Original Article

Dietary Glycemic Index, Glycemic Load, and Cardiovascular Disease Risk Factors: Tehran Lipid and Glucose Study

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Abstract

Background: Data available on the effect of quality (glycemic index [GI]) and quantity (glycemic load [GL]) of carbohydrates on the risk factors of cardiovascular disease (CVD) are inconsistent. The objective of this study was to examine the association between dietary GI, GL, and CVD risk factors among Tehranian adults, the participants of the Tehran Lipid and Glucose Study.

Methods: This population- based cross-sectional study was conducted on 2457 subjects (46% men and 54% women), aged 19 to 84 years. Dietary GI and GL were measured using a validated 168- item semiquantitative food frequency questionnaire. Anthropometrics, blood pressure, fasting blood glucose, and lipid profiles were measured.

Results: The mean intakes of GI and GL were 68.3 and 244.8, respectively. Rice (26.6%) and bread (19.0%) were the major contributors to dietary GI and GL, respectively. Higher dietary GI and GL were associated with high intakes of carbohydrate, fiber, refined grain, fruits, simple sugar, snack, and desserts. After adjustment for lifestyle and dietary variables, a higher dietary GI was positively associated with triglycerides and high-density lipoprotein (HDL) cholesterol concentrations among obese subjects. Dietary GL was positively associated with fasting and 2-h blood glucose among nonobese subjects, after adjustment for confounders.

Conclusion: Dietary GI and GL were associated with a few CVD risk factors, and body mass index levels may modulate these associations.

Keywords: Cardiovascular disease risk factors, glycemic index, glycemic load

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Introduction

The prevalence of cardiovascular disease (CVD), one of the main causes of mortality in Iran,[1] continues to rise.[2] Diet is a first- line intervention in the prevention and treatment of CVD risk factors; therefore, many studies have been designed to assess the effects of dietary determinants on the metabolic risk factors.[3,4] Carbohydrate is a macronutrient, the role of quality and quantity of which in CVD risk factors has not been studied extensively. The quality of carbohydrate as determined by glycemic index (GI), has been gaining attention because it can influence the digestion rate and hence affect blood glucose and lipid profiles.[3,5] GI is a measure of how much each carbohydrate-containing food raises blood glucose compared with a standard food of either glucose or white bread.[6] Glycemic load (GL) is a measure of quantity of carbohydrate that reflects both the GI of dietary carbohydrate as well as the amount of carbohydrate ingested.[7] Little is known about the effect of GI and GL on the risk of the CVD, especially in populations with bread and white rice as their staple food.[4,5] Among Iranian population, the average percentage of total energy intake from carbohydrate is 65% and those of total carbohydrate consumption from bread and white rice are 34.2% and 14.8%, respectively.[8] Also, many foods rich in carbohydrates readily available to Iranian population such as bread, rice, potato, and snack foods have a high GI.[9] Thus, increase intakes of carbohydrates with high GI and GL, consumed by Iranians, may lead to a higher prevalence of CVD risk factors; effects documented in some of the previous studies,[4,5,10] but not in others.[3,11] Current studies suggest that the dietary carbohydrate, GI, and GL might affect CVD through an effect on body mass index (BMI).[4,12,13] Considering the limited data on dietary GI and GL in Iranian adults, the aim of this study was to assess the cross-sectional relationship between GI, GL, and CVD risk factors, based on stratification of BMI into obese and nonobese subjects.

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Materials and Methods

Study participants

This population-based cross-sectional study was conducted within the framework of the Tehran Lipid and Glucose Study (TLGS), which is a prospective study performed on residents of district 13 of Tehran, with the aim of determining the prevalence of noncommunicable disease risk factors and developing a healthy lifestyle to improve those risk factors. The design of the study has been described before.[14,15] Briefly, a total of 15005 subjects, aged ≥ 3 y, were selected randomly by a multistage cluster sampling method and followed up, every three years. During the

third examination survey of the TLGS (2006 – 2008), a total of 12523 subjects completed the examinations, of whom 4920 were randomly selected for completing the dietary assessment. Finally, the dietary data for 2979 subjects who agreed to participate and completed the food frequency questionnaire (FFQ) were available (response rate: 70%). Participants were excluded if they had prior medical history of myocardial infarction (n = 22), stroke (n = 19), and cancer (n = 7) because of possible changes in diet associated with these conditions, or those left more than 70 items blank on the FFQ, and reported daily energy intake outside the range of 800 – 4200 kcal/d (n = 167); also excluded were those for whom physical activity, anthropometric, or biochemical data were missing (n = 103), and those with data on hyperlipidemia, hyperglycemia, and hypertension that had changed their dietary intakes (n = 204). The final study sample included 2457 participants (1327 males and 1130 females). Informed written consents were obtained from all participants and the study protocol was approved by the Research Council of the Research for Endocrine Sciences, Shahid Beheshti University of Medical Sciences.

