Original Article

Dietary glycemic load and metabolic status in newly diagnosed type 2 diabetes in southeastern China

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Background and Objectives: Large-scale epidemiological investigations worldwide have shown that dietary glycemic load is associated with metabolic diseases, including diabetes. However, only a few studies have examined the correlations between glycemic load and blood glucose and lipids in Chinese diabetic patients. Therefore, this study aimed to determine these correlations in southeastern China. Methods and Study Design: 201 patients with newly diagnosed type 2 diabetes and 126 participants with normal blood glucose were enrolled at the Sun Yat-sen Memorial Hospital, Guangdong Province. Carbohydrate intake and glycemic load were assessed based on 3-day dietary records. Using glycemic load as the dependent variable, a correlation analysis and multiple regression analyses were used to analyze the correlations between glycemic load and blood glucose and lipids. Results: The mean glycemic load in diabetic patients was significantly higher than that in the control group (p < 0.05). Correlation analysis showed that glycemic load was positively correlated with body mass index and glycated hemoglobin in diabetic patients (p < 0.05) but negatively correlated with high-density lipoprotein cholesterol in all subjects (p < 0.05). Multivariable regression analysis indicated that, among participants in southeastern China, a higher glycemic load increased the odds of having diabetes, a low high-density lipoprotein cholesterol, and higher Charlson weighted index of comorbidities score, as well as being overweight. Conclusions: A high-glycemic load diet may be associated with a risk of diabetes, glycemic control, lipid metabolism, prognosis of diseases, and body composition. It is necessary to control dietary glycemic load for both patients with diabetes and healthy people in southeastern China.

Key Words: type 2 diabetes mellitus, Chinese, glycemic load, blood glucose, blood lipid

INTRODUCTION

Diabetes is a major risk factor for morbidity and mortality worldwide; complications include ischemic heart disease, stroke, chronic kidney disease, blindness, amputation, and cancer.¹ China has one of the highest diabetes prevalence in Asia and the largest absolute burden of diabetes worldwide. Indeed, the most current epidemiological investigation showed that approximately 11.6% of Chinese adults may have diabetes and 50.1% may have prediabetes.² Therefore, preventing the development of diabetes and improving glycemic control among patients with diabetes in China are of great importance.

The dietary glycemic index (GI) was introduced to quantify the glycemic response to carbohydrates in different foods. The dietary glycemic load (GL), which is calculated by multiplying a food's GI by its carbohydrate content (representing both carbohydrate quality and quantity), has been associated with an elevated risk of insulin resistance, coronary heart disease (CHD), stroke, and type 2 diabetes mellitus (T2DM) in large-scale epidemiological investigations worldwide.³⁻⁵ However, although refined starches have become staple foods in the average Chinese diet, epidemiological data concerning the correlation between dietary GL and diabetes are limited.

Therefore, the purpose of this study was to investigate the relationship between dietary GL and metabolic indicators, such as blood glucose and lipid profiles, in patients with newly diagnosed T2DM.

METHODS

Participants

The sample included patients with newly diagnosed T2DM and participants with normal glucose tolerance (control group). Participants were all >40 years old and recruited between June 2011 and March 2012 for the REACTION Study, a prospective cohort study using a random sample of community residents across mainland China.⁶ Participants were enrolled at the Sun Yat-sen University's Sun Yat-sen Memorial Hospital in Guang-dong Province, one of the four major hospitals in south-

