Int J Nutr Sci 2023;8(3):150-157

International Journal of Nutrition Sciences

Journal Home Page: ijns.sums.ac.ir

ORIGINAL ARTICLE

Higher Glycemic Index and Load Could Increase Risk of Dyslipidemia

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ARTICLE INFO

Keywords:
Glycemic index
Glycemic load
Carbohydrate indices
Lipid profile

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Received: Feb 2, 2023
Revised: April 22, 2023
Accepted: May 1, 2023

ABSTRACT

Background: To quantify carbohydrates, various indicators such as glycemic load (GL) and glycemic index (GI) were introduced. In order to address the effect of dietary carbohydrate content on lipid profile, we investigated the relationship between dietary GI and GL with lipid profile in adults living in Shiraz, Iran.

Methods: In a cross-sectional study, 236 participants aged between 20 and 50 years were selected using cluster random sampling in Shiraz, Iran. For assessing the food intake, a 168-item food frequency questionnaire (FFQ) was utilized. Dietary GI and GL were calculated based on food items intake.

Results: Higher GI was associated with increased odds ratio (OR) of low-density lipoprotein-cholesterol (LDL-C, OR: 2.51; *p*-trend=0.008), non-high-density lipoprotein-cholesterol (HDL, OR: 2.34; *p*-trend=0.01) and LDL to HDL ratio (OR: 2.13; *p*-trend=0.02) in crude model. In adjusted model, direct association was observed between GI and total cholesterol (TC, OR: 2.40; *p*-trend=0.01), LDL-C (OR: 2.50; *p*-trend=0.01) and non-HDL-C (OR: 2.48; *p*-trend=0.01). Association was noted between higher GL with TC (OR: 2.50; *p*-trend=0.01), LDL-C (OR: 2.22; *p*-trend=0.02), non-HDL-C (OR: 2.49; *p*-trend=0.005) and LDL-C to HDL-C ratio (OR: 2.29; *p*-trend=0.01) in crude model. After adjusting for potential cofounder, association remained for TC (OR: 3.97; *p*-trend=0.01), LDL-C (OR: 4.39; *p*-trend=0.005) and non-HDL-C (OR: 3.72; *p*-trend=0.008).

Conclusion: Dietary GI and GL may have an association with higher odds of abnormal lipid profile. It seems that a diet with a low GI and GL (which full of whole grains, fruits, vegetables, nuts and legumes) can play an effective role in favorable lipid profile.

Please cite this article as: Soltani M, Gerami S, Ghaem Far Z, Rajabzadeh-Dehkordi M, Dehzad MJ, Najafi M, Faghih S. Higher Glycemic Index and Load Could Increase Risk of Dyslipidemia. Int J Nutr Sci. 2023;8(3):150-157. doi: 10.30476/IJNS.2023.97742.1219.

Introduction

Carbohydrate is one of the most important macronutrients in the diet, accounting for a major portion of daily energy intake (about 45% to 65%) (1). According to previous studies, carbohydrate intake is associated with serum lipid levels like total cholesterol (TC), low-density lipoprotein-cholesterol (LDL), high-density lipoprotein-cholesterol (HDL) and triglyceride, which are the risk factors for cardiovascular diseases (CVDs) (2-4). Therefore, in recent years, study of the effects of quantity and quality of carbohydrates on the incidence of CVDs has received much attention (5, 6).

To quantify carbohydrates, various indicators such as glycemic load (GL) and glycemic index (GI) have been introduced. GI is a measure of the potential for an increase in blood glucose of the carbohydrate content of a food compared to the reference food (generally pure glucose) (7). While, GL takes into account the amount of carbohydrates in addition to the type of carbohydrate and the GI of the food (8). By consuming foods with a high GI, blood glucose levels rise rapidly, followed by elevated insulin levels, resulting in the release of counter-regulatory hormones and increased plasma free fatty acids. Thus, there is a decrease in insulin sensitivity and the development of dyslipidemia (9).