Dietary assessment and calculation of GI and GL

Dietary data were collected by well-trained interviewers with a validated 168-item semiquantitative FFQ,[16] which asked the participants to provide their usual intake over a period of 12 months. The participants were asked to designate their consumption frequency for each food item consumed during the previous year on a daily, weekly, or monthly basis and this was converted to daily intakes. The portion sizes of these were then converted to grams using household measures.[17] Each food and beverage was analyzed for energy and nutrient intake using the United State Department of Agriculture's (USDA) food composition table (FCT), because the Iranian FCT is incomplete. Dietary GI and GL were derived from the FFQ as follows:[7]

Dietary GI = [(carbohydrate content of each food item) × (number of servings/d) × (GI)]/total daily carbohydrate intake

Dietary GL= (carbohydrate content of each food item) × (number of servings/d) × (GI)

Dietary GL thus represents the quality and quantity of the total intake of carbohydrates. Each unit of dietary GL represents the equivalent of 1 g carbohydrate from glucose. The average GI represents the GL per unit of carbohydrate and reflects the average quality of carbohydrate intake. The GI value of each food item was obtained from the international table of GI,[7] the GI online database maintained by the University of Sydney, [18] and from the publication that lists the GI of Iranian foods.[9] The GI for whole and refined grain, potatoes, starchy vegetables, legumes, and some of fruits was obtained from the publication that lists the GI of Iranian foods[9] and the GI for fruits, dairy products, and nuts was obtained from the international table of GI.[7] The reference of GI values was white bread (GI for white bread =100). When several GI values were available for a food item, the mean GI value was used for analysis. For foods for which a GI value had not been determined, a value was assigned based on the most similar food item. In addition, food items with very low carbohydrate content were ignored because their GI values cannot be accurately measured.

Clinical and biologic measurements

Weight was measured to the nearest 100 g with digital scales, while the subjects were minimally clothed without shoes. Height was measured to the nearest 0.5 cm, in a standing position without shoes, using a tape meter. Waist circumference was measured to the nearest 1 cm, at the umbilical level and that of the hip, at the maximum level, over light clothing, using an outstretched tape meter, without any pressure to the body. BMI was calculated as weight (kg) divided by square of the height (m^2). For blood pressure measurements, two measurements of blood pressure were taken on the right arm, after a 15-minute rest in the sitting position, using a standardized mercury sphygmomanometer; the mean of the two measurements was considered as the participant's blood pressure. Physical activity was assessed using an oral questionnaire, including a list of common activities of daily life; the frequency and amount of time spent on activities per week over the past 12 months were documented.[19] Levels of physical activity were expressed as metabolic equivalent hours per week (METs h/wk)[20] and categorized as light (> 3 METs h/wk), moderate (3 – 6 METs h/wk), and heavy (≥ 6 METs h/wk).[21] Cigarette smoking status was categorized as current smoker, nonsmoker, and ex-smoker. Additional covariate information about age, medical history, and current use of medications was obtained using an oral questionnaire.

Biochemical assessment

Fasting blood samples were taken after 12 –14 h, from all study participants. Serum fasting and 2-h glucose (75 g oral glucose tolerance test) was measured by the enzymatic colorimetric method using the glucose oxidase technique. Total cholesterol was assayed using the enzymatic colorimetric method with cholesterol esterase and cholesterol oxidase. Triglyceride levels were measured by enzymatic colorimetric analysis with glycerol phosphate oxidase. High-density lipoprotein (HDL) cholesterol was measured after precipitation of the apolipoprotein β-containing lipoproteins with phosphotungistic acid. Analyses were performed using Pars Azmun kits (Pars Azmun Inc., Tehran, Iran) and a Selectra 2 autoanalyzer (Vital Scientific, Spankeren, Netherlands). Low-density lipoprotein (LDL) cholesterol was calculated according to the Friedewald method.[22] It was not calculated when the serum concentration of triacylglycerol was > 400 mg/dL. Both inter- and intra-assay coefficients of variations were 2.2% for serum glucose, 2% and 0.5% for HDL cholesterol, and 1.6% and 0.6% for triglycerides, respectively.

