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Adiposity Indices in the Prediction of Metabolic Abnormalities associated with Cardiovascular Disease in Non-Diabetic Adults

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Abstract

Background and Aims—The prevalence of insulin resistance and cardiovascular disease (CVD) increases with degree of obesity. Whether measurement of generalized or abdominal obesity differs in ability to predict changes associated with increased CVD risk is debated. We compared the prevalence of metabolic abnormalities in 275 women and 204 men stratified by categories of body mass index (BMI) and waist circumference (WC), and assessed the ability of these adiposity indices in combination with metabolic risk variables to predict insulin resistance.

Methods and Results—Healthy, non-diabetic volunteers underwent measurements of BMI, WC, blood pressure, fasting plasma glucose (FPG), lipoprotein concentrations, and direct quantification of insulin-mediated glucose uptake. Insulin resistance was defined as the top tertile of steady-state plasma glucose (SSPG) concentrations. BMI and WC were highly correlated (P <0.001) in both women and men. Abnormal SSPG and triglyceride concentrations were associated with increasing adiposity by either index in both genders. Among women, abnormal FPG and high density lipoprotein cholesterol (HDL-C) concentrations were associated with increasing BMI and WC. In men, abnormal HDL-C was associated with increasing BMI only. Elevated systolic blood pressure (SBP) was associated with increasing BMI in both genders. The odds of insulin resistance were greatest in women with elevated FPG and triglycerides (4.5-fold). In men, the best predictors were BMI and SBP, and WC and HDL-C (3-fold).

Conclusion—BMI is at least comparable to WC in stratifying individuals for prevalence of metabolic abnormalities associated with increased CVD risk and predicting insulin resistance.

Keywords

insulin resistance; obesity; cardiovascular disease; metabolic risk

Introduction

While the link between obesity and risk of cardiovascular disease (CVD) has been well-established [1,2], recent attention has shifted to the relative importance of central or

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abdominal obesity as a specific marker of increased CVD risk. This distinction has been highlighted by inclusion of waist circumference (WC) in the diagnosis of metabolic syndrome (MS), as either a necessary [3] or one of several [4] criteria. Nonetheless, the emphasis placed on WC over generalized adiposity as measured by body mass index (BMI) may not be merited, as large-scale population studies have shown that WC and BMI are highly correlated [5,6], and that BMI is at least as effective at predicting incidence of adverse clinical outcomes [6,7]. The present study was initiated to address this issue in a somewhat different manner. Given the reliance on pre-defined cutpoints to classify individuals into `healthy' and `unhealthy' weight categories, we sought to determine whether use of WC cutpoints was indeed, superior to conventional cutpoints for BMI in identifying individuals at risk for metabolic abnormalities. Specifically, we used the four metabolic abnormalities that comprise a diagnosis of MS, as well as total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and insulin resistance, and compared the prevalence of these abnormalities across pre-defined categories of BMI and WC. Furthermore, since there is evidence that insulin resistance is the link between excess adiposity and CVD [8], we evaluated the ability of individual and combined metabolic variables to predict insulin resistance.

Methods

Subjects and study design

The study population consisted of 275 women and 204 men who had responded to study advertisements describing our research interest in the role of insulin resistance in human disease. All subjects gave informed consent, and the Stanford Human Subjects Committee approved the study protocols. All studies were performed at the Clinical and Translational Research Unit at Stanford University Medical Center. Participants were apparently healthy, with normal physical examination findings and health histories, and non-diabetic defined according to the American Diabetes Association [9]. Of note, a small subset of individuals were receiving anti-hypertensive or lipid-lowering therapy, although information was not available for all subjects. Height and weight were measured when subjects wore light clothing and no shoes. BMI was calculated (kilogram/meter^2), and WC (centimeters) measured as the midpoint between iliac crest and rib cage at end-expiration [10]. Plasma samples for glucose, insulin, and lipid/lipoprotein concentrations were measured after overnight fast by methods that were identical during the period of study.