Corresponding Author: Dr Chaogang Chen, 107 Yanjiang West Road in Guangzhou, China. Tel: +86 13711789673; Fax: 020-81332199 Email: ccg2002@163.com; 99350094@qq.com Manuscript received 13 May 2016. Initial review completed 06 June 2016. Revision accepted 12 October 2016. doi: 10.6133/apjcn.052017.03 eastern China to participate in the REACTION Study. Diabetes was diagnosed according to the 2010 American Diabetes Association diagnostic criteria: fasting plasma glucose (FPG) ≥7.0 mmol/L, 2-h postprandial blood glucose (2hPG) ≥11.1 mmol/L, or glycated hemoglobin (HbA1c) ≥6.5%. Normal blood glucose was defined as FPG <5.6 mmol/L, 2hPG <7.8 mmol/L, and HbA1c <5.7%. The control and diabetes groups were matched by age and sex. Subjects with acute or severe vital organ dysfunction (liver, kidney, heart, or lung), severe infection or ketosis, pregnancy, cancer, or positive results for diabetes autoantibodies were excluded. At the time of enrollment, none of the patients with diabetes had received hypoglycemic agents. The study protocol was approved by the Sun Yat-sen Memorial Hospital Ethics Committee (No. [2013] 伦审研第(12)号), and the study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from each participant before data collection.

Study design

A uniform questionnaire was used to collect continuous 3-day dietary records. The forms were to be completed over 2 workdays and 1 rest day, and everything each patient ate and drank was recorded. The questionnaire also collected data regarding the type of food, eating time, eating location, and amount of food. Trained staff guided the subjects in documenting this information and showed the participants examples of food and containers. Subjects were encouraged to buy electronic scales to measure their food at home. After forms were returned, incomplete forms were discarded, and participants with completed forms were enrolled. Our department designed a software program to calculate GL according to dietary GI values and carbohydrate intake using the following formula: GL = \sum (GI of food item \times carbohydrate content of food item/100). The total GL was calculated for each of the 3 days, and the 3 values were averaged. Food sources of dietary GL were calculated as \sum (GL of food item)/total daily GL \times 100%. GI was divided into three levels: high-GI (>70), medium-GI (55–70), and low-GI (<55).⁷ Height, weight, waist circumference (Wc), blood pressure, and body fat percentage after an overnight fast were measured by the same researcher using consolidated tools. Seated blood pressure was measured in triplicate with an automatic sphygmomanometer (HEM-7112, Omron, Dalian, China). Hypertension was defined as systolic blood pressure \geq 140 mmHg or diastolic blood pressure \geq 90 mmHg. Body fat percentage was estimated using bioelectrical impedance analysis (UM-41, TANITA, Dongguan, China), and $\geq 25\%$ in men or $\geq 30\%$ in women was considered outside the limit. Body mass index (BMI) was calculated as weight divided by height squared (kg/m²); overweight was defined as BMI ≥ 24 kg/m². The Charlson weighted index of comorbidities (WIC) was used to assess health status.8 After a 12-h overnight fast, venous blood samples were drawn to measure FPG, HbA1c, fasting insulin (Fins), total cholesterol (TC), triglyceride (TG), highdensity lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) concentrations. All subjects then underwent a 75-g oral glucose tolerance test, and 2hPG was determined using the glucose oxidase

method (GF-D200 semi-automatic biochemistry analyzer, China). Insulin was measured using the direct chemiluminescent method (Immulite 2000 automatic analyzer, USA). HbA1c was assayed using high-pressure liquid chromatography (BIO-RAD, USA). Lipid concentrations (TC, TG, HDL-C, and LDL-C) were measured using enzymatic colorimetry (GF-D200 semi-automatic biochemistry analyzer, China). Dyslipidemia was defined as TC \geq 6.22 mmol/L, TG \geq 2.26 mmol/L, HDL-C <1.04 mmol/L, or LDL-C \geq 4.14 mmol/L. Insulin sensitivity was evaluated using the homeostasis model assessment for insulin resistance (HOMA-IR): HOMA-IR = FPG \times Fins/22.5. The basic secretory function of β -cells was measured using the homeostasis model assessment for β -cell function (HOMA-B): HOMA-B = $20 \times \text{Fins}/(\text{FPG} - 3.5)$. A family history of diabetes was defined as positive if any firstor second-degree relative had T2DM. A current smoker was defined as someone who smoked at least 1 cigarette per day for at least 6 months, and a current drinker was defined as someone who drank alcohol at least once a week for more than 6 months. The short form of the International Physical Activity Questionnaire (IPAQ) was used to determine the physical activity level during leisure time.9