Number of reports have shown direct relationship between GI and GL and CVDs risk factors such as serum LDL (10, 11). However, the findings of a number of studies have shown no clear association between these indices and some blood lipids (10). Moreover, there were differences in the results between two sexes. For instance, Knopp *et al.*, reported that in response to a high glycemic diet, the rate of decrease in HDL and increase in triglycerides level were higher among women than men. So in order to address the effect of dietary carbohydrate content on lipid profile, we investigated the relationship between dietary GI and GL with lipid profile in adults living in Shiraz, Iran.

Materials and Methods

The present study was a cross-sectional which was done on 236 participants aged between 20 and 50, selected by using cluster random sampling method. Individuals were included in the study if did not follow a particular diet and had no history of any chronic disease. A written consent form was signed by each participant. This study was confirmed by Shiraz University of Medical Sciences Ethics Committee (IR.SUMS.REC.1394.S146), while the detailed data on this study have been previously published (12-14). A 168-item validated food frequency questionnaire (FFQ) was used to assess the food intake (15). After completing the questionnaires, food intakes were changed to grams. NUTRITIONIST IV software (Version 7.0; N-Squared Computing, Salem, OR, USA) was used to compute the participants' intakes of nutrients and energy. Dietary GI was computed by this equation: GI×available carbohydrate/total available carbohydrate, in which the meaning of available carbohydrate was total carbohydrate intake minus fiber intake. Fiber and total carbohydrate content of foods was estimated utilizing The United States Department of Agriculture (USDA) food-composition table. GL of the foods was computed based on the equation: total GI×total available carbohydrate/100 (16-18).

For measuring lipid profile including HDL cholesterol, LDL cholesterol, triglyceride, and total cholesterol, the blood sample of individuals was provided. Lipid profile was measured by enzymatic kits (Pars Azmoon, Tehran, Iran). Anthropometric measurements such as waist circumference (WC), height, weight, and waist to hip ratio (WHR) were done by a nutritionist. A demographic questionnaire was used for gathering some information like sex, age, smoking and alcohol use. In this study, we used International Physical Activity Questionnaire (IPAQ) to evaluate the participants' physical activity (19).

Dyslipidemia was defined as HDL cholesterol less than 50 mg/dL for women and less than 40 mg/dL for men, triglyceride more than 150 mg/dL, LDL cholesterol more than 130 mg/dL, total cholesterol more than 200 mg/dL, and non-HDL-C more than 130 mg/dL (20). SPSS software (Version 20.0, SPSS Inc., Chicago, IL, USA) was used for statistical analyses. P-value<0.05 was considered significant. Normal distribution of the variables was assessed by Kolmogorov-Smirnov test. Analysis of variance (ANOVA) was used to assess the association between GI and GL tertile with quantitative variables and Chi-Square test was utilized to assess the association between qualitative variables. Also, ANCOVA test was applied to control the role of confounders. We used logistic regression models to evaluate the correlation between lipid profile and GI and GL tertile. In adjusted models, the role of BMI, sex, age, energy, physical activity and smoking history were controlled.

Results

Basic characteristics of the participants were shown in Table 1. Percent of males (p=0.009) was higher in the last tertile; but education was higher in the first tertile (p=0.03) of GI. Moreover, weight, height, WC and WHR were higher in the last tertile of GL (p<0.001 for all, except WC). Based on Table 2, the intake of energy, macronutrients, vitamin B_6, B_9, magnesium, whole and refined grains were higher in the last tertile of GL compared to the first teritle (p<0.001 for all, except whole grains). But for GI, participants in the last tertile had higher intake of whole and refined grains, but less intake of vegetables, fruits, legumes and dairy compared to the first tertile (p<0.001 for all).

Table 1: Baseline characteristics of study population across the tertile of GI and GL.