Individuals were categorized two ways. First, conventional cutpoints of BMI <25 and 25–29.9 kg/m^2 defined `normal' and `overweight', respectively. BMI 30–34.9 and ≥35 kg/m^2 constituted categories in the `obese' BMI range. The second method of classification was by WC. Because ATPIII criteria identifies `healthy' from `unhealthy' abdominal girth solely as above and below a cutpoint, we used their criteria in women (≥88 cm) and men (≥102 cm) as points of reference [11], then defined the other categories around those cutpoints to parallel those of BMI. Based on the correlation between BMI and WC as evidenced later and for convenience of use, we chose 10 cm increments to define WC categories. Thus, categories of WC for women were defined as <88 (normal), 88–97.9, 98–107.9, and ≥108 cm. Categories of WC for men were defined as <102 (normal), 102–111.9, 112–121.9, and ≥122 cm. Metabolic variables from ATPIII criteria for MS were defined as fasting plasma glucose (FPG) ≥100 mg/dl (≥5.55 mmol/L), systolic blood pressure (SBP) ≥130 mmHg, diastolic blood pressure (DBP) ≥85 mmHg, triglycerides (TG) ≥150 mg/dl (≥1.70 mmol/L), high density lipoprotein cholesterol (HDL-C) <50 mg/dl (<1.30 mmol/L) in women, and HDL-C <40 mg/dl (<1.04 mmol/L) in men. Additional variables were TC ≥200 mg/dl (≥5.18 mmol/L) and LDL-C ≥160 mg/dl (≥4.14 mmol/L).

Insulin-mediated glucose disposal was quantified by a modification [12] of the insulin suppression test as originally described and validated by our research group [13,14]. After an overnight fast, subjects were infused for 180 minutes with octreotide (0.27 μg/m^2/min), insulin (32 mU/m^2/min), and glucose (267 mg/m^2/min). Plasma glucose and insulin were measured every 10 minutes during the 150- to 180-minute period and averaged to determine steady-state plasma glucose (SSPG) and insulin (SSPI) concentrations. Because SSPI is similar for all individuals, SSPG provides a direct measure of the ability of insulin to mediate disposal of an infused glucose load; the higher the SSPG, the more insulin-resistant the individual. Measurements of insulin-mediated glucose uptake using the insulin suppression test were shown to be essentially identical to those obtained using the hyperinsulinemic euglycemic clamp method [13]. Based on earlier studies showing increased risk of clinical events in the subgroup in the upper tertile of SSPG, we defined cut-off values for insulin resistance as SSPG ≥180 mg/dl (≥10.0 mmol/L) [15,16].

Statistical analysis

Statistical analyses were performed using SPSS software 15.0 (Chicago, IL, USA). Pearson's correlation coefficients and linear regression analyses were used to characterize relationships between BMI and WC. Clinical characteristics between women and men were compared using unpaired *t*-tests. Frequency distribution of variables across BMI and WC categories were analyzed using chi-squared tests.

Clinical utility of metabolic risk variables in identifying insulin resistant individuals was assessed in univariate analysis, then included in a backward step-wise logistic regression model for multivariate analysis. The positive predictive value (PPV) of a given variable was calculated as the percentage of subjects with the risk variable that were correctly identified as insulin resistant. Positive likelihood ratios (PLR) were calculated as sensitivity/ (1 – specificity). Negative likelihood ratios (NLR) were calculated as (1 – sensitivity)/ specificity., *P*-value <0.05 was taken to indicate statistical significance.

Results

The study population included mostly Caucasians (72%), with smaller representation from Asians (12%), Hispanics (9%), African-Americans (6%), and other minorities (1%) (Table 1). While there were no differences in age, BMI, SSPG, TC, or LDL-C, men had higher mean values of WC, FPG, SBP, DBP, and TG, and lower HDL-C than women.