Statistical analysis

All analyses were conducted using the Statistical Package for Social Sciences (version 15.0; SPSS Inc, Chicago IL). CVD risk factors were shown to have a normal distribution except for triglycerides for which log-transform value was used to normalize the data and geometric means for triglyceride concentrations were calculated. Significant differences in characteristics across tertiles of dietary GI and GL were evaluated using the one-way analysis of variance (ANOVA) for quantitative variables and the chi-square test for qualitative variables. To evaluate the relationship between dietary GI, GL, and CVD risk factors, the general linear model was used. There was a significant effect of interactions by BMI on the association of GI and GL and CVD risk factors. Therefore, the analysis was done separately according to BMI (nonobese (< 30 kg/m2) and obese (≥ 30 kg/m2) subjects). The means were adjusted for age (years), gender, physical activity (light, moderate, or heavy), smoking status (current, ex-smoker, nonsmoker),

Table 1. Characteristics of the participants of the Tehran Lipid and Glucose Study by tertiles of dietary GI and GL

	Tertile of GI				Tertile of GL			
	1	2	3	P-value	1	2	3	P-value
Characteristics								
Median intake	56.4	69.2	80.1		155.2	227.7	330.5	
Range of intake	≤ 64	65–74	≥ 75		≤ 193	194–272	≥ 273	
Participants (n)	818	819	820		819	819	819	
Female (%)	55.1	47.3	54.1	0.622	68.1	53.5	40.4	< 0.001
Age (y)	38.4 ± 13.6	38.8 ± 13.1	40.5 ± 14.1	0.008	39.9 ± 13.9	39.2 ± 13.2	38.6 ± 13.8	0.126
BMI (kg/m^2)	26.6 ± 5.1	26.5 ± 4.8	27.2 ± 4.7	0.022	27.1 ± 4.9	26.7 ± 4.9	26.6 ± 4.8	0.194
Physical activity (%)				0.462				.050
Light	67.8	65.0	64.3		66.2	19.0	14.8	
Moderate	16.6	16.7	17.3		65.8	16.4	17.8	
Heavy	15.5	18.3	18.4		65.1	15.3	19.7	
Educational levels (%)				< 0.001				0.113
Primary and secondary	21.6	25.1	31.2		28.0	24.1	25.7	
High school	58.2	45.9	54.3		55.9	55.1	56.5	
University	20.1	20.1	14.5		16.1	20.8	17.8	
Current smokers (%)	5.5	6.5	4.5	0.678	6.6	10.4	11.6	0.008

BMI: Body Mass Index; [a]Mean ± SD for all such values, except for variables was determined; [b]ANOVA for quantitative variables and chi-square test for qualitative variables.

Table 2. Cardiovascular risk factors across tertiles of dietary GI according to BMI categories in the participants of the Tehran Lipid and Glucose Study.