Statistical analysis

Statistical analysis was performed using SPSS 13.0 software for Windows (SPSS Inc., Chicago, IL). Normally distributed variables are expressed as mean \pm standard deviation, while variables that were not normally distributed are expressed as median (interquartile range). Qualitative data were compared using chi-square tests. Normally distributed quantitative data were compared using ttests; data without a normal distribution were compared using rank sum tests. HOMA-IR and HOMA-B were logarithmically transformed (lg₁₀). Pearson correlation coefficients were computed to assess the associations between variables. Dietary GL was included as the dependent variable in the multivariable regression analysis to explore the relative risk of metabolic status. Intakes of legumes, milk, and animal foods were all divided into two levels according to the recommendations of the Balanced Diet Pagoda of Chinese Residents (i.e., 30 g/d, 300 g/d, and 150 g/d, respectively). A two-sided p value <0.05 was considered statistically significant.

RESULTS

General characteristics

A total of 220 patients with newly diagnosed T2DM and 150 subjects with normal plasma glucose concentrations were recruited; however, 19 patients and 24 control subjects were excluded because their forms were not fully completed. Finally, 201 patients with newly diagnosed T2DM and 126 subjects with normal plasma glucose concentrations were enrolled. Only 12.6% of the patients with diabetes and 20.8% of the control subjects were currently employed; the remaining subjects were retired or homemakers. There were no significant differences between these two groups regarding age, sex, education (percentage who completed high school), occupation (percentage employed), proportion of current smokers, proportion of current drinkers, physical activity level,

body fat percentage, TC concentration, or LDL-C concentration (p>0.05). However, the proportion with a family history of diabetes, BMI, Wc, blood pressure, WIC, HOMA-IR, and concentrations of FPG, 2hPG, HbA1c, Fins, and TG were significantly higher, while HOMA-B and HDL-C concentration was significantly lower in patients with diabetes (p<0.05) (Table 1). Energy, carbohydrate, and fat intakes as well as dietary GL were also significantly higher in patients with diabetes (p<0.05) (Table 2). White rice was the major contributor to dietary GL, followed by wheat flour, tubers, fruits, coarse grains, milk, legumes, and vegetables in all subjects (Table 3).

Correlations between dietary glycemic load/carbohydrate intake and clinical features In patients with newly diagnosed T2DM, dietary GL and carbohydrates were both positively correlated with BMI, but negatively correlated with HDL-C concentration (p < 0.05). In addition, dietary GL was positively correlated with HbA1c (p < 0.05), while carbohydrate intake was not. Neither dietary GL nor carbohydrate intake was significantly correlated with HOMA-IR; HOMA-B; or concentrations of FPG, PPG, Fins, LDL-C, TC, and TG in patients with diabetes (p>0.05). On the other hand, in the control group, dietary GL and carbohydrate intake were significantly and inversely

Table 1. General characteristics of participants with newly diagnosed type 2 diabetes mellitus and those with normal blood glucose (mean±standard deviation)