Variable	GI				GL			
	T_1	T_2	T_3	*P* value	T_1	T_2	T_3	*P* value
Age (year)	47.78±12.82	46040±10.04	45.76±12.18	0.93	46.59±11.58	46.03±11.54	45.33±12.18	0.79
Weight (41)	74.51±15.43	75.35±13.92	74.39±13.53	0.90	68.47±14.71	76.85±13.49	78.63±12.75	<0.001
Height (cm)	160.93±8.55	162.20±10.90	164.32±9.27	0.08	157.64±9.22	163.32±8.60	166.15±9.21	<0.001
BMI (kg/m^2)	28.75±5.29	28.51±4.07	27.54±4.51	0.23	27.55±5.55	28.81±4.46	28.45±3.92	0.22
Waist (cm)	94.42±11.75	94.89±10.80	93.33±11.51	0.68	90.77±11.89	94.64±11.19	96.96±10.24	0.002
WHR	0.89±0.08	0.91±0.07	0.90±0.07	0.17	0.88±0.08	0.89±0.06	0.93±0.07	<0.001
Physical activity (MET.h/day)	15.19±2.24	27.73±6.64	21.08±4.16	0.15	17.93±3.95	19.50±4.73	25.50±4.91	0.46
Gender, male (%)	28.6	44.0	51.9	0.009	19.5	40.8	61.4	<0.001
Smoking history, no (n) (%)	90.5	89.3	84.4	0.45	87.0	89.5	88.0	0.89
Education, high school and higher (n (%))	61.9	50.0	59.3	0.03	54.0	56.1	61.4	0.49

GI, glycemic index; GL, glycemic load; BMI, body mass index; WHR, waist to hip ratio. Values were mean for continuous and percentage for categorical variables using ANOVA for continuous and Chi-square test for categorical variables. *P*-value<0.05 was considered significant.

Table 2: Nutrients and food group's intakes of study population across the tertile of GI and GL.

Variable	GI				GL			
Nutrient items	T_1	T_2	T_3	*P* value	T_1	T_2	T_3	*P* value
Energy (kcal/d)	2713.1±113.2	2823.19±130.7	2788.9±114.2	0.79	1934.7±52.82	2608.1±59.54	3701.2±119.3	<0.001
Carbohydrate (g/d)	402.35±16.48	446.77±20.71	450.05±19.63	0.13	288.7±7.89	400.58±7.79	593.75±17.60	<0.001
Protein (g/d)	90.36±4.47	91.59±4.19	88.07±3.83	0.83	64.07±1.99	83.89±2.32	119.6±4.57	<0.001
Fat (g/d)	89.60±4.80	81.75±4.48	76.10±3.62	0.08	62.63±2.64	80.20±3.80	103.61±4.92	<0.001
SFA (mg/d)	24.82±1.56	22.84±1.10	20.80±1.00	0.07	17.32±0.77	22.14±8.57	28.71±13.93	<0.001
Vitamin B_6 (mg/d)	2.20±0.82	2.24±0.91	2.05±0.83	0.34	1.58±0.43	2.01±0.56	2.84±0.91	<0.001
Vitamin B_9 (µg/d)	684.14±26.93	726.88±30.59	773.58±29.31	0.08	514.69±13.92	693.34±15.35	954.52±27.82	<0.001
Magnesium (mg/d)	497.80±22.87	487.52±25.72	426.64±25.18	0.08	330.91±13.40	437.68±17.30	632.37±26.24	<0.001
Food items								
Whole grains (g/d)	176.51±13.86	207.01±14.48	267.09±17.64	<0.001	188.35±9.05	211.23±15.13	245.32±20.08	0.03
Refined grains (g/d)	323.44±16.99	458.54±15.46	593.21±19.14	<0.001	392.88±13.28	449.18±16.41	516.23±27.72	<0.001
Vegetables (g/d)	529.62±23.54	407.41±23.21	325.93±16.91	<0.001	447.68±19.76	436.10±23.11	391.87±26.44	0.20
Fruits (g/d)	537.89±29.97	562.70±32.09	412.35±38.84	0.004	484.19±22.02	515.83±29.95	513.86±45.04	0.77
Legumes (g/d)	88.40±7.95	54.11±3.38	48.16±3.22	<0.001	69.01±6.06	62.02±5.14	62.24±6.34	0.64
Nuts (g/d)	22.62±3.76	18.04±2.21	14.59±3.17	0.19	22.67±3.89	15.88±1.61	17.16±3.47	0.28
Dairy (g/d)	358.31±24.44	298.50±15.77	234.74±15.38	<0.001	302.23±17.89	328.74±18.17	268.73±23.14	0.10
Meats (g/d)	55.55±8.23	56.59±4.27	53.51±5.15	0.94	64.41±3.04	53.32±3.79	48.42±9.25	0.18

GI, dietary glycemic index; GL, dietary glycemic load. Values were presented as mean (42) using ANOVA. *P* value<0.05 were considered significant.