BMI and WC were highly correlated (*P* <0.001) in both women (*r*= 0.78) and men (*r*= 0.84). Linear regression analysis revealed the following relationships: WC= 35.2 + (2.05 * BMI) in women, and WC= 39.05 + (2.18 * BMI) in men.

Table 2 presents the prevalence of metabolic risk variables by BMI and WC categories for women. *P*-values for trends of FPG, SSPG, TG, HDL-C, and MS across BMI and WC categories were statistically significant. Additionally, trend for increased SBP was evident across BMI categories, but not WC categories. On the other hand, elevated DBP, TC, and LDL-C did not appear to increase in frequency by either adiposity estimate. Qualitative side-by-side comparisons of BMI and WC values within each category also revealed comparable frequencies of a given variable, although few differences were noted in the lowest (normal weight) category. Among women grouped by WC in Category 1 (C1), 16% were insulin resistant and had elevated SBP, and 64% had abnormal BMI. In contrast, none of the women with normal BMI were insulin resistant, and only half as many (8% vs 16%) had elevated SBP. The other metabolic abnormalities in C1 were similar whether grouped by BMI or WC. The rationale for using 10 cm increments to approximate WC categories to parallel that of BMI is evident in the correlation between BMI and WC in women; based on the linear

regression equation described earlier, BMI 25, 30, and 35 kg/m^2 were analogous to WC 85, 97, and 107 cm, respectively.

Among men, trends of SSPG, TG, and MS paralleled increasing adiposity by BMI or WC categories (Table 3). However, trends for abnormal SBP and HDL-C were statistically significant only when stratified by BMI categories. Unlike in women, prevalence of impaired FPG did not differ across BMI or WC categories in men.

Turning to comparisons of BMI and WC groups within each category, several differences were noted in the lowest category. Specifically, all metabolic abnormalities were present at a higher prevalence in men when grouped by WC as compared with BMI in C1. Compared to those grouped by BMI, men grouped by WC had increased prevalence of abnormal SSPG (20 vs 5%), BMI (76% vs 0%), SBP (35% vs 10%), TG (33 vs 10%), HDL-C (51 vs 20%), and MS (19 vs 0%). These differences were somewhat less evident in Category 2 (C2), where men grouped by WC as compared to BMI, had increased prevalence of abnormal SSPG (52 vs 27%), WC (100 vs 36%), and MS (68 vs 42%). These discrepancies may be explained upon further examination of the relationship between BMI and WC in men. Using the linear regression equation reported earlier, BMI values 25, 30, and 35 kg/m^2 approximated WC 94, 104, and 115 cm, respectively. If we extrapolate from these data, men with WC 94–102 cm (C1) may be more comparable to men with BMI 25–29.9 kg/m^2 (C2), than men with BMI <25 kg/m^2 (C1). Similarly, men with WC 102–111.9 cm (C2) may be more comparable to men in the BMI range of Category 3 (30–34.9 kg/m^2). Nonetheless, the prevalence of metabolic abnormalities between BMI and WC in Categories 3 and 4 were fairly comparable, suggesting there is a threshold effect of adiposity on the prevalence of risk variables; the prevalence of metabolic abnormalities in men with larger BMI or WC values is overall high, rendering differences in prevalence less apparent.

We then evaluated the odds ratios for association of insulin resistance, as defined by the top tertile of SSPG, with metabolic risk variables (Table 4). Women with abnormal FPG, WC, TG, and HDL-C had 3- to 4-fold higher odds of insulin resistance as compared to women with normal values (P <0.001). Of note, the odds ratio could not be calculated for the variable BMI because no women with BMI< 25 kg/m^2 were insulin resistant. In multivariate analysis, women with abnormal FPG, TG, and HDL-C (P <0.001) had increased odds of insulin resistance. In men, WC, BMI, SBP, TG, and HDLC were significantly associated with increased odds of insulin resistance (P <0.05). BMI 30 kg/m^2 was used in the analyses of men because it better approximated the WC cut-off 102 cm. In multivariate analysis, WC, BMI, SBP, TG, and HDL-C remained statistically significant.

