

## Original Article

# Overall obesity had similar ability to identify the insulin resistance and pancreatic $\beta$ -cell function compared with abdominal obesity in Chinese community-dwelling population without type 2 diabetes

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**Abstract:** Background: Just as the prevalence of obesity is increasing, so is the prevalence of type 2 diabetes (T2DM). Obesity is considered to be a factor that drives insulin resistance (IR), and IR is the underlying mechanism through which persons develop T2DM. This analysis was to test the hypothesis that body mass index (BMI, and overall obesity) had similar ability to identify the IR and pancreatic  $\beta$ -cell function compared with waist circumference (WC, and abdominal obesity) in Chinese community-dwelling population without T2DM. Methods: This analysis consisted of 1330 participants without T2DM, who received the homeostasis model assessment of IR (HOMA-IR) and HOMA- $\beta$  determination. Results: Age ranged from 18 to 80 years, with a median of 38 years. Logistic regression analyses indicated that gender, BMI (and overall obesity), WC (and abdominal obesity), FBG and TG levels were independently associated with IR status and pancreatic  $\beta$ -cell function expressed as HOMA-IR and HOMA- $\beta$ . C-statistic of BMI to identify the IR status and pancreatic  $\beta$ -cell function had no statistically significant difference with that of WC. Conclusions: Both BMI (and overall obesity) and WC (and abdominal obesity) could be used to identify the IR status and pancreatic  $\beta$ -cell function in Chinese community-dwelling population without T2DM. BMI (and overall obesity) had similar ability to identify the IR status and pancreatic  $\beta$ -cell function compared with WC (and abdominal obesity). BMI (and overall obesity) and WC (and abdominal obesity) provided similar guidance for clinical doctors to determine the risk of T2DM and the need for further medical check-up before the onset of T2DM.

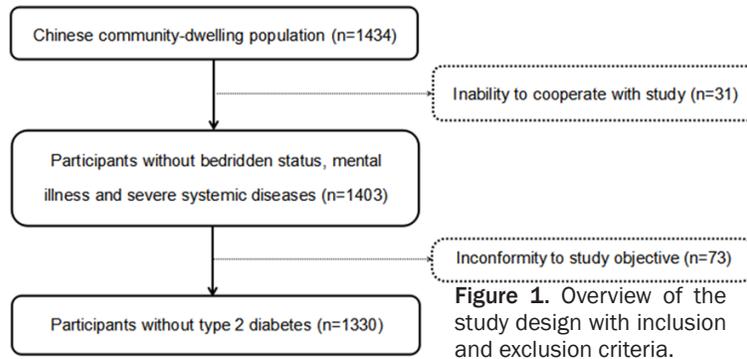
**Keywords:** Overall obesity, insulin resistance, pancreatic  $\beta$ -cell function, abdominal obesity

## Introduction

Just as the prevalence of obesity is increasing, so is the prevalence of type 2 diabetes (T2DM) [1, 2]. Obesity is considered to be a factor that drives the insulin resistance (IR) [3]. IR is an underlying mechanism through which persons develop T2DM [4-7]. Several studies have supported that waist circumference (WC) is an index of abdominal obesity that best identifies the IR status and pancreatic  $\beta$ -cell function [8-10]. However, few studies have been conducted to assess the relationship of body mass index (BMI) with IR status and pancreatic  $\beta$ -cell function. One study has reported that as an index of overall obesity, BMI was negatively related to insulin sensitivity in patients with T2DM [11]. In addition, BMI has been suggest-

ed by another study to be associated with pancreatic  $\beta$ -cell function and endogenous insulin secretion in patients with T2DM [12]. It is not clear whether BMI has similar ability to identify the IR status and pancreatic  $\beta$ -cell function compared with WC in general population without T2DM, and BMI and WC provide similar guidance for clinical doctors to determine the risk of T2DM and the need for further medical check-up before the onset of T2DM. Moreover, due to an ethnic difference in the relationship of anthropometric indice with IR status and pancreatic  $\beta$ -cell function, Chinese may possess different relationship compared with Westerners. It is therefore significant to explore the abilities of BMI (and overall obesity) and WC (and abdominal obesity) to identify the IR status and pancreatic  $\beta$ -cell function in China

## Overall obesity had alike ability to identify IR compared with abdominal obesity



so that preventive measures can be taken before they develop T2DM and its complications. The current analysis was designed to test the hypothesis that BMI (and overall obesity) had similar ability to identify the IR and pancreatic  $\beta$ -cell function compared with WC (and abdominal obesity) in Chinese community-dwelling population without T2DM.

