*
BMI (kg/m^2)	23.2 ± 1.4	22.4 ± 1.8	3.50	.0006
HOMA-IR	4.19 ± 1.35	1.46 ± 0.59	15.51	.0001
HOMA–β cell	46.1 ± 36.9	21.9 ± 47.5	4.19	.0001
HbA_{Ic} (%) (4-6)*	8.0 ± 2.1	7.9 ± 2.1	0.48	.63
Fasting glucose (mmol/L)	9.6 ± 2.9	8.6 ± 2.8	2.42	.016
Fasting insulin (pmol/IL)	55.2 (33-163.2)	24.6 (0.6-192.0)		.0001†
				.004
Triglycerides (log transformed)	5.2 ± 0.6	4.9 ± 0.7	2.84	0.01‡
				.003§
				.35
Total cholesterol (mmol/L)	4.89 ± 0.94	5.04 ± 1.10	0.94	
				.50§
				.08
LDL-cholesterol (mmol/L)	2.76 ± 1.0	3.03 ± 1.03	1.74	
				.13§
				.009
HDL-cholesterol (mmol/L)	1.13 ± 0.3	1.25 ± 0.3	2.62	.01‡
				.01§

NOTE. Insulin-resistant: HOMA-IR ≥ 2.5; insulin-sensitive: HOMA-IR < 2.5. Data are N (%)/mean ± SD/median (range).
*Reference value of HbA_{Ic}.
†Wilcoxon rank sum test.
‡P^{ANCOVA}: *P* value of ANCOVA (covariate = BMI).
§P^{ANCOVA}: *P* value of ANCOVA (covariate = fasting glucose).

Table 3. Correlation of HOMA-IR to Measure of Variables

	r (*P*)
BMI (kg/m^2)	0.25 (.0001)
Triglycerides	0.16 (.01)
HDL-C	−0.18 (.006)

NOTE. n = 267.

used to evaluate the significant factors associated with the HOMA-IR. The analysis of variance (ANOVA) and Scheffe test were used to evaluate the difference in the HOMA-IR according to the BMI groups.

RESULTS

The general characteristics and the clinical profiles of the insulin-resistant and insulin-sensitive Korean non-obese type 2 diabetic patients are shown in Tables 1 and 2. There were no significant differences according to age, disease duration, and HbA_{1c} levels between the 2 groups. The BMI was significantly higher in the insulin-resistant group than in the insulin-sensitive group. The HOMA-IR value in the patients with insulin resistance and those with normal insulin sensitivity were 4.2 ± 1.4 and 1.5 ± 0.6, respectively. The HOMA–β-cell values in the patients with insulin resistance and those with normal insulin sensitivity were 46.1 ± 36.9 and 21.9 ± 47.5, respectively. The mean fasting glucose level was significantly higher in the insulin-resistant group than in the insulin-sensitive group. The median fasting insulin level was also significantly higher in the insulin-resistant group than in the insulin-sensitive group. There was no significant difference in total cholesterol and LDL-C levels between the 2 groups. However, the patients with insulin resistance had significantly higher log-transformed TG levels (5.2 ± .6 *v* 4.9 ± .7) than those with normal insulin sensitivity. The HDL-C level was significantly lower in the insulin-resistant group (1.13 ± 0.3 mmol/L) than in the insulin-sensitive group (1.25 ± 0.3 mmol/L).

After adjusting for BMI, the patients with insulin resistance had significantly higher log-transformed TG levels than those with normal insulin sensitivity. In addition, the HDL-C level was significantly lower in the insulin-resistant group than in the insulin-sensitive group (Table 2).

For the correlation factors to insulin resistance, the BMI (r = .25) and the TG levels (r = .16) were positively correlated with the HOMA-IR, and the HDL-C levels were negatively correlated with the HOMA-IR (r = −0.18) (Table 3).

The HOMA-IR value was independently predicted by BMI and HDL-C, which explained 7% and 3% of the variability in the insulin-resistant group, respectively (Table 4). However, the TG levels were not independently associated with HOMA-IR. Logistic regression analysis, the significant factor associated with HOMA-IR, was only the BMI (Table 5). This result

Table 4. Variables Effect on HOMA-IR

	Partial R^2	F	*P*
BMI (kg/m^2)	0.07	16.99	.0001
HDL-C	0.03	7.68	.006

NOTE. n = 267.