Table 5 presents data on the PPV, PLR, and NLR of individual and combined metabolic risk variables to predict insulin resistance. Among women, the PPV was greatest for the combined risk of having abnormal values of FPG and TG (72%), or PLR 4.52. Testing positive for abnormal FPG and HDL-C conferred the next greatest odds of having insulin resistance (4-fold). Among men, testing positive for BMI and SBP (PPV 65%, PLR 2.83), and abnormal WC and HDL-C (PPV 64%, PLR 2.71) were the strongest predictors. For both women and men, testing negative for either measure of adiposity yielded the smallest NLR, i.e. decreased the false negative error rate the most.

Discussion

Our study goals were two-fold—namely, to compare prevalence of metabolic changes associated with increased risk of CVD when stratified by categories of BMI and WC, and to evaluate the ability of these adiposity indices and metabolic markers to predict insulin resistance in women and men.

Our study re-demonstrates [5,17] that BMI and WC measurements are highly correlated. We sought to assess the strength of this association by determining whether it could be translated across pre-defined weight categories when comparing prevalence of metabolic risk variables. Our results indicate that metabolic abnormalities including and related to insulin resistance such as SSPG, TG, and MS increased in prevalence across categories for women and men whether stratified by BMI or WC. Additionally, FPG and HDL-C were statistically significant across BMI and WC categories in women. Abnormal HDL-C was statistically significant in men when stratified by BMI only, and SBP was statistically significant by BMI only in both genders. Prevalence of abnormal TC and LDL-C values did not trend across categories, signifying that these abnormalities did not vary as a function of increasing adiposity. Taken together, these results suggest that BMI and WC are comparable in their abilities to identify individuals with metabolic abnormalities that increase risk of CVD. If anything, the prevalence of abnormal SBP (in men and women) and HDL-C (in men) varied as a function of increased BMI but not WC, suggesting a stronger adverse effect of generalized than central adiposity on these variables. Prior studies support that blood pressure may be more strongly associated with BMI than abdominal obesity [18,19].

Turning now to comparison of prevalence of metabolic abnormalities within each category, women demonstrated roughly equivalent proportions of any given metabolic risk factor at corresponding categories of BMI or WC, with the exception of few differences in C1 (normal weight). Differences in C1 were more striking among men; indeed, men deemed to have normal WC had higher prevalence of virtually all metabolic abnormalities than men with normal BMI. As discussed earlier, a mismatch in WC and BMI cut-offs may account for these differences. These findings may tempt one to propound a lowering of the WC cutoff of 102 cm, the threshold set by ATPIII for abnormal abdominal girth in men. Indeed, the International Diabetes Federation's criteria for MS using WC ≥94 cm (in European men) is likely based on this rationale [3]. Nonetheless, it is important to point out that use of a lower threshold value carries with it the risk of losing specificity when identifying individuals with cardiovascular risk factors [20]. Indeed, if the goal were to increase sensitivity at the expense of specificity, BMI would be superior to WC in capturing men with either abdominal or generalized obesity. To illustrate this point, BMI 25 kg/m^2 as a cut-off excluded no men with WC ≥102 cm, and would overlook 5 of 20 men with WC 94–102 cm (data not shown). On the other hand, lowering the WC threshold to 94 cm would still fail to identify 16 of 31 men with BMI ≥25 kg/m^2 in our population sample. At the very least, these results re-emphasize that using WC does not provide any obvious advantage over BMI in identifying individuals at risk for metabolic changes associated with CVD.