### Methods

#### Study population

In the current analysis, there were 1434 participants of Han origin, 18 years and above, from a large health check-up program in Beijing, China, from May 2007 to July 2009. A stratified cluster sampling design was used in this survey. In the first stage of sampling, three districts (Fengtai, Shijingshan and Daxing) were selected from 18 districts in Beijing. In the second stage of sampling, four communities were selected from these districts. In the third stage of sampling, participants were selected from these communities. Exclusion criteria: 1) the inability to cooperate with study: 31 participants with bedridden status, mental illness and severe systemic diseases were excluded from the current analysis; 2) the inconformity to study objective: 73 participants with T2DM (fasting blood glucose [FBG]  $\geq 7.0$  mmol/L, postprandial blood glucose [PBG]  $\geq 11.1$  mmol/L or taking medications for T2DM) were excluded from the current analysis. Finally, there were 1330 participants without T2DM (**Figure 1**), in the current analysis. The study protocol was approved by Ethics Committee of Chinese People's Liberation Army General Hospital (Beijing, China). Each participant provided written informed consent to be included in the study.

#### Physical examination

Blood pressure was measured with the mercury sphygmomanometer and appropriately sized cuff using the first (systolic blood pressure, SBP) and fifth (diastolic blood pressure, DBP) Korotkoff phases based on a standardized protocol. Two measurements were conducted at one-minute interval after participants

had taken a five-minute break and the mean of two readings was used for the analysis. During the measurements, participants sat in a chair with feet on the floor and arm supported at heart level. Standard methods were used to measure the height, weight and WC, and BMI was calculated through dividing weight (in kilograms) by height (in meters) squared [13]. Participants were categorized as not overall obese or overall obese with BMI of  $28 \text{ kg/m}^2$  as the cutoff point, and not abdominal obese or abdominal obese with WC of 85 cm for men and 80 cm for women as the cutoff points, all in line with Guidelines on Preservation and Control of Overweight and Obesity in Chinese Adults [14].

#### Laboratory inspection

After overnight fasting of at least 12 hours, fasting blood specimens were collected in tubes between 8:00 AM and 10:00 AM by well-trained physicians and processed as appropriate at Department of Biochemistry within the same day of sampling. FBG, triglyceride (TG) and high-density lipoprotein-cholesterol (HDL-c) levels were measured by qualified technicians blinded to clinical data using the enzymatic assays (Roche Products Ltd., Basel, Switzerland) on a full automatic biochemical analyzer (COBAS c6000; Roche Products Ltd, Basel, Switzerland), and low-density lipoprotein-cholesterol (LDL-c) levels were calculated. Oral glucose tolerance test was carried out routinely, and PBG levels were tested 2 hours after the 75-g glucose load. Fasting insulin (FINS) levels were determined by DPC kit (DPC cirrus Inc., Los Angeles, CA, USA) on a fully automatic chemiluminescence analyzer (DPC IMMULITE 1000, DPC cirrus Inc., Los Angeles, CA, USA). HOMA-IR and HOMA- $\beta$  were the representa-

## Overall obesity had alike ability to identify IR compared with abdominal obesity

**Table 1.** Characteristics of participants divided by their IR status and  $\beta$ -cell function expressed as HOMA-IR and HOMA- $\beta$