	DM	CON	Statistical	
	(n=201)	(n=126)	value	р
Sociodemographic factors				
Sex (M/F)	80/121	52/74		
Age (y)	57.9±6.48	55.7±7.59	0.790	0.06
Education (% completed high school)	63.8	61.8	0.128	0.721
Occupation (% in work)	12.6	20.8	3.63	0.057
Family history of diabetes (%)	24.4	15.1	4.07	0.044
Behavioral factors				
Current smoker (%)	16.4	10.8	1.74	0.188
Current drinker (%)	22.8	17.5	1.16	0.281
Physical activity				
Low	64.8	66.7	0.120	0.729
Moderate to high	35.2	33.3	0.120	0.729
Anthropometric assessment				
$BMI(kg/m^2)$	24.4±3.50	22.4±4.04	4.70	< 0.01
Wc (cm)	92.8±6.84	84.7±9.60	8.84	< 0.01
Systolic pressure (mmHg)	134.0±16.7	125±15.9	5.11	< 0.01
Diastolic pressure (mmHg)	77.7±10.2	74.0±8.9	3.38	< 0.01
Body fat (%)	29.9±5.77	28.8±6.27	1.66	0.098
Biochemical variables				
FPG (mmol/L)	7.32±1.96	5.13±0.44	15.3	< 0.01
PPG (mmol/L)	12.6±2.77	6.11±0.98	30.1	< 0.01
Fins (umol/L)	9.57±5.12	7.08±3.23	4.89	< 0.01
HbA1c (%)	7.1±1.5	5.1±0.4	13.5	< 0.01
TC (mmol/L)	5.55±1.53	5.45±1.04	0.728	0.5
TG (mmol/L)	1.7 (1.15, 2.41)	1.22 (1.28, 1.57)	5.11	< 0.01
HDL-C (mmol/L)	1.27±0.36	1.46 ± 0.35	-4.68	< 0.01
LDL-C (mmol/L)	3.32±1.30	3.29±0.79	0.002	0.99
WIC	1.11±0.32	0.15±0.36	25.3	< 0.01
lgHOMA-IR	0.42 ± 0.27	0.16±0.20	9.11	< 0.01
lgHOMA-B	1.70±0.32	1.91±0.22	-6.45	< 0.01

DM: diabetes mellitus group; CON: control group; N: number; M: male; F: female; BMI: body mass index; Wc: waist circumference; FPG: fasting plasma glucose; PPG: postprandial plasma glucose; Fins: fasting insulin; HbA1c: glycated hemoglobin; TC: total cholesterol; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; WIC: Charlson weighted index of comorbidities; HOMA-IR (= FPG × Fins/22.5): homeostasis model assessment for insulin resistance; HOMA-B (= $20 \times$ Fins/(FPG - 3.5): homeostasis model assessment for β -cell function.

Table 2. Dietary characteristics of participants with newly diagnosed type 2 diabetes mellitus and those with normal blood glucose (mean±standard deviation)

Group	N (M/F)	EN (kcal/d) [‡]	CHO (g/d)	FAT (g/d)	PRO (g/d)	DF (g/d)	GL
DM	201 (80/121)	1933±324	241±57.9	72.3±18.5	80.6±21.3	11.5±3.9	170±44.8
CON	126 (52/74)	1816±309	219±46.3	64.8±16.3	86.4±25.2	11.8±4.3	158±38.5
Z/t	-0.405	2.36	3.68	1.89	-1.07	-0.209	2.51
р	0.686	0.038^{*}	0. 001**	0.046^{*}	0.053	0.783	0.013^{*}

[†]EN: energy; CHO: carbohydrate; PRO: protein; DF: dietary fiber; GL: glycemic load; the other abbreviations are the same as table 1. [‡]1 kcal=4.184 kJ.

p < 0.05, p < 0.01.

Food types	DM	CON	GI degree
White rice	83.5±10.6	81.3±10.6	High
Wheat flour	11.8±6.89	12.6±6.33	High
Coarse grain	0.75±0.57	1.01 ± 0.62	Medium
Tubers	1.38±0.63	2.26±2.33	Medium
Legumes	0.32±0.17	0.37±0.28	Low
Milk	0.82±0.67	0.87±0.71	Low
Vegetables	0.28±0.36	0.33±0.14	Low to high
Fruits	1.15±1.89	1.35 ± 0.58	Low to high

Table 3. Food sources of dietary glycemic load in all subjects (%, mean±standard deviation)

[†]GI: glycemic index; the other abbreviations are the same as table 1

[‡]GI degree: GI >70, high; GI 55–70, medium; GI <55, low.