The second goal of our study was to assess how well these adiposity indices and the other metabolic markers predicted insulin resistance. It is important to re-address that not all obese individuals are at equal risk for developing adverse health consequences, and that presence of insulin resistance identifies those at greatest risk for developing type 2 diabetes and CVD [16,21,22]. We have shown previously that either estimate of adiposity accounts for only one-third of the variability in insulin action in non-diabetic individuals [17], and that identifying those who are insulin resistant has clinical utility [23]. In this study, abnormal FPG, TG, and HDL-C levels—but neither BMI nor WC—were independent predictors of insulin resistance in women. Supporting these findings, these variables in combination provided the greatest PPV/ PLR in predicting insulin resistance. In men, along with SBP, TG, and HDL-C, BMI and WC independently predicted insulin resistance. It is worthwhile to re-emphasize that BMI and WC performed comparably in predicting insulin resistance in both groups. Thus, it seems reasonable to question the view that an abnormal WC imparts risk of CVD above that of other variables [3]. Our results extend findings of a previous study in which we showed that mean values for select metabolic markers were similar when compared as stratified above and below BMI and WC thresholds [24]. It is also noteworthy

that while having MS did increase the odds of insulin resistance, it was not the strongest predictor of the top tertile of SSPG for women or men.

A few words about the gender differences found are warranted. Interestingly, FPG was modulated by obesity in women but not men, and was more important than either obesity estimate in CVD risk assessment for women. By contrast, both obesity measures contributed substantially to prediction of insulin resistance in men. One might speculate whether gender-specific disparities related to body fat storage account for these findings. At the very least, these data implicate gender as an additional factor in influencing CVD risk assessment.

It should be pointed out that our study included mostly subjects of European ancestry and may not be generalized broadly. It is also possible that selecting for apparently healthy individuals muted discernible differences between BMI and WC categories. In this context, it is helpful to consider these results with respect to other study populations. For all evidence emphasizing the utility of central over generalized adiposity measures in predicting metabolic abnormalities or adverse outcomes [25,26], there are comparable data reporting the opposite or at least, equivalent efficacy [2,6,27]. BMI had the highest hazard ratio of various adiposity measurements in predicting incident diabetes in Pima Indians [28]. In a large 10-year prospective study of Finnish subjects [7], BMI rather than WC was a superior predictor of CVD risk. Abdominal obesity did not contribute more than other metabolic variables to a predefined cardiovascular risk factor cluster in a European cohort of non-diabetic subjects. [29]. We have also shown that while WC provides some additive benefit to BMI in predicting insulin resistance, the converse is also true [17]. It should also be reminded that WC measurement does not differentiate visceral from subcutaneous abdominal adiposity. But of studies that have quantified regional fat mass, the prevailing data are that the two adipose tissue depots correlate similarly with insulin action [8].

In conclusion, our results demonstrate that metabolic abnormalities associated with CVD risk vary comparably as a function of BMI or WC. Furthermore, when evaluated for their ability to predict insulin resistance, both obesity estimates performed similarly, whether independently or in combination with other metabolic variables. In this context it should be noted that either adiposity estimate performed better at predicting insulin resistance in men than in women. It can be argued that measurement of WC is more difficult than BMI, as varying measurements can be obtained depending upon which guidelines are followed [30]. Nonetheless, our data provide guidelines for clinicians who may choose to use either BMI or WC in the office setting, to help identify individuals at increased risk for insulin resistance and CVD.

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Table 1

Clinical characteristics of the study population

	Women (n=275)	Men (n=204)	
	Mean (Range)	Mean (Range)	P[a]
Age (years)	50 (22–71)	51 (27–65)	0.43
BMI (kg/m^2)	29.8 (20–50.5)	29.9 (18.8–41.3)	0.70
WC (cm)	96 (67.5–140.5)	104 (73.5–134)	<0.001
SSPG (mmol/L)	8.33 (2.28–17.3)	8.78 (2.11–17.1)	0.23
FPG (mmol/L)	5.28 (3.89–6.94)	5.44 (3.61–6.94)	0.01
SBP (mmHg)	121 (89–169)	127 (99–167)	<0.001
DBP (mmHg)	71 (48–95)	78 (58–102)	<0.001
TG (mmol/L)	1.28 (0.17–4.60)	1.91 (0.25–11.4)	<0.001
HDL-C (mmol/L)	1.32 (0.52–2.59)	1.04 (0.28–2.12)	<0.001
TC (mmol/L)	5.02 (2.80–7.61)	4.97 (2.72–10.5)	0.55
LDL-C (mmol/L)	3.13 (1.40–5.26)	3.13 (0.28–9.12)	0.97