Characteristics	HOMA-IR $\leq$ 1.37 (n = 665)	HOMA-IR $>$ 1.37 (n = 665)	P value	HOMA- $\beta$ $\leq$ 98.11 (n = 665)	HOMA- $\beta$ $>$ 98.11 (n = 665)	P value
Age (year)	35 (30-52)	41 (32-58)	$<$ 0.001	36 (31-53)	40 (32-59)	0.005
Males (%)	468 (70.4)	462 (69.5)	0.720	474 (71.3)	456 (68.6)	0.282
Smoking (%)	181 (27.2)	196 (29.5)	0.361	168 (25.3)	209 (31.4)	0.013
BMI (kg/m <sup>2</sup> )	23.46 (21.48-25.47)	26.01 (24.22-27.96)	$<$ 0.001	23.74 (21.77-25.71)	25.85 (23.71-27.90)	$<$ 0.001
Overall obesity (%)	39 (5.9)	164 (24.7)	$<$ 0.001	241 (36.2)	494 (74.3)	$<$ 0.001
WC (cm)	81 (73-87)	89 (82-93)	$<$ 0.001	81 (74-88)	88 (81-93)	$<$ 0.001
Abdominal obesity (%)	45 (6.8)	158 (23.8)	$<$ 0.001	264 (39.7)	471 (70.8)	$<$ 0.001
SBP (mmHg)	120 (112-130)	125 (117-135)	$<$ 0.001	121 (113-131)	124 (115-134)	$<$ 0.001
DBP (mmHg)	73 (68-79)	77 (70-83)	$<$ 0.001	74 (68-80)	76 (70-82)	$<$ 0.001
FBG (mmol/L)	4.68 (4.41-4.93)	4.97 (4.68-5.24)	$<$ 0.001	4.92 (4.64-5.22)	4.72 (4.41-5.00)	$<$ 0.001
FINS (mU/L)	4.35 (3.20-5.41)	9.36 (7.60-12.30)	$<$ 0.001	4.61 (3.25-5.91)	9.17 (6.96-12.3)	$<$ 0.001
PBG (mmol/L)	5.06 (4.34-5.99)	5.73 (4.75-6.84)	$<$ 0.001	5.23 (4.41-6.17)	5.52 (4.58-6.52)	0.001
TG (mmol/L)	1.05 (0.80-1.51)	1.50 (1.12-2.13)	$<$ 0.001	1.13 (0.84-1.54)	1.49 (1.03-2.10)	$<$ 0.001
HDL-c (mmol/L)	1.44 (1.22-1.69)	1.24 (1.06-1.50)	$<$ 0.001	1.43 (1.20-1.67)	1.26 (1.07-1.52)	$<$ 0.001
LDL-c (mmol/L)	2.38 (1.95-2.89)	2.52 (2.09-2.99)	0.001	2.38 (1.94-2.88)	2.52 (2.07-2.99)	0.001
HOMA-IR	0.91 (0.67-1.15)	2.05 (1.66-2.69)	$<$ 0.001	1.01 (0.68-1.34)	1.96 (1.41-2.64)	$<$ 0.001
HOMA- $\beta$	72.56 (49.65-95.75)	137.63 (99.38-191.99)	$<$ 0.001	68.68 (48.61-82.01)	148.43 (118.75-201.69)	$<$ 0.001

IR, insulin resistance; HOMA, homeostasis model assessment; BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; FBG, fasting blood glucose; FINS, fasting insulin; PBG, postprandial blood glucose; TG, triglyceride; HDL-c, high-density lipoprotein-cholesterol; LDL-c, low-density lipoprotein-cholesterol.

tives of IR status and pancreatic  $\beta$ -cell function, respectively.  $\text{HOMA-IR} = \text{FINS (mU/L)} \times \text{FBG (mmol/L)} / 22.5$ ;  $\text{HOMA-}\beta = 20 \times \text{FINS (mU/L)} / [\text{FBG (mmol/L)} - 3.5]$  [15].

### Statistical analysis

The current analysis provided the summary statistics as mean with standard deviation for continuous variables with normal distribution, median with interquartile range for continuous variables with skewed distribution, and number with percentage for categorical variables. Comparison of variables between different groups was made using Student's *t* test for continuous variables with normal distribution, Mann-Whitney *U* test for continuous variables with skewed distribution, or  $\chi^2$  analysis for categorical variables. Bivariate correlation was assessed using Pearson (continuous variables with normal distribution) or Spearman (continuous variables with skewed distribution and categorical variables) coefficients. Logistic regression analysis for IR status and pancreatic  $\beta$ -cell function was controlling for age, gender, smoking, BMI (or overall obesity), WC (or abdominal obesity), SBP, DBP, FBG, PBG, TG, HDL-c and LDL-c levels. Receiver-operating characteristic (ROC) curve and area under the curve (c-statistic) were used to compare the abilities of BMI

and WC to identify the IR and pancreatic  $\beta$ -cell function. Statistical analysis was significant if two-sided *p* value was less than 0.05, and conducted using Statistical Package for the Social Science version 17 (SPSS Inc., Chicago, IL, USA).

