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Higher ratio of triglyceride to high-density lipoprotein cholesterol may predispose to diabetes mellitus: 15-year prospective study in a general population

Sen He[a,1], Si Wang[a,1], Xiaoping Chen[a,*], Lingyun Jiang[b], Yong Peng[a], Longxin Li[a], Liyan Wan[a], Kaijun Cui[a]

[a] Department of Cardiovascular Medicine, West China Hospital, Sichuan University, Chengdu 610041, China
[b] Department of Internal Medicine, West China Hospital, Sichuan University, Chengdu 610041, China

ARTICLE INFO

Article history:
Received 11 December 2010
Accepted 4 May 2011

ABSTRACT

The aims of the study were to examine whether the triglyceride to high-density lipoprotein cholesterol ratio (TG/HDL-C) could predict future diabetes mellitus (DM) in a general population during a 15-year follow-up. The data were collected in 1992 and then again in 2007 from the same group of 711 individuals. Because 24 of them were found to be diabetic in 1992, our analysis was eventually based on the usable data collected from the remaining 687 individuals (male, 58.1%). During the period 1992-2007, 74 individuals were found to have developed DM (10.8%). After adjusting the associated variables, it was found that TG and TG/HDL-C were independent DM risk factors, with the odds ratios being 1.292 ($P = .047$) and 1.341 ($P = .010$), respectively, although they were poor in their DM discriminatory power (area under the receiver operating characteristic curve, 0.662 and 0.672, respectively). Combined with other risk factors (fasting plasma glucose, waist circumference, and family history of DM), the DM discriminatory power of TG and TG/HDL-C was improved (area under the receiver operating characteristic curve, 0.764 and 0.767, respectively). The DM incidence increased with ascending risk score. Single HDL-C seems unable to predict future DM. Triglycerides and TG/HDL-C were independent DM risk factors; and of the two, TG/HDL-C was a stronger risk factor. The DM discriminatory power of TG and TG/HDL-C was poor; therefore, it is recommended that they be used in combination with other risk factors. Diabetes mellitus incidence increased with ascending risk score.

1. Introduction

Several studies [1,2] have shown that diabetes mellitus (DM) is an important predictor for death, especially for cardiovascular diseases; and DM is recognized as a major global public health problem. More than 135 million people have DM worldwide, and the number keeps growing [3]. It has been predicted that the number of diabetic patients would increase to 300 million in 2025 [4]. Inevitably, DM and its complications will emerge as one of the major threats to future public

Author contributions: design of the study: Sen He, Xiaoping Chen; conduct of the study: Xiaoping Chen, Kaijun Cui; data collection: Longxin Li, Liyan Wan, Sen He; data analysis: Sen He, Si Wang; data interpretation: Lingyun Jiang, Yong Peng; manuscript writing: Sen He, Xiaoping Chen.

* *Corresponding author*. Tel.: +86 028 85422343; fax: +86 028 85422175.
E-mail address: happensky@163.com (X. Chen).

[1] Sen He and Si Wang equally contributed to the article.

0026-0495/$ – see front matter © 2012 Elsevier Inc. All rights reserved.
doi:10.1016/j.metabol.2011.05.007

health resources throughout the world, particularly in developing countries.

There is no cure for DM; thus, prevention is the best intervention. Identifying individuals who have a high risk of developing DM is potentially of significant benefit if preventive measures are used. Overweight, habitual physical inactivity, smoking, drinking, and dyslipidemia are the traditional risk factors for DM. Among these risk factors, dyslipidemia, especially high triglyceride (TG) and low high-density lipoprotein cholesterol (HDL-C), is a common feature in prediabetic and diabetic states [5]. Recently, some surveys [6-8] showed that, in some populations, TG and TG/HDL-C were positively related to insulin resistance (IR), which was negatively related to HDL-C, and TG/HDL-C had better predictive ability of IR than TG or HDL-C alone [6-8]. Some studies [9-13] have shown that higher TG levels were an independent risk factor for developing DM, as well as lower HDL-C [9,10,13,14]. However, currently, it is uncertain whether TG/HDL-C could predict future DM better than TG or HDL-C on their own. Moreover, these previous studies focused mainly on non-Chinese populations. Therefore, the main purposes of the study were to assess the DM predictive power of TG/HDL-C, as well as that of TG and HDL-C on their own, based on the follow-up data over 15 years collected from a general Chinese group.