	BMI < 30 kg/m^2				BMI > 30 kg/m^2			
	1	2	3	P-values	1	2	3	P-values
Range of intake	≤ 64	65–74	≥ 75		≤ 64	65–74	≥ 75	
Total cholesterol (mg/dL)								
Model 1[d]	180 ± 1.5 [a]	180 ± 1.5	181 ± 1.5	0.773	195 ± 2.7	196 ± 2.7	197 ± 2.7	0.993
Model 2[e]	181 ± 1.4	180 ± 1.3	179 ± 1.4	0.684	195 ± 2.6	195 ± 2.7	197 ± 2.6	0.875
Model 3[f]	181 ± 1.4	180 ± 1.4	179 ± 1.4	0.680	196 ± 2.7	195 ± 2.7	197 ± 2.7	0.914
Model 4[g]	181 ± 1.4	180 ± 1.3	178 ± 1.4	0.299	196 ± 2.7	195 ± 2.7	197 ± 2.7	0.915
LDL cholesterol (mg/dL)								
Model 1	110 ± 1.3	112 ± 1.3	112 ± 1.3	0.635	122 ± 2.4	124 ± 2.4	123 ± 2.4	0.838
Model 2	111 ± 1.2	112 ± 1.2	111 ± 1.2	0.790	122 ± 2.3	124 ± 2.4	123 ± 2.4	0.847
Model 3	111 ± 1.2	112 ± 1.2	111 ± 1.3	0.748	122 ± 2.4	124 ± 2.4	123 ± 2.5	0.882
Model 4	111 ± 1.2	112 ± 1.2	110 ± 1.3	0.500	122 ± 2.4	124 ± 2.4	123 ± 2.5	0.879
HDL cholesterol (mg/dL)								
Model 1	44.1± 0.4	42.7 ± 0.4	42.5 ± 0.4	0.015	41.6 ± 0.7	41.3 ± 0.6	40.0 ± 0.6	0.183
Model 2	43.9 ± 0.3	42.8 ± 0.4	42.5 ± 0.4	0.029	41.7 ± 0.6	41.4 ± 0.6	39.4 ± 0.6	0.022
Model 3	43.8 ± 0.4	42.8 ± 0.4	42.6 ± 0.4	0.075	41.6 ± 0.6	41.3 ± 0.6	39.5 ± 0.6	0.062
Model 4	43.7 ± 0.4	42.7 ± 0.4	42.9 ± 0.4	0.138	41.6 ± 0.6	41.4 ± 0.6	37.5 ± 0.7	0.043
Triglyceride concentrations (mg/dL)[b]								
Model 1	112 ± 2.8 [b]	113 ± 2.9	114 ± 2.9	0.112	136 ± 2.9	136 ± 2.9	156 ± 3.5	0.024
Model 2	113 ± 2.9	113 ± 2.9	114 ± 2.9	0.660	137 ± 2.9	136 ± 2.9	157 ± 3.5	0.016
Model 3	113 ± 2.9	113 ± 2.9	114 ± 2.9	0.977	137 ± 2.9	136 ± 3.1	155 ± 3.5	0.047
Model 4	113 ± 3.0	114 ± 3.1	114 ± 2.9	0.649	138 ± 2.9	137 ± 3.2	154 ± 3.6	0.049
Diastolic blood pressure (mm Hg)								
Model 1	111 ± 0.7	110 ± 0.7	112 ± 0.7	0.087	117 ± 1.2	118 ± 1.2	117 ± 1.4	0.886
Model 2	111 ± 0.6	110 ± 0.6	111 ± 0.6	0.199	117 ± 1.1	117 ± 1.1	116 ± 1.2	0.867
Model 3	110 ± 0.6	110 ± 0.6	112 ± 0.6	0.119	116 ± 1.1	117 ± 1.1	117 ± 1.3	0.866
Model 4	111 ± 0.6	110 ± 0.6	111 ± 0.6	0.280	116 ± 1.1	117 ± 1.1	117 ± 1.2	0.866
Systolic blood pressure (mm Hg)								
Model 1	74.1 ± 0.5	74.1 ± 0.4	74.5 ± 0.5	0.750	78.3 ± 0.8	79.2 ± 0.8	80.2 ± 0.8	0.282
Model 2	74.1 ± 0.4	73.9 ± 0.4	74.1 ± 0.4	0.932	78.1 ± 0.7	78.8 ± 0.7	79.9 ± 0.8	0.242
Model 3	73.9 ± 0.4	73.9 ± 0.4	74.2 ± 0.5	0.863	78.1 ± 0.7	78.8 ± 0.8	80.0 ± 0.8	0.261
Model 4	74.1 ± 0.4	74.0 ± 0.4	74.1 ± 0.4	0.998	78.1 ± 0.7	78.8 ± 0.8	80.0 ± 0.8	0.254
Fasting blood glucose (mg/dL)								
Model 1	88.1 ± 0.7	88.6 ± 0.8	89.3± 0.7	0.504	96.6 ± 2.1	97.3 ± 2.1	99.4 ± 2.1	0.630
Model 2	88.4 ± 0.7	88.4 ± 0.6	88.4 ± 0.7	0.999	97.5 ± 2.0	97.0 ± 2.0	95.6 ± 2.0	0.750
Model 3	88.5 ± 0.7	88.4 ± 0.7	88.3 ± 0.7	0.949	96.4 ± 2.1	97.6 ± 2.0	96.4 ± 2.1	0.894
Model 4	88.6 ± 0.7	88.5 ± 0.6	88.1 ± 0.7	0.833	96.4 ± 2.0	97.6 ± 2.0	96.4 ± 2.1	0.884
2-h blood glucose (mg/dL)								
Model 1	93.2 ± 1.5	97.7 ± 1.5	97.7 ± 1.5	0.055	108.1 ± 3.4	110.4 ± 3.4	112.7 ± 3.4	0.631
Model 2	93.4 ± 1.5	97.9 ± 1.4	96.5 ± 1.5	0.097	108.2 ± 3.3	111.0 ± 3.3	109.6 ± 3.4	0.838
Model 3	94.3 ± 1.5	97.9 ± 1.5	95.6 ± 1.6	0.201	109.1 ± 3.4	111.0 ± 3.3	108.7 ± 3.4	0.876
Model 4	94.5 ± 1.5	98.1 ± 1.4	95.2 ± 1.5	0.184	109.2 ± 3.4	111.1 ± 3.3	108.6 ± 3.4	0.864
Waist circumference (cm)								
Model 1	83.3 ± 0.4	84.4 ± 0.4	85.6 ± 0.4	0.001	103.5 ± 0.7	102.7 ± 0.7	103.7 ± 0.7	0.558
Model 2	83.7 ± 0.3	84.1 ± 0.3	85.1 ± 0.4	0.021	103.4 ± 0.6	102.9 ± 0.6	103.6 ± 0.6	0.717
Model 3	83.7 ± 0.4	84.2 ± 0.3	85.1 ± 0.4	0.027	103.2 ± 0.6	102.9 ± 0.6	103.7 ± 0.6	0.655
Model 4	84.1 ± 0.2	84.5 ± 0.2	84.3 ± 0.2	0.302	103.2 ± 0.4	103.1 ± 0.4	103.7 ± 0.4	0.549