### Results

Age ranged from 18 to 80 years, with a median of 38 years. Median (interquartile range) of HOMA-IR was 1.37 (0.91-2.05), and that of HOMA- $\beta$  was 98.11 (68.68-148.48). Characteristics of participants divided by their IR status and pancreatic  $\beta$ -cell function expressed as HOMA-IR and HOMA- $\beta$  are shown in **Table 1**. Participants with high IR status had significantly higher age, BMI, WC, SBP, DBP, FBG, FINS, PBG, TG and LDL-c levels, higher percentages of overall and abdominal obesity, and lower HDL-c levels than those with low IR status ( $P < 0.05$  for all). Results for pancreatic  $\beta$ -cell function were similar but there were significantly lower FBG levels and higher percentage of smokers in participants with high  $\beta$ -cell function ( $P < 0.05$  for all).

Correlation coefficients among the variables of interest and IR status and pancreatic  $\beta$ -cell function expressed as HOMA-IR and HOMA- $\beta$

## Overall obesity had alike ability to identify IR compared with abdominal obesity

**Table 2.** Correlation coefficients among the variables of interest and IR status and  $\beta$ -cell function expressed as HOMA-IR and HOMA- $\beta$

Characteristics	HOMA-IR		HOMA- $\beta$	
	Correlation coefficient	P value	Correlation coefficient	P value
Age (year)	0.137	< 0.001	0.092	0.001
Gender (%)	0.001	0.972	0.034	0.211
Smoking (%)	0.027	0.319	0.062	0.023
BMI (kg/m <sup>2</sup> )	0.460	< 0.001	0.365	< 0.001
Overall obesity (%)	0.261	< 0.001	0.236	< 0.001
WC (cm)	0.449	< 0.001	0.364	< 0.001
Abdominal obesity (%)	0.383	< 0.001	0.313	< 0.001
SBP (mmHg)	0.188	< 0.001	0.125	< 0.001
DBP (mmHg)	0.211	< 0.001	0.142	< 0.001
FBG (mmol/L)	0.387	< 0.001	-0.309	< 0.001
PBG (mmol/L)	0.252	< 0.001	0.106	< 0.001
TG (mmol/L)	0.394	< 0.001	0.311	< 0.001
HDL-c (mmol/L)	-0.305	< 0.001	-0.267	< 0.001
LDL-c (mmol/L)	0.106	< 0.001	0.095	0.001

IR, insulin resistance; HOMA, homeostasis model assessment; BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; FBG, fasting blood glucose; PBG, postprandial blood glucose; TG, triglyceride; HDL-c, high-density lipoprotein-cholesterol; LDL-c, low-density lipoprotein-cholesterol.

**Table 3.** Logistic regression analysis including BMI and WC for IR status and  $\beta$ -cell function expressed as HOMA-IR and HOMA- $\beta$