2. Methods

2.1. Study population

In 2007, health examinations were performed on 711 individuals in an urban community located in Chengdu, Sichuan province, China. These individuals were a part of a study supported by megaprojects of science research for China's 11th 5-year plan (trends in the incidence of metabolic syndrome and integrated control in China). These individuals also accepted health examinations in 1992 for cardiovascular disease (CVD) risk factors, which were supported by a project from China's eighth national 5-year research plan (the Chinese multiprovincial cohort study); therefore, we picked up the data of these individuals in 1992. The detailed information of these participants has been reported elsewhere [15,16]. Because 24 participants had DM in 1992, they were excluded from the analysis. Therefore, the complete data of 687 individuals (male, 58.1%) were available for analysis.

2.2. Data collection

In 1992, medical professionals did a survey of CVD risk factors according to the Multinational MONItoring of trends and determinants in CArdiovascular disease (MONICA) protocol [17]. The survey content included standardized questionnaire, physical examination, and laboratory tests. The questionnaire included sex, age, and CVD risk factors, such as smoking status, alcohol consumption levels, physical activity, and family history of CVD. The physical examination included measurement of blood pressure, height, weight, waist circumference, hip circumference, and so on. Laboratory tests included measurement of fasting plasma glucose (FPG), fasting serum total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), HDL-C, and TG. Blood was drawn from the antecubital vein in the morning after 12-hour fasting. These biochemistry parameters were measured at the laboratory of West China Hospital (Chengdu, China). In 2007, we repeated a survey of these participants with the same methods. This study was approved by the Ministry of Health of China, as well as by the Ethics Committee of West China Hospital of Sichuan University. The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a priori approval by the institution's human research committee. All participants provided written informed consent.

2.3. Related definitions

Those with *hypertension* were defined as having systolic blood pressure (SBP) of at least 140 mm Hg and/or diastolic blood pressure (DBP) of at least 90 mm Hg and/or currently taking antihypertensive medications. *Diabetes mellitus* was defined by self-reported history or a fasting plasma glucose of at least 7.0 mmol/L. *Smoking* was defined as average cigarette consumption of at least 1 per day. *Alcohol intake* was defined as average intake of alcohol of at least 50 g/d. *Physical activity* was defined as exercise one or more times per week, at least 20 minutes for each time.

2.4. Statistical analysis

Data are presented as mean ± SD. Smoking, alcohol intake, physical activity, hypertension, and family history of DM were used as dummy variables. Comparisons between groups were performed by paired or independent t test for normally distributed variables and by the nonparametric Mann-Whitney or Wilcoxon test for skewed variables. Interactions between categorical variables were evaluated with Pearson χ^2 test or McNemar test. To assess the effects of TG, HDL-C, and TG/HDL-C on the new onset of DM, 3 logistic regression models were used to estimate the odds ratios (ORs) of them. To estimate whether combinations with other variables could improve the DM discriminatory power of TG and TG/HDL-C, parsimonious logistic regression models were constituted of variables that could predict future DM independently. We assessed the goodness of fit of all models by using the Hosmer-Lemeshow test. The area under the receiver operating characteristic curve (AROC) quantified the discrimination between diabetic and nondiabetic participants. The AROCs were presented with 95% confidence interval (CI): AROC of 0.5 = no discrimination, AROC of at least 0.7 but less than 0.8 = acceptable, AROC of at least 0.8 but less than 0.9 = excellent, AROC of at least 0.9 = outstanding [18]. We also set the optimal cutoff point, which represents the optimal combination of sensitivity and specificity for the study sample. Sensitivity and specificity were calculated by using 2 × 2 tables. Likelihood ratios were calculated as the ratios of sensitivity/(1 – specificity) (positive likelihood ratio) and (1 – sensitivity)/specificity (negative likelihood ratio). We used χ^2 test for trend to estimate the cumulative incidence across the different score groups, and the score values were derived from the original β coefficients of these logistic models. For statistical analysis, a combination of the MedCalc (version 8.2.1.0) and SPSS (version 10.0; SPSS, Chicago, IL) software was used. Statistical significance was defined as $P < .05$.