Model 1 was crude; Model 2 was adjusted for age, gender, physical activity, smoking status, and educational levels; Model 3 was further adjusted for total energy intake, percentage of energy from carbohydrate, percentage of energy from fat, percentage of energy from protein, fiber, and magnesium; Model 4 was further adjusted for BMI; [a] Mean ± SEM; [b] Geometric mean ± SEM.

educational levels (primary and secondary, high school, university), total energy intake (continuous), percentage of energy from carbohydrate (continuous), percentage of energy from fat (continuous), percentage of energy from protein (continuous), fiber (continuous), magnesium (continuous), and BMI (continuous). To determine the *P*-value for trends across tertile categories, we assigned the median intake of each tertiles category to individuals' variables in the general linear model for CVD risk factors. P-values < 0.05 were considered as statistically significant.

Results

Of 2457 study participants, 54.0% were females and 46% males, with the mean ages of 38.0 ± 12.8 and 40.7 ± 14.4 years, respectively. The mean intakes of protein, fat, and carbohydrate were 13.6%, 31.4%, and 57.5% of energy, respectively. The mean intakes of dietary GI and GL were 68.3 and 244.8, respectively. Rice (26.6%) and bread (19.0%) were the major contributor to dietary GI and GL, followed by fruits (10.8%), simple sugar (8.2%), snack and dessert (4.0%), potato and potato chips (2.1%), soft drinks (1.7%), pasta and noodle (1.4%), and honey and jams (1.4%). Among breads, white bread including Lavash (9.8) had a higher GI and GL, compared with dark breads: Sangak (4.8%), Taftoon (2.8%), and Barbari (2.6%). Table 1 shows characteristics of the participants by tertiles of dietary GI and GL. Participants with a high dietary GI tended to be older, had higher educational levels, and higher BMI. No significant differences were found between the smoking status and physical activity levels across tertiles of dietary GI. Participants with a high dietary GL had lower physical activity levels and most of them were smokers. No significant differences were found between the age, BMI, and educational levels across tertiles of dietary GL.

The association between dietary GI and CVD risk factors is shown in Table 2. Among nonobese subjects, a high dietary GI was positively associated with higher waist circumference, lower HDL cholesterol, and 2-h blood glucose, an association which disappeared after adjustment for confounding variables. Among obese subjects, a higher dietary GI intake was significantly associated with higher triglyceride concentrations and lower HDL cholesterol, after adjustment for confounding variables.

The association between dietary GL intake and the CVD risk factors is shown in Table 3. Among nonobese subjects, after adjustment for confounding variables, a higher dietary GL intake was significantly associated with higher fasting blood sugar and 2-h blood glucose. Among obese subjects, no association was found between dietary GL and CVD risk factors after adjustment for confounders.

Discussion

In this population-based cross-sectional study, conducted among a Tehranian population, a positive association between dietary GI and high serum triglycerides and low HDL cholesterol concentrations were found in obese subjects. Among nonobese subjects, a positive association was also seen between dietary GI and enlarged waist circumference, an association which disappeared after adjustment for confounding variables. We also found that dietary GL was positively associated with fasting and 2-h blood glucose after controlling for potentially confounding factors in nonobese subjects.