Table 4. Pearson's correlations between dietary glycemic load/carbohydrate intake and clinical features among the total sample (r [*p* value])

Group	n	BMI	FPG (mmol/L)	PPG (mmol/L)	HbA1c (%)	Fins
CON						
GL	126	0.087 (0.334)	0.005 (0.957)	-0.025 (0.783)	-0.109 (0.222)	0.044 (0.624)
CHO	126	0.091 (0.310)	0.009 (0.923)	0.011 (0.905)	0.127 (0.155)	0.048 (0.596)
DM						
GL	201	$0.187 (0.009)^{**}$	0.077 (0.275)	0.064 (0.366)	$0.154 (0.029)^{*}$	0.001 (0.987)
CHO	201	$0.171 \left(0.017 ight)^{*}$	0.049 (0.491)	0.062 (0.38)	0.117 (0.099)	-0.017 (0.815)
Crown	HDL-C	LDL-C	TC	TG		1-IIOMA D
Group	(mmol/L)	(mmol/L)	(mmol/L)	(mmol/L)	IgnOMA-IK	Ідпома-в
CON						
GL	-0.197 (0.027)*	-0.115 (0.198)	-0.213 (0.057)	-0.034 (0.704)	0.019 (0.830)	0.002 (0.981)
CHO	-0.191 (0.032)*	-0.125 (0.162)	-0.188 (0.065)	0.156 (0.080)	0.025 (0.784)	0.002 (0.983)
DM						
GL	-0.207 (0.003)**	-0.149 (0.135)	-0.150 (0.134)	0.048 (0.494)	0.033 (0.639)	0.021 (0.769)
CHO	-0.183 (0.009)**	0.118 (0.097)	0.112 (0.112)	0.042 (0.554)	0.008 (0.908)	0.012 (0.870)

[†]Abbreviations are the same as in Table 1.

p*<0.05, *p*<0.01.

correlated with HDL-C concentrations (p<0.05) but were not significantly correlated with BMI; HOMA-IR; HOMA-B; or concentrations of FPG, PPG, HbA1c, Fins, LDL-C, TC, or TG (p>0.05) (Table 4).

Multivariable analysis examining the relative risks of diabetes and dyslipidemia

To explore the relative risk of metabolic status in multivariable regression analysis, all subjects were classified as having a high GL or normal GL based on the average GL of the control group (i.e., 158). A high-GL diet increased the odds of having diabetes, a low HDL-C concentration, and higher WIC score as well as being overweight (odds ratios [ORs]=1.83, 1.78, 1.96. and 2.00, respectively; 95% confidence intervals [CIs]=1.16–2.87, 1.15–2.64, 1.23–3.14, and 1.28–3.11, respectively; p<0.05) after adjusting for age, sex, education, occupation, family history of diabetes, and behavioral habits (smoking, drinking, and physical activity) (Table 5).

DISCUSSION

In the current study, dietary GL and carbohydrate intake were both significantly higher in patients with newly diagnosed T2DM than in participants with normal blood glucose concentrations. Dietary GL was also positively correlated with HbA1c in patients with newly diagnosed T2DM, while carbohydrate intake was not. In addition, the multivariable regression analysis showed that a higher GL (>158) increased the odds of having diabetes (OR=1.83, 95% CI=1.16-2.87), suggesting that a high-GL diet may play an important role in the occurrence of T2DM. These results are similar to those reported in large-scale epidemiological investigations in America that tracked 65,173 women aged 40-65 years and 42,759 men aged 40-75 years for 6 years.10,11 Furthermore, Bhupathiraju et al confirmed that consumption of a high-GL diet is associated with a higher risk of T2DM among 74,248 women from the Nurses' Health Study (1984-2008), 90,411 women from the Nurses' Health Study II (1991-2009), and 40,498 men from the Health Professionals Follow-Up Study (1986-2008) who were free of diabetes, cardiovascular disease, and cancer at baseline.3 However, a 12-year follow-up study among 16,835 participants in eight European cities (during which 11,559 participants developed incident diabetes) found that dietary GI, GL, and carbohydrate intake were not associated with diabetes risk after adjusting for confounding factors.¹² Despite the small sample size in the present study, a GL >158 increased the risk of diabetes by approximately 1.8 times among participants in Guangdong Province in southeastern China. However, a large epidemiological investigation is needed to explore the classification of a high-GL diet in China as well as its association with the risk of diabetes. Furthermore, the mechanism underlying the role of a high-GL diet in the occurrence of T2DM remains unclear. There was no relationship between dietary GL and HOMA-IR or HOMA-B in our study, which was similar to former observations.¹³