3. Results

3.1. *Demographic data of the population at baseline (1992) and at the end of follow-up (2007)*

Table 1 presents the demographic data of the 687 participants in 1992 and 2007. During a 15-year follow up, SBP and DBP were significantly higher in 2007 than in 1992, as well as FPG, TC, LDL-C, HDL-C, LDL-C/HDL-C, body mass index (BMI), and waist and hip circumference; however, TG and TG/HDL-C decreased. There were an increase in the proportion of physical activity and a reduction in smoking, and alcohol intake did not change. The percentage of subjects with hypertension increased from 15.1% in 1992 to 50.9% in 2007, and that of DM increased from 0.0% to 10.8%.

3.2. *Baseline characteristics according to DM status during the follow-up*

Table 2 presents the demographic data of the 687 participants in 1992, stratified according to the subsequent diabetic patients or not. Age, BMI, SBP, FPG, TC, TG, TG/HDL-C, waist circumference, hip circumference, and family history of DM were statistically significantly greater in the subsequent diabetic group; and HDL-C was statistically significantly lower. Other variables did not differ between the 2 groups.

3.3. *Logistic regression models for prediction of DM in different models*

The univariate logistic regression analysis presented that many variables could statistically predict future DM (Table 3), whereas in the multivariate logistic regression models, most variables could not predict future DM independently. In model 1, entering age, sex, SBP, FPG, LDL-C, TG, HDL-C, waist circumference, hip circumference, and family history of DM, only the ORs of FPG, TG, waist circumference, and family history of DM were statistically significant (Table 3). We entered age, sex, SBP, FPG, TG, LDL-C/HDL-C, waist circumference, hip circumference, and family history of DM into model 2; and the variables that could independently predict future DM were similar to those of model 1 (Table 3). In model 3, excepting FPG, waist circumference, and family history of DM, TG/HDL-C also could independently predict future DM (OR, 1.341; $P = .010$) (Table 3). Finally, after adjustment for other associated parameters, only 5 variables (FPG, TG, waist circumference, family history of DM, and TG/HDL-C) could independently predict future DM.

Table 1 – Demographic data of the population at baseline (1992) and at the end of follow-up (2007)

Variable	1992	2007	*P* value
Age (y)	48.1 ± 6.2	63.1 ± 6.2	<.001
Sex (male)	399 (58.1)	399 (58.1)	NA
BMI (kg/m^2)	23.4 ± 2.8	23.6 ± 3.2	.004
SBP (mm Hg)	114.5 ± 15.3	134.8 ± 19.1	<.001
DBP (mm Hg)	73.7 ± 9.1	79.1 ± 10.0	<.001
FPG (mmol/L)	4.3 ± 0.7	5.0 ± 1.6	<.001
TC (mmol/L)	4.5 ± 0.8	4.9 ± 0.9	<.001
TG (mmol/L)	2.1 ± 1.0	1.9 ± 1.5	<.001
LDL-C (mmol/L)	2.3 ± 0.8	3.0 ± 0.8	<.001
HDL-C (mmol/L)	1.2 ± 0.2	1.4 ± 0.3	<.001
LDL-C/HDL-C	1.9 ± 0.9	2.1 ± 0.6	<.001
TG/HDL-C	1.8 ± 1.0	1.5 ± 2.1	<.001
WC (cm)	76.5 ± 7.9	82.6 ± 10.5	<.001
HC (cm)	92.2 ± 5.8	94.8 ± 8.2	<.001
Smoking	248 (36.1)	184 (26.8)	<.001
Alcohol intake	87 (12.7)	87 (12.7)	NA
Physical activity	146 (21.2)	401 (58.4)	<.001
Hypertension	104 (15.1)	350 (50.9)	<.001
Family history of DM	26 (3.8)	26 (3.8)	NA
DM	0	74 (10.8)	<.001

Data are means ± SD or number (percentage). WC indicates waist circumference; HC, hip circumference.