Epidemiologic studies have shown that high triglyceride and low HDL cholesterol concentrations are independent risk factors for CVD.[23,24] These lipid profiles are typical lipoprotein disturbances associated with the metabolic syndrome [25] which has a high prevalence in Iran.[26,27] In the current study, a positive association between dietary GI and low HDL cholesterol and triglyceride concentrations was found in obese subjects, after controlling for confounders; however, no association was seen between dietary GL and these lipid profile variables. Studies on dietary GI in relation to HDL cholesterol and triglyceride concentrations have documented inconsistent results. Some epidemiologic studies reported an inverse association[28,29] while others reported no association.[5,11,30] Furthermore, although some clinical trials report the beneficial effect of a low GI diet on HDL cholesterol and triglyceride concentrations,[31] others do not.[32,33] In the current study, no association was shown between dietary GL and HDL cholesterol and triglyceride concentrations, findings is consistent with a one year longitudinal study which showed GL was not associated with triglyceride and HDL cholesterol concentrations;[3] in the Whitelall II study as well no association was shown between GL and triglyceride concentrations.[34] However, other clinical and cross-sectional studies have shown an inverse association between dietary GL and HDL cholesterol and triglyceride concentrations.[5,35–37] These varying results may be due to differences in study design, target populations, and food patterns determining the dietary GI and GL in epidemiologic studies. In addition, BMI may modulate this association. Two previous studies suggest that effects of carbohydrates and, GI and GL intakes on HDL cholesterol and triglyceride concentrations were dependent on BMI levels.[4,13] Shikany, et al. also showed that GL was inversely associated with HDL cholesterol concentration in normal- weight subjects, but not in overweight and obese subjects.[11] In addition, Zhang, et al. showed that subjects who had higher HOMA-IR values had smaller reductions in triglycerides and HDL cholesterol after a low GI diet.[31] The present study has also shown the significant interactive effects of dietary GI and BMI levels on high triglyceride and HDL cholesterol concentrations, by additionally adjusting BMI for these lipid profiles to show the relationship between dietary GI and these CVDs are dependent on adiposity.

Despite some international diabetes organizations advocating the use of low dietary GI in prevention and management of diabetes, the American Diabetes Association (ADA) and 2005 USDA Dietary Guidelines suggest further research on dietary GI and GL in relation to risk of type 2 diabetes, because of insufficient information about the relationship between those two.[38–40] In the current study, dietary GL, but not GI, was positively associated with plasma fasting glucose and 2-h fasting glucose concentrations, only among nonobese subjects. In some cross-sectional studies no association was reported between dietary GI and fasting blood glucose[34,41,42] and 2-h blood glucose;[34] however, one study reported that diet high in GL was positively associated with HbA1c.[42] Although the GI relates to a standard amount of carbohydrates in a food, GL is a concept derived from both the GI and the amount of carbohydrate intake. The usefulness of GL is based on the idea that postprandial blood glucose and insulin responses not only depend on the quality (GI) of carbohydrates from a food or diet, but also on the quantity.[43] Also, in the current study, compared with dietary GI, dietary GL had a stronger correlation with intakes of refined grain, simple sugar, soft drinks, honey, and jams and therefore may consequently be associated with high glycemic

Table 3. Cardiovascular risk factors across tertiles of dietary GL, according to BMI, in the participants of the Tehran Lipid and Glucose Study[a]