Variables	High HOMA-IR group		High HOMA- $\beta$ group	
	HR (95% CI)	P value	HR (95% CI)	P value
Age (year)	0.993 (0.982-1.004)	0.223	0.990 (0.979-1.001)	0.083
Gender (%)	2.794 (1.947-4.009)	< 0.001	2.751 (1.937-3.907)	< 0.001
Smoking (%)	1.022 (0.755-1.384)	0.886	0.883 (0.656-1.187)	0.408
BMI (kg/m <sup>2</sup> )	1.161 (1.082-1.245)	< 0.001	1.146 (1.071-1.226)	< 0.001
WC (cm)	1.054 (1.028-1.081)	< 0.001	1.055 (1.030-1.081)	< 0.001
SBP (mmHg)	0.998 (0.985-1.012)	0.801	0.992 (0.979-1.005)	0.229
DBP (mmHg)	1.016 (0.997-1.035)	0.108	1.019 (0.999-1.038)	0.058
FBG (mmol/L)	4.106 (3.003-5.615)	< 0.001	0.160 (0.117-0.220)	< 0.001
PBG (mmol/L)	1.089 (0.990-1.198)	0.081	1.135 (1.033-1.247)	0.008
TG (mmol/L)	1.182 (1.026-1.363)	0.021	1.223 (1.043-1.435)	0.013
HDL-c (mmol/L)	0.573 (0.375-0.876)	0.010	0.692 (0.460-1.041)	0.077
LDL-c (mmol/L)	0.808 (0.666-0.982)	0.032	0.942 (0.779-1.141)	0.543

BMI, body mass index; WC, waist circumference; IR, insulin resistance; HOMA, homeostasis model assessment; HR, hazard ratio; CI, confidence interval; SBP, systolic blood pressure; DBP, diastolic blood pressure; FBG, fasting blood glucose; PBG, postprandial blood glucose; TG, triglyceride; HDL-c, high-density lipoprotein-cholesterol; LDL-c, low-density lipoprotein-cholesterol.

are shown in **Table 2**. Age, BMI (and overall obesity), WC (and abdominal obesity), SBP, DBP, FBG, PBG, TG, HDL-c and LDL-c levels were closely related to IR status and pancreatic  $\beta$ -cell function ( $P < 0.05$  for all). And that smok-

ing was closely related to pancreatic  $\beta$ -cell function ( $P < 0.05$  for all).

As shown in **Tables 3** and **4**, Logistic regression analyses indicated that gender, BMI (and overall obesity), WC (and abdominal obesity), FBG and TG levels were independently associated with IR status and pancreatic  $\beta$ -cell function expressed as HOMA-IR and HOMA- $\beta$  ( $P < 0.05$  for all). Additionally, HDL-c levels were independently associated with IR status, while PBG levels were independently associated with pancreatic  $\beta$ -cell function ( $P < 0.05$  for all). C-statistic of BMI to identify the IR status and pancreatic  $\beta$ -cell function had no statistically significant difference with that of WC ( $P > 0.05$  for all; **Table 5**).

### Discussion

The current analysis demonstrated the independent relationship of IR status and pancreatic  $\beta$ -cell function not only with both BMI and WC as the continuous variables, but also with both overall and abdominal obesity in a large Chinese community-dwelling population without T2DM. In addition, BMI (and overall obesity) had similar ability to identify the IR and pancreatic  $\beta$ -cell function compared with WC (and abdominal obesity) in Chinese community-dwelling population without T2DM.

With the change in modern lifestyle and increase in obese population, the prevalence of

## Overall obesity had alike ability to identify IR compared with abdominal obesity

**Table 4.** Logistic regression analysis including overall and abdominal obesity for IR status and  $\beta$ -cell function expressed as HOMA-IR and HOMA- $\beta$

Variables	High HOMA-IR group		High HOMA- $\beta$ group	
	HR (95% CI)	P value	HR (95% CI)	P value
Age (year)	0.991 (0.980-1.002)	0.112	0.989 (0.978-1.000)	0.059
Gender (%)	2.054 (1.476-2.859)	< 0.001	1.961 (1.418-2.712)	< 0.001
Smoking (%)	1.010 (0.746-1.368)	0.948	1.158 (0.861-1.559)	0.333
Overall obesity (%)	2.154 (1.418-3.273)	< 0.001	2.444 (1.626-3.675)	< 0.001
Abdominal obesity (%)	3.555 (2.624-4.816)	< 0.001	3.404 (2.512-4.614)	< 0.001
SBP (mmHg)	1.004 (0.991-1.017)	0.584	0.996 (0.983-1.009)	0.569
DBP (mmHg)	1.018 (0.999-1.037)	0.069	1.021 (1.001-1.040)	0.035
FBG (mmol/L)	4.323 (3.166-5.905)	< 0.001	0.176 (0.129-0.239)	< 0.001
PBG (mmol/L)	1.088 (0.990-1.196)	0.079	1.127 (1.026-1.238)	0.013
TG (mmol/L)	1.191 (1.029-1.379)	0.019	1.221 (1.031-1.422)	0.019
HDL-c (mmol/L)	0.481 (0.318-0.728)	0.001	0.581 (0.388-0.870)	0.008
LDL-c (mmol/L)	0.840 (0.692-1.019)	0.077	0.975 (0.806-1.178)	0.791