Table 2 – Baseline characteristics of the population according to DM status at follow-up

Variable	Subsequent diabetic patients (n = 74)	Subsequent nondiabetic patients (n = 613)	*P* value
Age (y)	49.8 ± 5.7	47.9 ± 6.2	.013
Sex (male)	48 (64.9)	351 (57.3)	.210
BMI (kg/m^2)	25.1 ± 3.3	23.2 ± 2.6	<.001
SBP (mm Hg)	118.9 ± 18.2	114.0 ± 14.9	.021
DBP (mm Hg)	75.7 ± 9.6	73.4 ± 9.0	.095
FPG (mmol/L)	4.6 ± 0.8	4.2 ± 0.7	<.001
TC (mmol/L)	4.7 ± 0.7	4.5 ± 0.8	.023
TG (mmol/L)	2.6 ± 1.2	2.1 ± 0.9	<.001
LDL-C (mmol/L)	2.3 ± 0.9	2.3 ± 0.8	.776
HDL-C (mmol/L)	1.18 ± 0.24	1.25 ± 0.24	.007
LDL-C/HDL-C	2.04 ± 0.95	1.89 ± 0.85	.149
TG/HDL-C	2.29 ± 1.22	1.73 ± 0.95	<.001
WC (cm)	82.0 ± 8.4	75.9 ± 7.6	<.001
HC (cm)	94.9 ± 7.1	91.9 ± 5.6	<.001
Smoking	32 (43.2)	216 (35.2)	.176
Alcohol intake	12 (16.2)	75 (12.2)	.331
Physical activity	14 (18.9)	132 (21.5)	.604
Hypertension	16 (21.6)	88 (14.4)	.099
Family history of DM	6 (8.1)	20 (3.3)	.039

Data are means ± SD or number (percentage).

3.4. *AROCs for different models and cumulative incidence of DM in different score groups*

In these models predicting newly detected DM (Table 4), TG or TG/HDL-C alone had poor discriminatory power compared with the model 4 (AROCs, 0.662, 0.672 vs 0.747, respectively). Inclusion of either TG (model 4) or TG/HDL-C status (model 6) in model 4 improved the discriminatory power, and the AROCs increased from 0.747 in model 4 to 0.764 in model 5 ($P = .0242$) and to 0.767 in model 6 ($P = .0144$) (Table 4). For other variables, the AROCs for new DM were 0.627 (95% CI, 0.556-0.697) for FPG and 0.701 (95% CI, 0.641-0.760) for waist, respectively. The optimal cutoff points to predict future DM were the following values: –2.52 for model 4, –2.56 for model 5, and –2.31 for model 6, respectively; and other parameters for

Table 3 – Univariate and multivariate logistic regression models for prediction of DM in different models

Variable	Univariate regression		Model 1		Model 2		Model 3	
	OR	*P* value	OR	*P* value	OR	*P* value	OR	*P* value
Age (y)	1.051	.014	1.013	.591	1.011	.654	1.013	.599
Sex (1 = male, 2 = female)	0.726	.216	1.025	.941	1.009	.978	1.013	.968
SBP (mm Hg)	1.019	.010	1.008	.341	1.008	.362	1.008	.360
FPG (mmol/L)	1.900	<.001	1.879	<.001	1.866	<.001	1.874	<.001
LDL-C (mmol/L)	1.043	.776	1.168	.365			1.181	.286
TG (mmol/L)	1.452	<.001	1.292	.047	1.337	.012		
HDL-C (mmol/L)	0.258	.014	0.460	.189				
WC (cm)	1.103	<.001	1.111	<.001	1.111	<.001	1.112	<.001
HC (cm)	1.090	<.001	0.966	.344	0.967	.362	0.966	.337
Family history of DM	2.616	.046	3.325	.035	3.235	.035	3.269	.036
LDL-C/HDL-C	1.459	<.001			1.267	.093		
TG/HDL-C	1.490	<.001					1.341	.010