	GL >158 cases (n)	$GL \leq 158$ cases (n)	OR (95% CI)	р
Diabetes	113	88	1.83 (1.16-2.87)	0.009^{**}
$TC \ge 6.22 \text{ mmol/L}$	38	25	1.64 (0.937-2.87)	0.093
$TG \ge 2.26 \text{ mmol/L}$	39	30	1.36 (0.798-2.33)	0.280
HDL-C <1.04 mmol/L	54	37	1.78 (1.15-2.64)	0.036^{*}
LDL-C \geq 4.14 mmol/L	26	34	0.704 (0.401-1.24)	0.254
BMI $\geq 24 \text{ kg/m}^2$	88	59	2.00 (1.28-3.11)	0.003^{**}
$Wc \ge 85 \text{ cm} (M)/80 \text{ cm} (F)$	129	120	1.14 (0.672-1.92)	0.689
SBP ≥140 mmHg	47	40	1.23 (0.749-2.01)	0.453
DBP ≥90 mmHg	14	8	1.80 (0.732-4.41)	0.269
Body fat $\geq 25\%$ (M)/30% (F)	101	99	1.08 (0.673-1.73)	0.810
WIC ≥1	123	97	1.96 (1.23-3.14)	0.007^{**}
Legumes <30 g/d	145	144	0.906 (0.460-1.78)	0.863
Milk <300 g/d	158	160	0.282 (0.058-1.38)	0.174
Animal foods <150 g/d	55	67	0.726 (0.457-1.15)	0.197

Table 5. Relative risk of metabolic status based on the average glycemic load of the control group

[†]Abbreviations are the same as in Table 1.

^{*}Adjusted by age, sex, education, occupation, family history of diabetes, and behavioral habits (smoking, drinking, and physical activity). *p<0.05, **p<0.01.

The positive correlation between dietary GL and HbA1c also illustrates that dietary GL is closely related to blood glucose control in patients with diabetes, results that are similar to those in a cross-sectional study of 640 patients with T2DM aged 28-75 years.¹⁴ This suggests that it is necessary to reduce dietary GL (i.e., restrict dietary carbohydrate intake and modify the dietary structure) in patients with poorly controlled diabetes. This hypothesis is supported by the results of a study by Ziaee et al,¹⁵ in which a low-GL, high-fat diet (GL=67; energy=1800 kcal; total fat=36%; fat derived from olive oil and nuts=15%; carbohydrates=42%; protein=22%) was administered to 100 patients with poorly controlled diabetes (HbA1c >8.0%) for 10 weeks. The results showed that a low-GL diet had a significant effect on FPG and HbA1c. This study also showed that the effect of a low-GL diet on glycemic control was greater among those with more severe dysglycemia. Another study showed that a canola oil-enriched low-GL diet provided for 3 months to 55 patients with different T2DM courses enrolled in a Canadian academic center resulted in a 0.47% decrease in HbA1c.¹⁶ However, a low-GL, high-fat dietary pattern may not be suitable for Chinese patients because of differences in eating habits between China and Canada. For example, white rice was the staple food and contributed more than 80% of the food sources of dietary GL in all subjects in our study, which is unlikely to happen in Canada. Moreover, the glycemic impact of foods will depend not only on the level of refinement, type, cultivar, and cooking method but also on accompanying foods during the same eating occasion. Therefore, identification of an appropriate low-GL diet for the Chinese population is still needed.