Table 3: Lipid profile of study population across the tertiles of GI and GL.

Variables	GI				GL			
	T_1	T_2	T_3	*P* value	T_1	T_2	T_3	*P* value
TG (mg/dL)								
Crude model	118.91±73.96	116.92±62.54	128.48±54.57	0.49	109.97±67.49	116.69±64.34	136.31±59.35	0.02
Adjusted model[a]	118.98±74.38	117.46±63.31	128.48±54.57	0.43	109.60±67.86	116.69±64.34	137.28±59.76	0.07
TC (mg/dL)								
Crude model	174.08±42.23	175.00±34.31	190.79±49.69	0.02	173.92±48.93	177.32±41.99	187.59±37.32	0.11
Adjusted model[a]	173.68±42.33	174.93±34.72	190.79±49.69	0.01	173.48±49.10	177.32±41.99	187.83±37.69	0.05
LDL (mg/dL)								
Crude model	103.41±31.59	103.70±28.03	121.28±38.09	0.001	101.98±34.92	105.81±32.38	119.38±31.77	0.002
Adjusted model[a]	103.24±31.74	103.41±28.35	121.28±38.09	0.001	101.77±35.10	105.81±32.38	119.50±32.15	0.002
HDL (mg/dL)								
Crude model	37.22±12.55	37.06±10.03	39.79±10.24	0.22	38.89±11.73	36.51±10.81	38.56±10.70	0.35
Adjusted model[b]	37.07±12.55	37.19±10.10	39.79±10.24	0.15	38.75±11.73	36.51±10.81	38.71±10.75	0.33
Non-HDL								
Crude model	136.85±41.24	137.93±32.57	151.00±45.40	0.05	135.02±45.28	140.81±39.98	149.02±35.33	0.08
Adjusted model[a]	137.03±41.50	137.79±33.20	151.00±45.40	0.36	135.17±45.65	140.81±39.98	149.31±35.93	0.56
LDL to HDL ratio								
Crude model	3.20±1.85	3.06±1.47	3.17±1.20	0.84	2.82±1.25	3.22±1.79	3.38±1.50	0.06
Adjusted model[a]	3.22±1.87	3.05±1.49	3.17±1.20	0.51	2.84±1.26	3.22±1.79	3.39±1.52	0.22

GI, dietary glycemic index; GL, dietary glycemic load; TG, triglyceride; TC, total cholesterol; LDL, low density lipoprotein; HDL, low density lipoprotein. [a] Adjusted for age, physical activity, energy intake, BMI, sex and smoking history. [b] Adjusted for age, physical activity, energy intake, BMI and smoking history. Values were presented as mean±SD using ANCOVA. *P* value<0.05 was considered significant.