BMI, body mass index; WC, waist circumference; SSPG, steady-state plasma glucose; FPG, Fasting plasma glucose; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triglycerides; HDL-C, high density lipoprotein cholesterol; TC, total cholesterol; LDL-C, low density lipoprotein cholesterol

[a] Statistical significance as determined by Student's *t* test.

Table 2

Prevalence (%) of metabolic abnormalities by categories of BMI and WC for women (n=275)

	Category 1		Category 2		Category 3		Category 4			
	BMI (kg/m^2)	WC (cm)	BMI (kg/m^2)	WC (cm)	BMI (kg/m^2)	WC (cm)	BMI (kg/m^2)	WC (cm)		
	(<25)	(<88)	(25–29.9)	(88–97.9)	(30–34.9)	(98–107.9)	(≥35)	(≥108)	*P*-values[a]	
Variable	**(n=26)**	**(n=61)**	**(n=137)**	**(n=101)**	**(n=82)**	**(n=62)**	**(n=30)**	**(n=51)**	**BMI**	**WC**
FPG ≥ 5.55 mmol/L	12%	18%	30%	31%	34%	40%	60%	45%	<0.001	0.01
SSPG ≥ 10.0 mmol/L	0%	16%	31%	35%	43%	44%	77%	57%	<0.001	<0.001
WC ≥ 88 cm	15%	0%	73%	100%	98%	100%	100%	100%	<0.001	---
BMI ≥ 25 kg/m^2	0%	64%	100%	96%	100%	100%	100%	100%	---	<0.001
SBP ≥ 130 mmHg	8%	16%	22%	27%	38%	32%	30%	29%	0.01	0.21
DBP ≥ 85 mmHg	8%	7%	4%	8%	12%	6%	7%	8%	0.20	0.98
TG ≥ 1.70 mmol/L	12%	13%	19%	20%	28%	27%	37%	35%	0.06	0.03
HDL-C < 1.30 mmol/L	31%	31%	45%	55%	54%	50%	73%	59%	0.01	0.01
TC ≥ 5.18 mmol/L	38%	39%	40%	39%	46%	42%	33%	47%	0.61	0.78
LDL-C ≥ 4.14 mmol/L	8%	8%	11%	14%	16%	6%	7%	18%	0.46	0.20
ATPIII criteria MS	8%	7%	31%	39%	44%	47%	67%	55%	<0.001	<0.001

ATPIII, Adult Treatment Panel III; MS, metabolic syndrome

[a] *P*-values reported are chi-squared tests for trends of each variable across categories of BMI and WC.

Table 3

Prevalence of metabolic abnormalities by categories of BMI and WC for men (n=204)

	Category 1		Category 2		Category 3		Category 4			
	BMI (kg/m^2)	WC (cm)	BMI (kg/m^2)	WC (cm)	BMI (kg/m^2)	WC (cm)	BMI (kg/m^2)	WC (cm)		
	(<25)	(<102)	(25–29.9)	(102–111.9)	(30–34.9)	(112–121.9)	(≥35)	(≥122)	P-values[a]	
Variable	(n=20)	(n=84)	(n=78)	(n=69)	(n=81)	(n=39)	(n=25)	(n=12)	BMI	WC
FPG ≥ 5.55 mmol/L	25%	31%	42%	45%	40%	46%	32%	25%	0.48	0.16
SSPG ≥ 10.0 mmol/L	5%	20%	27%	52%	49%	44%	72%	83%	<0.001	<0.001
WC ≥ 102 cm	0%	0%	36%	100%	84%	100%	96%	100%	<0.001	---
BMI ≥ 25 kg/m^2	0%	76%	100%	100%	100%	100%	100%	100%	---	<0.001
SBP ≥ 130 mmHg	10%	35%	42%	41%	40%	46%	64%	67%	<0.001	0.16
DBP ≥ 85 mmHg	15%	23%	26%	25%	20%	18%	24%	17%	0.69	0.83
TG ≥ 1.70 mmol/L	10%	33%	46%	58%	44%	33%	60%	67%	0.01	<0.001
HDL-C < 1.04 mmol/L	20%	51%	59%	54%	53%	49%	64%	83%	0.01	0.19
TC ≥ 5.18 mmol/L	20%	33%	37%	45%	46%	41%	40%	42%	0.20	0.52
LDL-C ≥ 414 mmol/L	0%	14%	13%	13%	17%	10%	8%	8%	0.18	0.89
ATPIII criteria MS	0%	19%	42%	68%	57%	64%	76%	83%	<0.001	<0.001