Model 1 included age, sex, SBP, FPG, LDL-C, TG, HDL-C, waist, hip, and family history of DM. Model 2 included age, sex, SBP, FPG, TG, LDL-C/HDL-C, waist, hip, and family history of DM. Model 3 included age, sex, SBP, FPG, LDL-C, TG/HDL-C, waist, hip, and family history of DM.

these optimal cutoff points are shown in Table 5. The total scores ranged between 0.6 and –5.2 for the 3 models, and the footnotes of Table 4 are instructions for these scoring models. We categorized these scores into 4 groups (group 1 is <–3, group 2 is between –2 to –3, group 3 is between –1 to –2, group 4 is >–1) so that we could estimate the cumulative incidence of DM in different groups. Fig. 1 shows that increasing score in 1992 led to an increase in the cumulative incidence of DM during the follow-up (trend $P < .001$). Compared with the reference group (group 1), ORs (95% CI) were 2.740 (2.076-3.616) for model 4, 2.797 (2.112-3.706) for model 5, and 2.579 (1.954-3.405) for model 6.

4. Discussion

Our goal was to assess whether TG, HDL-C, and TG/HDL-C could predict future DM on the basis of data collected from a general Chinese group during 15 years of follow-up. Our findings showed that TG and TG/HDL-C were independent DM risk factors and that TG/HDL-C was a stronger risk factor than TG. However, the 2 variables had poor discriminatory power for DM; and it is recommended that they be used in combination with other risk factors. The DM incidence increased with ascending risk score.

At baseline, the demographic data of the subsequent diabetic patients showed that some traditional risk factors, such as age, BMI, FPG, TG, waist circumference, and family history of DM, were statistically higher than the subsequent nondiabetic patients and that HDL-C was statistically significantly lower. All these might be the causes of the different incidence of DM between the 2 groups. Identifying independent risk factors among them is important for us to offer preventive measures. With the years gone, the demographic data showed that most traditional DM risk factors were statistically greater during the follow-up, such as age, BMI, FPG, waist circumference, and prevalence of hypertension. On the other hand, some traditionally protective factors were enhanced, including decreased TG and increased HDL-C. Shigetoh et al [19] also showed similar results. Although

Table 4 – Area under the receiver operating characteristic curves of different predictive models

Variable	Model 4	Model 5	Model 6	Model with TG only	Model with TG/HDL-C only
TG		1.252 (1.023-1.533)		1.452 (1.196-1.761)	
TG/HDL-C			1.286 (1.053-1.570)		1.490 (1.227-1.808)
FPG	1.943 (1.391-2.715)	1.920 (1.373-2.685)	1.924 (1.377-2.689)		
WC	1.105 (1.069-1.143)	1.098 (1.061-1.136)	1.097 (1.060-1.135)		
Family history	2.538 (0.916-7.037)	2.629 (0.940-7.352)	2.671 (0.947-7.533)		
AROC	0.747 (0.691-0.803)	0.764 (0.710-0.818)	0.767 (0.712-0.821)	0.662 (0.594-0.730)	0.672 (0.605-0.738)
Goodness of fit (*P* value)	.789	.811	.528	.223	.133

Data are OR (95% CI) unless otherwise indicated. Model 4 score = –13.009 + 0.931 * (family history of DM) + 0.100 * waist circumference + 0.664 * FPG. Model 5 score = –12.922 + 0.966 * (family history of DM) + 0.093 * waist circumference + 0.225 * TG + 0.652 * FPG. Model 6 score =–12.824 + 0.983 * (family history of DM) + 0.092 * waist circumference + 0.252 * TG/HDL-C + 0.654 * FPG. For each model, FPG in millimoles per liter, TG in millimoles per liter, WC in centimeters, family history of DM: 1 if at least one parent or sibling has DM or 0 if not.