However, our results showed that dietary carbohydrate intake was not significantly correlated with HbA1c in patients with newly diagnosed T2DM, indicating that a higher dietary carbohydrate intake is unrelated to the occurrence of diabetes and glycemic control. This is consistent with the results reported by Greenwood et al,¹⁷ who conducted a meta-analysis of data extracted from 24 publications of 21 cohort studies and found that doseresponse trends were linear for GI and GL but more complex for total carbohydrate intake in relation to the risk of T2DM. Therefore, the amount and type of carbohydrates consumed should be considered. However, the findings of a large-scale population-based cohort study in Japan suggested that a low-carbohydrate diet was associated with a decreased risk of T2DM in Japanese women but not in Japanese men.¹⁸ Furthermore, as in Japan, white rice is the principal contributor to the total carbohydrate and energy intake in China, a trend that is seldom observed in Western populations. Therefore, a larger sample size is needed to determine whether controlling the dietary GL or restricting the dietary carbohydrate intake would be more effective in reducing the risk of T2DM in China.

Our study also showed that dyslipidemia in patients with T2DM is mainly characterized by an increased TG concentration and a decreased HDL-C concentration. The correlation analysis showed that dietary GL was negatively related with plasma HDL-C concentrations but unrelated with TG concentrations of both patients with newly diagnosed T2DM and participants with a normal blood glucose concentration. The multivariable regression analysis showed that a higher GL increased the odds of having low HDL-C concentrations (OR=1.778, 95% CI=1.154-2.644) in southeastern China. This is similar to findings reported in large-scale studies among the general population in America¹⁹ and Japan.²⁰ For example, Ford et al. found that dietary GL and HDL-C concentrations were inversely related in a cross-sectional study of 13,907 American participants aged ≥ 20 years. Furthermore, Kentaro et al investigated the association between dietary GL and metabolic risk factors in 1,354 healthy Japanese female farmers aged 20-78 years with traditional dietary habits. The authors reported that dietary GL was independently and negatively associated with serum HDL-C concentrations but positively associated with serum TG concentrations. However, studies examining the association between dietary GL and blood lipids in large-scale Chinese populations are lacking. Unlike the study by Kentaro et al, our results did not show a correlation between dietary GL and TG concentration, which may due to the smaller sample size, sex-based differences, professional variations, or other factors. In addition, a study using the same target population and intervention strategy as Ziaee et al¹⁵ found that a low-GL diet had a significant effect on the lipid profile of participants, including TC, TG, LDL-C, and HDL-C concentrations.²¹

Furthermore, the findings in this study showed that dietary GL was positively correlated with BMI in patients with newly diagnosed type 2 diabetes and that the risk of being overweight (BMI ≥ 24 kg/m²) was twice as high (OR=2.00, 95% CI=1.28-3.11) among participants with a GL >158 in southeastern China. These results are similar to those of previous studies in obese patients (without diabetes),²² and this may be related to the postprandial insulin response, which is captured by GL. For example, foods and/or diets producing higher postprandial insulin responses—such as high-GL diets—induce lower satiety and increase voluntary food intake during the subsequent meal compared to foods inducing a low insulin demand.²³ Thus, people who consume a higher GL diet may have decreased satiety after eating, which in turn facilitates an increase in energy intake and thus BMI.

Given the correlations between dietary GL and blood glucose concentration, lipid concentrations, and BMI, the influence of dietary structure on cardiovascular or cerebrovascular diseases cannot be ignored. Indeed, several large-scale epidemiological surveys confirmed that a high-GL diet composed of refined carbohydrates could increase the risk of CHD^{24,25} and ischemic, but not hemorrhagic, stroke.²⁶ For patients with diabetes, the impact of dietary GL on the vascular complications of diabetes deserves further research.