[a] *P*-values reported are chi-squared tests for trends of each variable across categories of BMI and WC.

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Table 4

Odds ratios for the association of top tertile of steady-state plasma glucose concentrations with metabolic risk variables

	Women (n=275)					Men (n=204)			
	Univariate		Multivariate[a]			Univariate		Multivariate[a]	
Variables	OR(95% CI)	P	OR(95% CI)	P	Variables	OR(95% CI)	P	OR(95% CI)	P
FPG ≥ 5.55 mmol/L	3.79(2.23–6.46)	<0.001	3.33(1.87–5.92)	<0.001	FPG≥ 5.55 mmol/L	1.60(0.90–2.84)	0.11		
<5.55	1.00		1.00		<5.55	1.00			
WC ≥ 88 cm	3.77(1.82–7.83)	<0.001			WC≥ 102 cm	4.36(2.29–8.28)	<0.001	2.36(1.08–5.17)	0.03
<88	1.00				<102	1.00		1.00	
BMI ≥ 25 kg/m^2	N/A[b]				BMI≥ 30 kg/m^2	4.17(2.27–7.68)	<0.001	2.55(1.21–5.38)	0.01
<25	0.00				<30	1.00		1.00	
SBP ≥ 130 mmHg	1.33(0.77–2.30)	0.31			SBP≥ 130 mmHg	1.88(1.06–3.34)	0.03	2.17(1.06–4.48)	0.04
<130	1.00				<130	1.00		1.00	
DBP ≥ 85 mmHg	0.92(0.36–2.39)	0.87			DBP≥ 85 mmHg	0.82(0.41–1.63)	0.57		
<85	1.00				<85	1.00			
TG ≥ 1.70 mmol/L	3.60(2.00–6.45)	<0.001	2.72(1.43–5.16)	<0.001	TG≥ 1.70 mmol/L	2.55(1.43–4.54)	<0.001	2.02(1.04–3.91)	0.04
<1.70	1.00		1.00		<1.70	1.00		1.00	
HDL-C < 1.30 mmol/L	3.00(1.80–5.01)	<0.001	2.64(1.50–4.66)	<0.001	HDL-C< 1.04 mmol/L	2.18(1.22–3.90)	0.008	1.96(1.01–3.80)	0.046
≥ 1.30	1.00		1.00		≥ 1.04	1.00		1.00	
TC ≥ 5.18 mmol/L	0.97(0.59–1.59)	0.90			TC≥ 5.18 mmol/L	1.37(0.77–2.42)	0.29		
<5.18	1.00				<5.18	1.00			
LDL-C ≥ 4.14 mmol/L	0.64(0.29–1.45)	0.29			LDL-C≥ 4.14 mmol/L	0.96(0.41–2.25)	0.93		
<4.14	1.00				<4.14	1.00			

[a] In multivariate analysis, only variables that were statistically significant are listed.

[b] N/A- Not applicable because the odds of insulin resistance in women with BMI <25kg/m^2 equaled zero.