IR, insulin resistance; HOMA, homeostasis model assessment; HR, hazard ratio; CI, confidence interval; SBP, systolic blood pressure; DBP, diastolic blood pressure; FBG, fasting blood glucose; PBG, postprandial blood glucose; TG, triglyceride; HDL-c, high-density lipoprotein-cholesterol; LDL-c, low-density lipoprotein-cholesterol.

**Table 5.** C-statistic of BMI and WC to identify the IR status and  $\beta$ -cell function expressed as HOMA-IR and HOMA- $\beta$

Variables	C-statistic (95% CI)	P value	P value
HOMA-IR BMI	0.732 (0.705-0.758)	< 0.001	0.614 <sup>a</sup>
Waist	0.722 (0.695-0.749)	< 0.001	
HOMA- $\beta$ BMI	0.679 (0.651-0.708)	< 0.001	0.962 <sup>b</sup>
Waist	0.678 (0.649-0.706)	< 0.001	

Notes: <sup>a</sup>P value was drew from comparisons between c-statistic of BMI and WC to identify the IR status expressed as HOMA-IR; <sup>b</sup>P value was drew from comparisons between c-statistic of BMI and WC to identify the  $\beta$ -cell function expressed as HOMA- $\beta$ . BMI, body mass index; WC, waist circumference; CI, confidence interval; IR, insulin resistance; HOMA, homeostasis model assessment.

T2DM has been increasing all around the world [1, 2]. IR is strongly correlated with obesity and leads to the development of T2DM [4-7]. Obesity is considered to be an important determinant for the development of IR and a contributing factor of pancreatic  $\beta$ -cell function abnormality [3]. Increased secretion of free fatty acids, inflammatory cytokines and decreased secretion of adiponectin mediate the relationship between obesity and IR [16, 17]. IR causes the hyperglycemia through the suppression of glucose uptake in skeletal muscle and increase in hepatic glucose production [18].

Several studies have discussed the relationship of WC with IR or hyperinsulinemia [8, 9].

One study has reported a linear increase in the prevalence of hyperinsulinemia across the deciles of WC in 185 Canadian men [8]. Another study has realized that WC was strongly correlated with HOMA-IR [9]. In the identification of IR status and pancreatic  $\beta$ -cell function, more emphasis has been placed on WC, whereas little attention has been paid to BMI [10]. One study has suggested that BMI was strongly correlated with decreased insulin sensitivity in Korean with T2DM [11]. In addition, BMI has been considered by another study to be closely correlated to pancreatic  $\beta$ -cell function and endogenous insulin secretion in Japanese with T2DM [12]. Whether BMI has similar ability in the identification of IR status and pancreatic  $\beta$ -cell function compared with WC in general population, especially Chinese community-dwelling population without T2DM, has been a matter of controversy. It is evident in the current analysis that both BMI (and overall obesity) and WC (and abdominal obesity) were independently associated with IR status and pancreatic  $\beta$ -cell function. Moreover, the current study highlighted that BMI (and overall obesity) had similar ability to identify the IR and pancreatic  $\beta$ -cell function compared with WC (and abdominal obesity) in Chinese community-dwelling population without T2DM.

### Conclusions

Based on the current analysis, both BMI (and overall obesity) and WC (and abdominal obesi-

## Overall obesity had alike ability to identify IR compared with abdominal obesity

ty) could be used to identify the IR status and pancreatic  $\beta$ -cell function in Chinese community-dwelling population without T2DM. Moreover, the current analysis supported that BMI (and overall obesity) had similar ability to identify the IR status and pancreatic  $\beta$ -cell function compared with WC (and abdominal obesity). BMI (and overall obesity) and WC (and abdominal obesity) provided similar guidance for clinical doctors to determine the risk of T2DM and the need for further medical check-up before the onset of T2DM.