The mean and standard deviation of lipid profile in each tertile of GI and GL were demonstrated in Table 3. Participants in the highest tertile of GI had higher mean of TC and LDL levels in both crude (p=0.02 and p=0.001) and adjusted model (p=0.01 and p=0.001). Furthermore, for GL tertile, the same trend was seen in TG and LDL levels and the highest tertile was also associated with higher TG (p=0.02) and LDL (p=0.002) levels in both crude and adjusted model. According to Table 4, the chance of increasing in LDL-C (OR: 2.51; 95% CI: 1.24-5.07; p-trend=0.008) and non-HDL (OR: 2.34; 95% CI: 1.22-4.49; p-trend=0.01) were higher in associated with GI crude model. But in adjusted model, we observed direct association between GI and TC (OR: 2.40; 95% CI: 1.14–5.04; p-trend=0.01), LDL-C (OR: 2.50; 95% CI: 1.21-5.19; p-trend=0.01) and non-HDL-C (OR: 2.48; 95% CI: 1.24-4.93; p-trend=0.01). Furthermore, the developing of TC (OR: 2.50; 95% CI: 1.18-5.30; p-trend=0.01), LDL-C (OR: 2.22; 95% CI: 1.07-4.57; p-trend=0.02) and non-HDL-C (OR: 2.49; 95% CI: 1.31-4.75; p-trend=0.005) were more in the higher tertile of crude model of GL and also, after adjusting for potential cofounder, association remained for TC (OR: 3.97; 95% CI: 1.38-11.39; P-trend=0.01), LDL-C (OR: 4.39; 95% CI: 1.57-12.26; p-trend=0.005) and non-HDL-C (OR: 3.72; 95% CI: 1.40-9.89; p-trend=0.008).

Discussion

The results of this cross-sectional study showed that higher GI and GL was correlated with a higher risk of elevated TC, LDL-C and non-HDL cholesterol, but not triglycerides, HDL-C and LDL-C/HDL-C. Indeed, the study revealed a positive relationship between dietary GL or GI and lipid profile which serve as a risk factor for cardiovascular diseases. In accordance with the result of the present study, some studies did not find any association between triglycerides and GL and GI (21-23). For example, in

Table 4: Crude and multivariable-adjusted OR and 95% CIs across tertile of GI and GL.

Variable	GI				GL			
	T_1	T_2	T_3	P_{trend}	T_1	T_2	T_3	P_{trend}
TG (mg/dL)								
Crude model	Ref.	0.76 (0.36, 1.59)	1.20 (0.60, 2.39)	0.61	Ref.	1.27 (0.59, 2.70)	1.73 (0.84, 3.57)	0.12
Adjusted model[a]	Ref.	0.72 (0.33, 1.55)	1.15 (0.56, 2.36)	0.70	Ref.	1.19 (0.52, 2.72)	1.92 (0.69, 5.29)	0.21
TC (mg/dL)								
Crude model	Ref.	0.65 (0.29, 1.45)	1.95 (0.98, 3.90)	0.05	Ref.	1.52 (0.68, 3.39)	2.50 (1.18, 5.30)	0.01
Adjusted model[a]	Ref.	0.63 (0.26, 1.49)	2.40 (1.14, 5.04)	0.01	Ref.	1.95 (0.81, 4.69)	3.97 (1.38, 11.39)	0.01
LDL (mg/dL)								
Crude model	Ref.	0.90 (0.41, 1.98)	2.51 (1.24, 5.07)	0.008	Ref.	1.19 (0.54, 2.59)	2.22 (1.07, 4.57)	0.02
Adjusted model[a]	Ref.	0.87 (0.38, 1.97)	2.50 (1.21, 5.19)	0.01	Ref.	1.59 (0.68, 3.70)	4.39 (1.57, 12.26)	0.005
HDL (mg/dL)								
Crude model	Ref.	1.25 (0.58, 2.66)	0.61 (0.30, 1.22)	0.16	Ref.	2.00 (0.94, 4.26)	1.18 (0.59, 2.33)	0.64
Adjusted model[b]	Ref.	1.13 (0.50, 2.51)	0.51 (0.24, 1.07)	0.07	Ref.	2.38 (1.04, 5.43)	2.16 (0.76, 6.15)	0.11
Non-HDL								
Crude model	Ref.	1.41 (0.75, 2.65)	2.34 (1.22, 4.49)	0.01	Ref.	1.84 (0.96, 3.51)	2.49 (1.31, 4.75)	0.005
Adjusted model[a]	Ref.	1.23 (0.63, 2.39)	2.48 (1.24, 4.93)	0.01	Ref.	2.10 (1.01, 4.34)	3.72 (1.40, 9.89)	0.008
LDL to HDL ratio								
Crude model	Ref.	1.12 (0.59, 2.11)	2.13 (1.09, 4.16)	0.02	Ref.	1.59 (0.83, 3.04)	2.29 (1.18, 4.41)	0.01
Adjusted model[a]	Ref.	0.85 (0.44, 1.66)	1.87 (0.93, 3.77)	0.09	Ref.	1.53 (0.74, 3.14)	2.46 (0.93, 6.50)	0.06