Table 5. Risk Factors of HOMA-IR

		95% CI	
	Odds Ratio	Lower	Upper
BMI (kg/m^2)	1.51	1.20	1.89
Triglycerides	1.00	0.99	1.00
HDL-C	0.97	0.94	0.99

NOTE. n = 267.
Abbreviation: CI, confidence interval.

was not affected by age, because there was no relationship between age and BMI.

The HOMA-IR values in the BMI ≤ 20, 20 < BMI ≤ 21, 21< BMI ≤ 22, 22 < BMI ≤23, and 23 < BMI ≤ 24 and 24< BMI ≤ 25 were 1.3 ± .9, 1.8 ± 1.0, 1.7 ± 1.0, 2.0 ± 1.5, 2.3 ± 1.5, and 2.6 ± 1.6, respectively. The HOMA-IR values were significantly higher in the 24 < BMI ≤ 25 group (2.6 ± 1.6) than in the BMI < 20 group (1.3 ± 0.9) (Fig 1).

DISCUSSION

It has been shown that insulin-sensitive and insulin-resistant variants exist in non-obese Korean type 2 diabetic patients, and that BMI is the main determinant of insulin resistance even in non-obese type 2 diabetic patients.

Various methods have been proposed for insulin sensitivity in vivo. The euglycemic clamp study and minimal model analysis are standard tools for estimating insulin sensitivity, but the procedures are complex and expensive.[13,14] Insulin resistance index assessed by HOMA-IR is a tool to estimate insulin sensitivity from a single sample. HOMA-IR is closely correlated with insulin resistance index assessed by euglycemic clamp in type 2 daibetic patients.[15] Fukushima et al[16] recently demonstrated that the HOMA-IR value is highly correlated with insulin resistance calculated by the minimal model approach in subjects with varying degrees of glucose tolerance. These findings favor the use of HOMA-IR in the assessment of insulin resistance in large population studies. In this study, HOMA-IR greater than 2.5 were defined as being insulin resistant according to our data (Mean ± 1.5 SD of 1,901 normal subjects), which was also the same cut off point with a Japanese study.[17]

The percentage of insulin-resistant diabetic patients in this

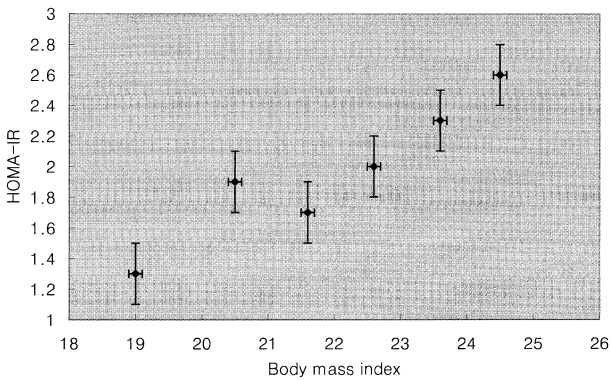

Fig 1. The relationship between BMI and HOMA-IR.

study was 23.6%, which is similar to data reported by Taniguchi et al[17] who showed that 31% of non-obese Japanese type 2 diabetic people were insulin resistant. However, Haffner et al[3] demonstrated that 92% of type 2 diabetics was insulin resistant in the white population. Banerji et al[18] previously disclosed that insulin resistance in black Americans with type 2 diabetes was 60% of those with a BMI 30 $\leq$ kg/m^2. Taniguchi et al[4] demonstrated that 40% of Japanese non–insulin-dependent diabetic patients were insulin resistant with a BMI of 16 to 44 kg/m^2. This discrepancy may be due to the ethnic differences, as well as the difference in the range of BMI in each study group. However, we confirmed that the major portion of type 2 diabetes has insulin-sensitive and insulin-deficient characteristics indicated by low HOMA–β-cell function in our group.

In this study, the insulin-resistant variant was small, which was associated with a higher BMI, TG, and lower HDL-C than those of the insulin-sensitive variant. Many previous studies demonstrated that the fasting hypertriglyceridemia and low HDL-C concentrations were associated with insulin resistance.[19,20] This dyslipidemia is important in the development of atherosclerosis and coronary heart disease.[18] These results supported previous data that showed a high fasting TG level and a low HDL-C level in the insulin-resistant variant.