	BMI < 30 kg/m²				BMI ≥ 30 kg/m²			
	1	2	3	P-values	1	2	3	P-values
Range of intake	**≤ 195**	**196–274**	**≥ 275**		**≤ 186**	**187–269**	**≥ 270**	
Total cholesterol (mg/dL)								
Model 1[d]	179 ± 1.5 [a]	182 ± 1.5	180 ± 1.5	0.407	198 ± 2.6	194 ± 2.8	195 ± 2.7	0.577
Model 2[e]	179 ± 1.4	181 ± 1.3	180 ± 1.4	0.572	195 ± 2.6	195 ± 2.7	198 ± 2.7	0.759
Model 3[f]	180 ± 1.8	181 ± 1.4	179 ± 1.8	0.663	192 ± 3.6	195 ± 2.7	201 ± 4.1	0.393
Model 4[g]	180 ± 1.8	181 ± 1.4	179 ± 1.8	0.629	192 ± 3.6	195 ± 2.7	201 ± 4.1	0.407
LDL cholesterol (mg/dL)								
Model 1	111 ± 1.3	112 ± 1.3	112 ± 1.3	0.787	126 ± 2.3	121 ± 2.5	122 ± 2.4	0.310
Model 2	111 ± 1.3	111 ± 1.2	111 ± 1.3	0.980	123 ± 2.3	122 ± 2.4	123 ± 2.4	0.901
Model 3	112 ± 1.6	111 ± 1.2	111 ± 1.6	0.932	120 ± 3.2	122 ± 2.4	127 ± 3.6	0.402
Model 4	111 ± 1.5	111 ± 1.2	111 ± 1.6	0.971	120 ± 3.2	122 ± 2.4	127 ± 3.6	0.413
HDL cholesterol (mg/dL)								
Model 1	43.8 ± 0.4	43.1 ± 0.4	42.3 ± 0.4	0.036	42.3 ± 0.6	40.1 ± 0.7	40.3 ± 0.7	0.034
Model 2	42.9 ± 0.4	43.1 ± 0.4	43.4 ± 0.4	0.692	41.1 ± 0.6	40.4 ± 0.7	40.9 ± 0.7	0.709
Model 3	42.4 ± 0.5	43.0 ± 0.4	43.9 ± 0.5	0.168	41.3 ± 0.9	40.3 ± 0.7	40.7 ± 0.9	0.604
Model 4	42.4 ± 0.5	42.9 ± 0.3	43.9 ± 0.5	0.228	41.3 ± 0.8	40.4 ± 0.6	40.7 ± 0.9	0.620
Triglyceride concentrations (mg/dL)								
Model 1	112 ± 2.8 [b]	113 ± 2.9	114 ± 2.9	0.001	136 ± 3.0	136 ± 2.9	137 ± 2.9	0.094
Model 2	113 ± 2.8	114 ± 2.9	114 ± 2.9	0.265	136 ± 3.1	136 ± 2.9	138 ± 2.9	0.191
Model 3	113 ± 2.9	114 ± 2.9	114 ± 3.0	0.144	137 ± 3.1	136 ± 2.9	138 ± 2.9	0.838
Model 4	114 ± 2.9	114 ± 2.9	114 ± 3.0	0.151	137 ± 2.9	136 ± 3.0	138 ± 2.9	0.889
Systolic blood pressure (mm Hg)								
Model 1	111 ± 0.6	111 ± 0.6	112 ± 0.7	0.760	118 ± 1.2	118 ± 1.3	116 ± 1.3	0.564
Model 2	111 ± 0.6	111 ± 0.6	111 ± 0.6	0.989	117 ± 1.1	117 ± 1.2	116 ± 1.2	0.752
Model 3	110 ± 0.8	111 ± 0.6	111 ± 0.7	0.912	118 ± 1.4	117 ± 1.2	114 ± 1.7	0.288
Model 4	110 ± 0.8	111 ± 0.6	111 ± 0.7	0.920	119 ± 1.4	117 ± 1.1	114 ± 1.7	0.210
Diastolic blood pressure (mm Hg)								
Model 1	73.9 ± 0.5	74.1 ± 0.4	74.5 ± 0.4	0.683	79.9 ± 0.7	79.0 ± 0.8	78.3 ± 0.8	0.345
Model 2	73.7 ± 0.5	73.9 ± 0.4	74.3 ± 0.4	0.592	79.1 ± 0.7	78.8 ± 0.7	78.7 ± 0.8	0.922
Model 3	74.1 ± 0.6	73.9 ± 0.4	74.1 ± 0.6	0.960	79.8 ± 1.0	78.8 ± 0.7	77.8 ± 1.2	0.590
Model 4	74.1 ± 0.6	73.9 ± 0.4	74.1 ± 0.6	0.979	79.9 ± 1.0	78.8 ± 0.7	77.6 ± 1.2	0.497
Fasting blood glucose (mg/dL)								
Model 1	88.2 ± 0.7	88.2 ± 0.7	89.5 ± 0.8	0.381	96.6 ± 2.1	97.6 ± 2.2	99.4 ± 2.2	0.657
Model 2	87.7 ± 0.7	88.1 ± 0.6	89.4 ± 0.7	0.213	96.2 ± 2.0	97.7 ± 2.1	96.6 ± 2.0	0.862
Model 3	85.3 ± 0.9	88.2 ± 0.6	91.7 ± 0.9	< 0.001	98.5 ± 2.7	97.7 ± 2.1	94.1 ± 3.0	0.599
Model 4	85.4 ± 0.9	88.3 ± 0.6	91.6 ± 0.9	< 0.001	98.7 ± 2.7	97.5 ± 2.0	93.9 ± 3.0	0.591
2-h blood glucose (mg/dL)								
Model 1	95.1 ± 1.5	94.3 ± 1.5	99.4 ± 1.5	0.045	110 ± 3.3	109 ± 3.4	110 ± 3.5	0.981
Model 2	95.4 ± 1.4	94.2 ± 1.4	98.4 ± 1.5	0.143	109 ± 3.3	109 ± 3.3	109 ± 3.4	0.994
Model 3	93.3 ± 1.9	94.3 ± 1.4	100 ± 1.9	0.029	115 ± 4.5	110 ± 3.3	102 ± 5.0	0.253
Model 4	93.5 ± 1.9	94.5 ± 1.4	100 ± 1.9	0.044	116 ± 4.5	109 ± 3.3	101 ± 5.0	0.221
Waist circumference (cm)								
Model 1	83.1± 0.4	84.3 ± 0.4	85.9 ± 0.4	< 0.001	101 ± 0.6	104 ± 0.6	104 ± 0.6	0.001
Model 2	84.4 ± 0.4	84.2 ± 0.3	84.4 ± 0.3	0.893	102 ± 0.5	104 ± 0.6	103 ± 0.6	0.346
Model 3	84.6 ± 0.4	84.2 ± 0.3	84.1 ± 0.4	0.761	102 ± 0.8	104 ± 0.6	103 ± 0.9	0.431
Model 4	84.2 ± 0.3	84.3 ± 0.2	84.4 ± 0.3	0.905	103 ± 0.6	103 ± 0.4	102 ± 0.6	0.773