GI, dietary glycemic index; GL, dietary glycemic load; TG, triglyceride; TC, total cholesterol; LDL, low density lipoprotein; HDL, low density lipoprotein. [a]Adjusted for age, physical activity, energy intake, BMI, sex and smoking history. [b]Adjusted for age, physical activity, energy intake, BMI and smoking history. These values were odds ratio (95% CIs). Obtained from logistic regression. *P* value<0.05 was considered significant.

a cross-sectional study on Spanish rural population, no significant correlation was found between triglycerides and GL and GI (22). This may be for beta-cell failure which happens only after long-term increase of insulin release which play a role in lipid accumulation to be still effective in young people, and dietary GL effects of glucose may not be observed yet (22, 24). On the other hand, in another study, dietary GL had an inverse correlation with blood total cholesterol and LDL-C in hospitalized Chinese patients (25). On the contrary, result of the healthy twin cohort study showed that GI and GL were positively related to triglycerides in participants with greater body mass index (26). It has been identified increased level of insulin resistance in obese participants, and play a key role in higher triglycerides level observed in this population (26, 27).

In line with the present study, some studies found an association between GL and GI and LDL-C (22, 28, 29), LDL/HDL (30), and non-HDL cholesterol (31). It is explained that lower GL is contributed to suppressed 5-hydroxyl-3-methylglutaryl-CoA reductase activity through reduced insulin stimulation. Thus, increased LDL-C receptors on the surface of the cells result in decreased circulating LDL-C levels (22, 24, 32). Regarding HDL-C levels, no significant correlation with GL and GI was seen in other consistent studies (21, 33). A cross-sectional study on 87 female participants failed to find any significant association between GI and HDL-C (21). On the other hand, some were able to find an inverse association between GL and GI with HDL-C (31, 34, 35). For instance, Murakami *et al.* showed that GL was inversely related to HDL-C in a cross-sectional study of 1354 Japanese female farmers (35). One possible explanation could be larger study population in the mentioned Japanese research.

It is proved that rapid spikes in blood glucose levels happen following high GI and GL intakes. This phenomenon leads to huge insulin secretion and then inhibition of counter-regulatory hormone production. Insulin is known for its anabolic effects on the body; it reduces gluconeogenesis and lipolysis; in addition, insulin promotes lipogenesis, glycogenesis, and cellular glucose uptake (24, 36). Secretion of counter-regulatory hormones is the body's response to insulin-induced hypoglycemia. Counter-regulatory hormones initiate lipolysis in the adipose tissue which results in higher free fatty acid levels in blood and dyslipidemia (24, 37). Additionally, higher insulin secretion and insulin resistance, per se, triggers an imbalanced release of free fatty acids from the liver and muscles following disturbed lipolysis (38).

There were limitations in this study based that should be considered for future researches. First, due to the cross-sectional design of the study, revealing the exact correlation between lipid profile and GL or GI over time is difficult. In addition, even food frequency questionnaire is a validated and reliable tool in evaluating glycemic and insulin indices, it is dependent on the memory of participants, and thus it can contribute to bias. Last but not the least, this study was performed on a healthy population with the age range of 20-50 years old, hence insulin resistance and dysregulated metabolism of glucose and lipid is less common.

Conclusion

This study suggests that dietary GI and GL have an association with higher odds of abnormal blood lipids such as TC, LDL-C, and non-HDL cholesterol. It seems that a diet with a low GI and GL (Full of whole grains, fruits, vegetables, nuts and legumes) can play an effective role in favorable lipid profile. Also, it might be beneficial to conduct prospective studies or clinical trials in an attempt to investigate the correlation of blood lipids and glycemic indices based on food intake reports over a long duration.

Acknowledgement

The authors received no financial support for the research, authorship, and/or publication of this article to acknowledge.

Conflict of Interest

None declared.

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