In addition to the association of dyslipidemia with insulin resistance, Taniguchi et al[4,20,21] also reported that hypertriglyceridemia is an independent factor on insulin resistance besides BMI in Japanese type 2 diabetic patients with a midrange BMI (21 to 26 kg/m^2). However, these finding were not observed in our study group. The factors influencing insulin resistance were analyzed after adjusting for BMI. HDL-C and log-transformed TG levels were significant factors after adjusting for BMI. The statistical significance of the log- transformed TG levels was lower than the HDL-C levels. However, multiple regression analyses showed that insulin resistance was independently predicted by both BMI and HDL-C levels and not by TG levels. Therefore, these results did not show hypertriglyceridemia as an independent factor for insulin resistance. The reason for this is unclear.

Bo et al[22] reported that BMI and uric acid levels were significantly associated with the presence of hypertriglyceridemia in type 2 diabetic patients. It is possible that TG levels and BMI are associated with insulin resistance not independently, but concurrently, with the same underlying factor.

Logistic analysis showed that BMI was the only determinant of insulin resistance. Although our study subjects were non-obese, BMI was the most important factor for their insulin resistance. Many studies have suggested that central obesity or an adverse fat distribution is associated with developing type 2 diabetes. These studies revealed the groups that have a high prevalence of diabetes without obesity.[23-25] In this study, the waist-to-hip ratio (WHR) as a marker of the fat distribution was not measured. Therefore, the possibility that WHR is associated with insulin resistance cannot be excluded.

Our subjects were non-obese according to the World Health Organization (WHO) classification,[26] and it is conceivable that Koreans with a BMI $>$23 kg/m^2 have a body composition whose percent body fat is comparable to that of Caucasians with BMI $>$25 and could explain the associated increased risk for insulin resistance. This study showed that the number of insulin-resistant patients with a BMI above 23 kg/m^2 sharply increased. Sung et al[27] reported that obesity is the risk factor for Koreans with non–insulin-dependent diabetes. They showed the relative risks for diabetes mellitus in those with a BMI $\geq$23 kg/m^2 were higher than those with a BMI $<$ 23 kg/m^2. In Korea, an increasing BMI is directly related to insulin resistance and type 2 diabetes, even in non-obese subjects. Therefore, it may be inappropriate to define this diabetic population as non-obese, because the results show that BMI $>$23 are associated with an increased risk of insulin resistance. These results show the possibility to change the classification for obesity in Asians. In addition, a greater proportion of our type 2 diabetic subjects were insulin sensitive, had a lower BMI, and were characterized by a lower HOMA–β-cell value, indicating lower insulin output. Thus, they may relate to the insulin-deficient, but insulin-sensitive subgroup of type 2 diabetes mellitus and a different therapeutic approach might be needed compared with insulin-resistant type 2 diabetic patients.

In conclusion, this study suggested that insulin resistance occurred in 23.6% of Korean non-obese type 2 diabetic patients. The factors associated with insulin resistance were a high BMI, as well as high TGs, and low HDL-C levels. However, the BMI was the only determinant of insulin resistance.

REFERENCES

1. Defronzo RA: Lilly Lecture 1987: The triumvirate: beta-cell, muscle, liver; a collusion responsible for NIDDM. Diabetes 37:667-687, 1988

2. Gerich JE: The genetic basis of type 2 diabetes mellitus: Impaired insulin secretion versus impaired insulin sensitivity. Endocr Rev 19: 491-503, 1988

3. Haffner SM, D'Agostino R Jr, Mykkanen L, et al: Insulin sensitivity in subjects with type 2 diabetes. Relationship to cardiovascular risk factors: The Insulin Resistance Atherosclerosis study. Diabetes Care 22:562-568, 1999

4. Taniguchi A, Fukushima M, Sakai M, et al: The role of body mass index and triglyceride levels in identifying insulin-sensitive and insulin resistant variants in Japanese non–insulin-dependent diabetic patients. Metabolism 49:1001-1005, 2000

5. Matthews D, Hosker J, Rudenski A, et al: Homeostasis model assessment: Insulin resistance and B-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 28:412-419, 1985