Model 1 was crude; Model 2 was adjusted for age, gender, physical activity, smoking status, and educational levels; Model 3 was further adjusted for total energy intake, percentage of energy from carbohydrate, percentage of energy from fat, percentage of energy from protein, fiber, and magnesium; Model 4 was further adjusted for BMI; [a] Mean ± SEM; [b] Geometric mean ± SEM.

measurement.

Our results are in agreement with two prospective cohort studies that did not show any association between dietary GI, fasting or 2-h glucose,[11] and diabetes.[44] A lack of association between dietary GI with glycemic measurement has also been reported by other population- based studies.[11,34,45,46] Conversely, in the Nurses' Health Study II of women, those in the highest quintile of dietary GI had a higher risk of diabetes versus the lowest.[47] In another cohort study, increase per 10 units of dietary GI was positively associated with a 32% increased risk of diabetes;[48] however, in these studies,[47,48] a positive association between dietary GI and intake of refined bread and a negative association between dietary GI and intake of magnesium, fiber, fruits, vegetables, and dairy products were shown. Yet, in the current study, consistent with a previous study,[44] no association was observed between dietary GI and glucose concentration, may be because dietary GI was positively associated with fiber and magnesium intake, factors that might benefit glucose metabolism. In addition, in the current study, a high dietary GI was associated with high intakes of fruits, vegetables, legumes, and dairy products that are inversely associated with diabetes and impaired fasting glucose. Thus, our finding shows that a lower dietary GI is not necessarily important to determine the glycemic response and compared with dietary GI, the healthy diet is important to improve glycemic response.

Few studies have assessed the association between waist circumference and dietary GI and GL. We found an increased risk of waist circumference across tertiles of GI in nonobese subjects, an association which disappeared after further adjustment for BMI; however, this was expected because BMI is strongly associated with waist circumference. Our results are in accordance with randomized clinical trials that have shown larger decrease in fat mass in low GI diets than in high GI diets.[33, 49,50] In contrast, no association has been documented between GI, GL, and waist circumference by observational studies.[4,41]

In the present study, neither total nor LDL cholesterol were associated with dietary GI and GL. In agreement with our findings, no association between dietary GI and GL or both with total and LDL cholesterol has been reported in three cross-sectional studies.[5,11,36] However, clinical trial studies, most of which were performed with insulin- sensitive or overweight subjects, have shown that low-GI diets lower total and LDL cholesterol concentrations.[31,33]

Among Iranian population, the average proportions of total en-

ergy intake from carbohydrates is 65% and that of total carbohydrate consumption from both white rice and bread is 49%.[8] In this study, the dietary GI and GL values were similar to those of Asian countries,[4,5] but higher than those of Western countries.[13,35] This may have resulted from the differences in the major food contributors. Dietary GIs and GLs in Western populations are determined by a variety of food items, including potatoes, breakfast cereals, bread, and rice.[50,51] However, in our study, similar to other Asian contraries,[4,5] white rice and bread especially white bread was the major contributor of dietary GL and GI.

In this study, we found an inverse correlation between dietary GI, GL, protein, and fat. It is logic that when dietary fat and protein are reduced, these calories will be replaced by dietary carbohydrate. Many of the carbohydrate items such as bread, rice, pasta, noodle, potato, and snack foods, readily available among the Iranian population, have a high GI. Decrease in dietary fat, inadvertently leads to an increase in intake of dietary carbohydrate, and therefore increase in dietary GI and GL, leading to a reduction in HDL cholesterol and increased triglyceride concentrations; a problem prevalent among Tehranian.[14,53]

This study had a few limitations. The FFQ was not designed specifically to measure dietary GI and GL; however, the validity of the FFQ for total carbohydrate was acceptable among this population.[16] The limited published GI values for many food items included in Iranian foods is another limitation. In addition, because the cross-sectional nature of the present study precludes any causal inferences, future longitudinal studies are needed to provide stronger evidence on this association.

In conclusion, our results suggest that dietary GI was positively associated with high triglyceride and low HDL cholesterol concentrations in obese subjects, and dietary GL was positively associated with fasting and 2-h blood glucose after controlling for confounding factors.

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