Table 5 – Prediction score with test characteristics for the future DM

Models	Optimal cutoff points	Sensitivity (%)	Specificity (%)	+LR	–LR
Model 4	–2.52	83.8	56.1	1.90	0.29
Model 5	–2.56	83.4	55.3	1.87	0.30
Model 6	–2.31	74.3	61.7	1.94	0.42

+LR indicates positive likelihood ratio; –LR, negative likelihood ratio.

overall risk factors were still more than protective factors, we should pay more attention to these individuals.

For these lipidic variables, including LDL-C, TG, HDL-C, LDL-C/HDL-C, and TG/HDL-C, the univariate logistic regression analysis showed that most of these variables could statistically predict future DM (Table 3). However, after adjusting for associated factors, they could not independently predict future DM, except TG and TG/HDL-C. In our study, LDL-C could not independently predict future DM; and this has been proven by some studies [20-22]. Some studies [9,10,13,14] have shown that lower HDL-C was an independent risk factor for developing DM, but we could not draw similar results. This may be due to interethnic differences in lipid profiles and/or to some differences of lifestyles. The relatively small sample size might be another reason. No matter if HDL-C could predict future DM statistically, both the previous results and our results showed that lower HDL-C levels could increase the risk for DM. Therefore, the combination of TG and HDL-C (TG/HDL-C) may have greater prediction ability than measurement of single TG or HDL-C. There are some possible mechanisms whereby the combination of TG and HDL-C may be better than the measurement of single TG or HDL-C. Firstly, when TG persists at high levels, heparin activates lipoprotein lipase to increase intravascular lipolysis of circulating TG, thus increasing tissue exposure to free fatty acids (FFAs). High FFAs may deteriorate insulin sensitivity. Several studies [23-25] have

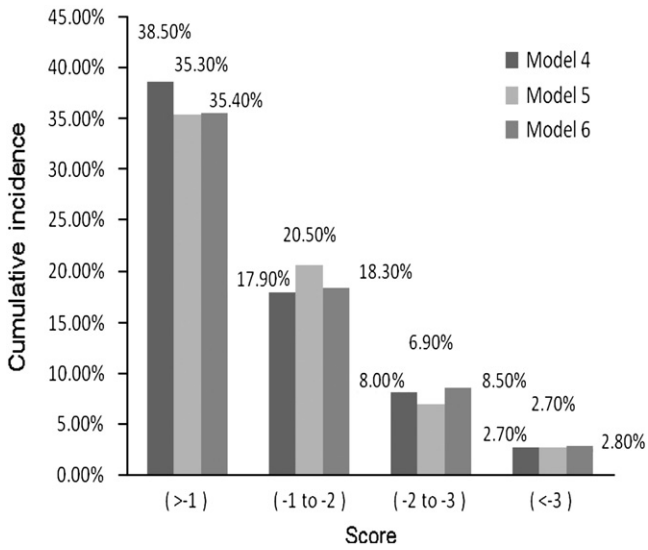

Fig. 1 – Cumulative 15-year DM incidence in different score groups.

reported that raising plasma FFA levels can develop peripheral tissue IR. Induction of tissue oxidative stress by long-chain FFAs may lead to tissue IR [26]. Secondly, oxidation and inflammation can predict IR. High-density lipoprotein cholesterol has the ability of antioxidation and anti-inflammation, so decreasing HDL-C may further lead to IR. The combination of TG and HDL-C may have stronger predictability for IR and, furthermore, for DM. Although it is unclear whether intervention of plasma TG and HDL-C can improve the insulin sensitivity, further decreasing the incidence of DM, a large observational study has given us some hope that using fibrates, drugs that could obviously decrease TG levels and increase HDL-C levels, can lower hazards for incidence of DM [27]. However, another study [28] showed that fenofibrate (a kind of fibrate) could not change intramuscular triglyceride saturation, which was closely related to insulin sensitivity. More studies may be needed to clarify the issue.