Table 5

Comparison of metabolic abnormalities and the ability to predict top tertile of steady-state plasma glucose concentrations

	Women (n=275)			Men (n=204)		
Variables	**PPV**	**PLR(95%CI)**	**NLR(95%CI)**	**PPV**	**PLR(95%CI)**	**NLR(95%CI)**
FPG ≥ 5.55 mmol/L	58%	2.36(1.68–3.31)	0.62(0.51–0.76)	46%	1.33(0.94–1.88)	0.83(0.68–1.02)
WC ≥ 88 cm women; ≥ 102 cm men	43%	1.27(1.14–1.43)	0.34(0.18–0.63)	53%	1.71(1.37–2.14)	0.39(0.25–0.61)
BMI ≥ 25 kg/m^2 women; ≥ 30 kg/m^2 men	41%	1.18(1.10–1.25)	0.00	55%	1.87(1.45–2.43)	0.45(0.31–0.65)
SBP ≥ 130 mmHg	42%	1.23(0.83–1.83)	0.93(0.81–1.06)	48%	1.44(1.04–2.00)	0.77(0.61–0.96)
TG ≥ 1.70 mmol/L	60%	2.62(1.68–4.07)	0.73(0.62–0.85)	52%	1.66(1.22–2.25)	0.65(0.50–0.85)
HDL-C < 1.30 mmol/L women; < 1.04 mmol/L men	49%	1.67(1.33–2.11)	0.56(0.42–0.74)	48%	1.41(1.10–1.81)	0.65(0.47–0.89)
FPG and TG	72%	4.52(2.08–9.83)	0.83(0.75–0.92)	57%	2.07(1.13–3.79)	0.85(0.75–0.97)
FPG and HDL-C	70%	4.06(2.28–7.22)	0.73(0.64–0.84)	55%	1.86(1.1–3.14)	0.83(0.72–0.97)
WC and TG	65%	3.26(1.98–5.38)	0.72(0.62–0.84)	62%	2.56(1.66–3.96)	0.64(0.52–0.80)
HDL-C and TG	64%	3.10(1.73–5.55)	0.80(0.71–0.90)	54%	1.82(1.21–2.73)	0.75(0.62–0.91)
BMI and TG	63%	2.98(1.87–4.73)	0.71(0.61–0.83)	63%	2.61(1.59–4.27)	0.71(0.59–0.85)
ATPIII criteria MS	63%	2.93(2.12–4.05)	0.48(0.37–0.62)	56%	1.98(1.49–2.63)	0.48(0.34–0.67)
FPG and WC	62%	2.81(1.92–4.13)	0.62(0.51–0.75)	56%	1.95(1.22–3.13)	0.78(0.66–0.93)
FPG and BMI	60%	2.56(1.80–3.64)	0.61(0.50–0.74)	60%	2.33(1.32–4.10)	0.80(0.69–0.93)
FPG and SBP	59%	2.52(1.30–4.88)	0.88(0.80–0.96)	44%	1.24(0.68–2.25)	0.95(0.85–1.07)
SBP and TG	57%	2.24(1.02–4.92)	0.92(0.86–1.00)	54%	1.79(1.04–3.10)	0.86(0.75–0.98)
SBP and HDL-C	54%	2.03(1.11–3.69)	0.89(0.81–0.98)	57%	2.02(1.21–3.36)	0.80(0.69–0.94)
BMI and HDL-C	52%	1.89(1.48–2.42)	0.52(0.39–0.69)	63%	2.61(1.67–4.07)	0.65(0.53–0.80)
WC and HDL-C	52%	1.88(1.44–2.45)	0.58(0.46–0.75)	64%	2.71(1.79–4.11)	0.59(0.47–0.74)
WC and SBP	48%	1.62(1.05–2.49)	0.86(0.76–0.98)	59%	2.25(1.42–3.59)	0.73(0.61–0.88)
BMI and SBP	43%	1.29(0.86–1.94)	0.91(0.80–1.04)	65%	2.83(1.68–4.76)	0.71(0.59–0.85)