6. Park YS, Lee HK, Kim SY, et al: Risk factors for non-insulin dependent diabetes mellitus. Diabetes 20:14-23, 1996

7. Yoon KH: Clinical characteristics of diabetes mellitus in Korea. Food Industry Nutr 4:73-82, 1999

8. Lee TH: Prevalence of obesity in Korean non-insulin–dependent diabetic patients. Diabetes Res Clin Pract 2:71-80, 1996

9. Park JY, Lee KU, Kim CH, et al: Past and current obesity in Koreans with non- insulin-dependent diabetes mellitus. Diabetes Res Clin Pract 35:49-56, 1997

10. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus: Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care 20:1183-97, 1997

11. Matsumoto K, Yamaguchi Y, Miyake S, et al: Glucose tolerance, insulin secretion, and insulin sensitivity in non obese and obese Japanese subjects. Diabetes Care 20:1562-1568, 1997

12. 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

13. Bergman RN, Phillips LS, Cobeli C: Physiologic evaluation of factors controlling glucose tolerance in man: Measurement of insulin sensitivity and beta cell glucose sensitivity from the response to intravenous glucose. J Clin Invest 68:1456-1467, 1981

14. Finegood DT, Hramiak IM, Dupre J: A modified protocol for estimation of insulin sensitivity with the minimal model of glucose kinetics in patients with insulin dependent diabetes. J Clin Endocrinol Metab 70:1538-1549, 1990

15. Matthews DR, Hosker JP, Rudenski AS, et al: Homeostasis model assessment: Insulin resistance and beta-cell function from fasting plasma glucose and insulin concentration in man. Diabetologia 28:412-419, 1985

16. Fukushima M, Taniguchi A, Sakai M, et al: Homeostasis model assessment as a clinical index of insulin resistance. Diabetes Care 22:1911-1912, 1999 (letter)

17. Taniguchi A, Nakai Y, Sakai M, Yoshi S, et al: Relationship of regional adiposity to insulin resistance in non obese Japanese type 2 diabetic patients. Diabetes 24:966-967, 2001

18. Banerji MA, Lebovitz HE: Insulin-sensitive and insulin-resistant variants in NIDDM. Diabetes 38:784-792, 1989

19. Fontbonne A, Eschwege E, Cambien F, et al: Hypertriglyceridaemia as a risk factor of coronary heart disease mortality in subjects with impaired glucose tolerance or diabetes: Results from the 11-year follow-up of the Paris Prospective Study. Diabetologia 32:300-304, 1989

20. Taniguchi A, Fukushima M, Sakai M, et al: Insulin-sensitive and insulin-resistance variants in nonobese Japanese type 2 diabetic patients: The role of triglycerides in insulin resistance. Diabetes Care 22:2100-2101 (letter), 1999

21. Taniguchi A, Fukushima M, Sakai M, et al: Remnant-like particle cholesterol, tirglycerides, and insulin resistance in nonobese Japanese type 2 diabetic patients. Diabetes Care 23:1766-1769, 2000

22. Bo S, Cavallo-Perin P, Gentile L, et al: Low HDL-cholesterol: A component metabolic syndrome only in the presence of fasting hypertriglyceridemia in type 2 diabetic patients. Diabetes Metab 27:31-35, 2001

23. Okosun IS: Ethnic differences in the risk of type 2 diabetes attributed to differences in abdominal adiposity in American women. J Cardiovasc Risk 7:425-430, 2000

24. Ramachandran A, Snehalatha C, Viswanathan V, et al: Risk of non insulin dependent diabetes mellitus conferred by obesity and central adiposity in different ethnic group. A comparative analysis between Asian Indians, Mexican Americans and Whites. Diabetes Res Clin Pract 36:121-125, 1997

25. Araneta MR, Wingard DL, Barrett-Connor E: Type 2 diabetes and metabolic syndrome in Filipina-American women: A high-risk non obese population. Diabetes Care 25:494-499, 2002

26. World Health Organization: Obesity. The Asia-Pacific perspective. Redefining obesity and its treatment. Health Communications Australia Pty Limited, International Diabetes Institute, 2000

27. Sung EJ, Sunwoo S, Kim SW, et al: Obesity as a risk factor for non-insulin-dependent diabetes mellitus in Korea. J Korean Med Sci 16:391-396, 2001