Along with TG and TG/HDL-C, there were some similar characteristics of other results: higher risk of developing DM and poor discriminatory power. Higher FPG concentration at baseline may predict future DM in our study, and other reports [9,10,20-22] have similarly shown that blood glucose is a strong risk factor for DM. The discriminatory power was poor in our study (blood glucose only; AROC, 0.627), and the discriminatory power was also inconsistent in the previous studies (blood glucose only; AROCs, 0.548-0.814) [20,29,30]. Further studies may be needed to clarify the issue. In our study, increased waist circumference was associated with substantially increased risk of developing DM, similar to some studies [10,20]. Body mass index and waist circumference were highly correlated. If we put the two of them in the models, it would be overadjusted in these models. Finally, we only put waist circumference in the models. The reasons are as follows: Firstly, Asian populations are more prone to abdominal obesity and low muscle mass with increased IR [31]; and waist circumference, reflecting central obesity, is a useful measure of obesity-related risk of DM, especially in individuals with normal BMI values [32]. Secondly, our population had a lower baseline BMI (average, 23.4 kg/m^2; Table 1); and the effect of waist circumference might be greater than BMI. Therefore, we put waist circumference in the models. Waist circumference was a strong risk factor in our study; and the discriminatory power might be acceptable [18], even better than FPG, TG, and TG/HDL-C (AROCs: 0.701 vs 0.627, 0.662 and 0.672, respectively). Having a parent with DM also contributed strongly to DM risk in our study, similar to the previous studies [9,10,20-22].

Although TG and TG/HDL-C were strong risk factors for DM, the DM discriminatory power was poor. It might be inappropriate to use TG or TG/HDL-C alone to predict future DM, and combination with other risk factors could improve the DM discriminatory power (Table 4). Several studies [9,10,13,25,29,33] have developed some scoring models in different populations, and the DM discriminatory power of these models was better than a single marker. Although combination with different risk factors could improve the discriminatory power, the premise is that we could identify every significant risk factor. Further studies are needed and warranted.

Our results have several strengths and weaknesses. To our knowledge, this might be the first study showing that TG/HDL-C could predict future DM more effectively than either TG or HDL-C alone. The TG/HDL-C ratio might be a new marker of developing DM and, as such, perhaps deserves more attention in clinical practice. However, given that a single variable has limited discriminatory power, it should be used in combination with different variables. Several potential limitations of this study should be mentioned. Firstly, the absence of an oral glucose tolerance test means that some individuals would have developed DM that was not detectable by changes in fasting glucose alone or by clinical history. However, oral glucose tolerance tests for all participants were not feasible for pragmatic reasons and logistics. Secondly, the levels of circulating TG are highly influenced by the fed-fasted state and lifestyles; and related factors cannot be completely controlled in the study. However, blood samples were drawn after a 12-hour fast; and this may improve the results. Thirdly, because of the relatively small sample size, the results of our study may have limited statistical power; but we still can get some clues. There is still another limitation: there were no comparisons between different races.

In summary, our findings showed that TG and TG/HDL-C were independent DM risk factors and that TG/HDL-C was a stronger risk factor than TG. The discriminatory power of TG or TG/HDL-C was poor for DM. Combination with other risk factors could improve the discriminatory power, and this should be recommended. Future researches may be warranted to assess whether decreasing TG and TG/HDL-C could prevent future DM in a large population, as well as other risk factors.

Acknowledgment

This study was supported by a project from China's eighth national 5-year research plan (grant 85-915-01-02) and by megaprojects of science research for China's 11th 5-year plan (grant 2006BAI01A01).

Conflict of Interest

No conflict of interest exists in the submission of this manuscript.

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