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### Disclosure of conflict of interest

None.

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### References

- [1] Ogden CL, Carroll MD, Kit BK and Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011-2012. *JAMA* 2014; 311: 806-814.
- [2] Xu Y, Wang L, He J, Bi Y, Li M, Wang T, Wang L, Jiang Y, Dai M and Lu J. Prevalence and control of diabetes in Chinese adults. *JAMA* 2013; 310: 948-959.
- [3] Graf C and Ferrari N. Metabolic syndrome in children and adolescents. *Pediatr Diabetes* 2007; 85: 79-81.
- [4] Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. *Nutrition* 1997; 13: 64.
- [5] Osei K, Rhinesmith S, Gaillard T and Schuster D. Impaired insulin sensitivity, insulin secretion, and glucose effectiveness predict future development of impaired glucose tolerance and type 2 diabetes in pre-diabetic African Americans: implications for primary diabetes prevention. *Diabetes Care* 2004; 27: 1439-1446.
- [6] Abdul-Ghani MA, Williams K, Defronzo RA and Stern M. What is the best predictor of future type 2 diabetes? *Diabetes Care* 2007; 30: 1544-1548.
- [7] Defronzo RA. Lilly lecture 1987. The triumvirate: beta-cell, muscle, liver. A collusion responsible for NIDDM. *Diabetes* 1988; 37: 667-687.
- [8] Lemieux I, Pascot A, Couillard C, Lamarche B, Tchernof A, Alméras N, Bergeron J, Gaudet D, Tremblay G and Prud'Homme D. Hypertriglyceridemic waist: a marker of the atherogenic metabolic triad (hyperinsulinemia; hyperapolipoprotein B; small, dense LDL) in men? *Circulation* 2000; 102: 179-184.
- [9] Wahrenberg H, Hertel K, Leijonhufvud BM, Persson LG, Toft E and Arner P. Use of waist circumference to predict insulin resistance: retrospective study. *BMJ* 2005; 330: 1363-1364.
- [10] Björntorp P. Adipose tissue distribution, plasma insulin, and cardiovascular disease. *Diabetes Metab* 1987; 13: 381-385.
- [11] Chang SA, Kim HS, Yoon KH, Ko SH, Kwon HS, Kim SR, Lee WC, Yoo SJ, Son HS and Cha BY. Body mass index is the most important determining factor for the degree of insulin resistance in non-obese type 2 diabetic patients in Korea. *Metabolism* 2004; 53: 142-146.
- [12] Funakoshi S, Fujimoto S, Hamasaki A, Fujiwara H, Fujita Y, Ikeda K, Hamamoto Y, Hosokawa M, Seino Y and Inagaki N. Analysis of factors influencing pancreatic beta-cell function in Japanese patients with type 2 diabetes: association with body mass index and duration of diabetic exposure. *Diabetes Res Clin Pract* 2008; 82: 353-358.
- [13] Pisunyer FX. NHLBI obesity education initiative expert panel on the identification, evaluation, and treatment of overweight and obesity in adults-the evidence report. *Obes Res* 1998; 6: 51S-209S.
- [14] Chen C and Lu FC. The guidelines for prevention and control of overweight and obesity in Chinese adults. *Biomed Environ Sci* 2004; 17: 1-36.
- [15] Wallace TM, Levy JC and Matthews DR. Use and abuse of HOMA modelling. *Diabetes Care* 2004; 27: 1487-1495.
- [16] Boden G, Shulman GI, Boden G and Shulman GI. Free fatty acids in obesity and type 2 diabetes: defining their role in the development of

## Overall obesity had alike ability to identify IR compared with abdominal obesity

- insulin resistance and B-cell dysfunction. *Eur J Clin Invest* 32, S14-S23. *Eur J Clin* 2002; 32: 14-23.
- [17] Matsuzawa Y. The metabolic syndrome and adipocytokines. *FEBS Lett* 2006; 580: 2917-2921.
- [18] Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. *Nutrition* 1997; 13: 64.