Finally, our study found that WIC was higher with dietary GL >158 (OR=1.96, 95% CI=1.23–3.14) among participants in southeastern China, which had not previously been reported. WIC was developed 29 years ago as a prognostic index of comorbid conditions for patients admitted to a general medical service and based on the presence of medical conditions weighted according to their effect on mortality for patients enrolled in longitudinal studies.8 Former studies have affirmed the appraisal value of WIC in the prognosis of various diseases, such as sepsis, pneumonia, cardiovascular disease, renal disease, tumor, fracture, and trauma.²⁷ Therefore, the control of dietary GL might have broad and long-term benefits for people beyond our current understanding.

Although our study has several strengths, there are several limitations. First, the sample size was relatively small. Second, the dietary data comprised continuous 3-day dietary records that may or may not represent a subject's general dietary GL. Third, measurement errors may exist because dietary GI and GL data derived from questionnaires are likely to have substantial errors arising both from the 3-day dietary records and the GI values used for the food items. However, because of the scarcity of data, it was necessary to use GI values measured in other countries for some food items even though the properties of food items may vary between countries. Nonetheless, our results provide valuable insights for prevention efforts.

In conclusion, our results showed that the mean dietary GL in patients with newly diagnosed T2DM was significantly higher than that in participants with normal glucose concentrations, and white rice was the major contributor to dietary GL. In addition, mean dietary GL was

positively related with HbA1c and BMI, and a higher GL increased the odds of having diabetes and higher WIC score as well as being overweight among participants in southeastern China. These findings indicate that a high-GL diet may be relevant to diabetes risk, glycemic control, prognosis of diseases, and body composition. Furthermore, dietary GL was inversely related with plasma HDL-C concentrations and increased the odds of having a low HDL-C concentration in both the diabetes and control groups, suggesting that dietary GL is closely related with lipid metabolism. Consequently, it is necessary to control dietary GL among patients with diabetes as well as individuals with normoglycemia in southeastern China.

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AUTHOR DISCLOSURES

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Breakfast	
Staple food	Porridge, noodles, dumplings, Various kinds of desserts (crispy durian cake, egg tart, steamed bun
	stuffed with barbecued roast pork, Sumai, sticky rice with chicken, et al.)
Proteins	Few milk, soybean milk or yoghourt
Vegetables / Fruits	Rarely
Oil	Rarely
Beverage	Oolong tea (Tie Guanyin), Pu'er tea, scented tea (chrysanthemum tea, jasmine tea)
Lunch and dinner	
Staple food	Rice, noodles, oatmeal, millet, sweet potato, corn, potato, lotus root, kidney bean, black soya bean
Proteins	Fish, pork, chicken, duck, beef, seafood, soybean, tofu, eggs
Vegetables / Fruits	Green vegetables, cucumber, pumpkin (high GI), carrot (high GI), pimento, eggplant, tomato, fungus,
	kelp, bitter gourd, ternip
	Apple, pear, peach, banana, orange, kiwi, cherry, strawberry, watermelon (high GI), pitaya, mango
	(medium GI), pineapple (medium GI), plum
Oil	Colza oil, peanut oil, olive oil, sesame oil, sunflower seed oil
Salt	Iodized salt

Supplementary table 1. Daily food intake for residents in Guangdong Province, China

Supplementary table 2. The values for diseases in Charlson's weighted index of comorbidities

Assigned weights for diseases	Conditions
1	Myocardial infarct
	Congestive heart failure
	Peripheral vascular disease
	Cerebrovascular disease
	Dementia
	Chronic pulmonary disease
	Connective tissue disease
	Ulcer disease
	Mild liver disease
	Diabetes
2	Hemiplegia
	Moderate or severe renal disease
	Diabetes with end organ damage
	Any tumor
	Leukemia
	Lymphoma
3	Moderate or severe liver disease
6	Metastatic solid tumor
	AIDS

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