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Otros

Usual dietary glycemic load is associated with cardiometabolic risk factors in physically active Brazilian middle-aged men

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Abstract

Introduction: The effects of dietary glycemic load (GL) on cardiometabolic risk factors in physically active subjects are not completely known.

Objective: This cross-sectional study assessed the association of habitual dietary GL with cardiometabolic risk factors in physically active Brazilian middle-aged men.

Methods: One-hundred seventy-six subjects (Age: 50.6 ± 5.0 years, BMI: 25.5 ± 3.6 kg/m²) were evaluated. Anthropometry, lifestyle features, insulin resistance, oxidative stress biomarkers (8-iso-prostaglandin F2α; 8-iso-PGF2α and 8-hydroxydeoxyguanosine; 8-OHdG) and lipid profile were assessed. Dietary intake was estimated through a quantitative food frequency questionnaire.

Results: The dietary GL was positively associated with free fatty acid concentrations (β = 0.311, r² = 0.13, P-value = 0.034) and triglycerides/HDL cholesterol ratio (β = 0.598, r² = 0.19, P-value = 0.028) regardless of confounding factors (central obesity, red meat consumption, age and energy intake). The oxidative stress biomarker, 8-OHdG, was associated with habitual dietary GL (β = 0.432, r² = 0.11, P-value = 0.004), regardless of previous confounding factors plus excessive alcohol consumption, iron intake and current smoking status.

Conclusions: The dietary GL was positively associated with lipid profile (free fatty acid concentrations and triglycerides/HDL cholesterol ratio) and oxidative stress biomarker (8-OHdG). These results indicate potential harmfulness of diet with higher GL to cardiometabolic risk factors in middle-aged men, even in physically active individuals.

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Key words: *Food habits. Carbohydrate. Triglycerides. High-density lipoproteins. Oxidative stress.*

LA CARGA GLUCÉMICA DIETÉTICA HABITUAL SE ASOCIA CON FACTORES DE RIESGO CARDIOMETABÓLICO EN HOMBRES BRASILEÑOS DE MEDIANA EDAD FÍSICAMENTE ACTIVOS

Resumen

Introducción: Los efectos de la carga glucémica (CG) de la dieta sobre los factores de riesgo cardiometabólico en sujetos físicamente activos no están establecidos por completo.

Objetivo: Este estudio transversal evaluó la asociación entre la CG de la dieta habitual y los factores de riesgo cardiometabólico en hombres brasileños de mediana edad físicamente activos.

Métodos: Ciento setenta y seis sujetos (índice de masa corporal: 25,5 ± 3,6 kg/m²; edad: 50,6 ± 5,0 años) fueron evaluados. Antropometría, características del estilo de vida, la resistencia a la insulina, biomarcadores del estrés oxidativo (8-iso-prostaglandina F2α, 8-iso-PGF2α y 8 hidroxideoxiguanosina, 8-OHdG) y el perfil lipídico fueron evaluados. La ingesta dietética se estimó por medio de un cuestionario cuantitativo de frecuencia de consumo.

Resultados: La CG de la dieta se asoció positivamente con las concentraciones de ácidos grasos libres (β = 0,311, r² = 0,13, P = 0,034) y la razón triglicéridos/colesterol HDL (β = 0,598, r² = 0,19, P = 0,028), independientemente de los factores de confusión (obesidad central, consumo de carne roja, edad e ingesta calórica). El biomarcador del estrés oxidativo, 8-OHdG, también se asoció con CG de la dieta habitual (β = 0,432, r² = 0,11, P = 0,004), independientemente de los factores de confusión anteriores más el consumo excesivo de alcohol, la ingesta de hierro y tabaquismo actual.

Conclusiones: La CG de la dieta se asoció positivamente con el perfil lipídico (concentraciones de ácidos grasos libres y razón triglicéridos/HDL colesterol) y el biomarcador de estrés oxidativo 8-OHdG. Estos resultados indican el potencial de nocividad de una dieta con mayor CG respecto a los factores de riesgo cardiometabólico en hombres de mediana edad, incluso en aquellos físicamente activos.

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Palabras clave: *Hábitos alimentares. Hidratos de carbono. Triglicéridos. Lipoproteínas de alta densidad. Estrés oxidativo.*

Abbreviations

8-iso-PGF2α: 8-iso-prostaglandin F2α.
8-OHdG: 8-hydroxydeoxyguanosine.
BMI: body mass index.
CVD: Cardiovascular diseases.
DM2: type 2 diabetes.
FFA: Free fatty acids.
FFQ: food frequency questionnaire.
GI: glycemic index.
GL: glycemic load.
HDL-C: high density lipoprotein.
HOMA-IR: Homeostatic model of assessment of insulin resistance.
NADH: Reduced nicotinamide adenine dinucleotide.
NAD+: Oxidized nicotinamide adenine dinucleotide.
TG/HDL-C: ratio between triglycerides and high density lipoprotein.
TG: triglycerides.

Introduction

Cardiovascular diseases (CVD) are the leading cause of mortality worldwide. The World Health Organization estimated that approximately 30% of deaths from CVD occurred in the world in 2008 and that this rate will reach higher proportions in 2030[1]. The occurrence of these diseases is highly influenced by environmental factors, particularly, the quality of food intake[2] and the level of physical activity[3].

The role of carbohydrate intake as a risk factor for manifestation of chronic diseases has received important attention in the scientific community. The dietary glycemic load (GL), obtained by multiplying the glycemic index (GI) of a food/meal by its available carbohydrate content[4], reflects the quality and the amount of consumed carbohydrate. The consumption of high-GL diets leads to postprandial hyperglycemia/hyperinsulinemia, increasing the risk for CVD[5]. Moreover, the increase of dietary GL has been associated with CVD risk factors, such as reduced concentrations of high density lipoprotein (HDL-C), high concentrations of triglycerides[6-8] and higher concentrations of oxidative stress marker[9]. Thus the dietary pattern adopted by the modern society, characterized by high consumption of carbohydrate-rich foods and high-GI diet[10], may increase the risk for CVD in the population.

Most of the previous studies associating dietary GI/GL with cardiovascular risk factors were typically conducted in women,[8,11] type 2 diabetes (DM2)[12] and obese subjects[13]. On the other hand, the relationship between dietary GL and cardiometabolic risk factors in physically active individuals, to our knowledge, is not clear yet. Taking into consideration that regular physical activity has beneficial effects against CVD[14] and that aging is a potent cardiovascular risk factor[1] this cross-sectional study was designed to assess the association of habitual dietary GL with cardiometabolic risk factors in physically active middle-aged men.

Materials and methods

Study population

One-hundred seventy-six men aged between 40 and 59 years participated in this study. The staff members of Universidade Federal de Viçosa, Brazil were recruited by systematic sampling using interview as previously described[15]. The following exclusion criteria were considered: body weight changes greater than 3 kg in the three months preceding the beginning of the study, thyroid diseases, heart failure, cerebrovascular diseases, infectious diseases, inflammatory diseases, gastrointestinal tract diseases, liver disease, chronic kidney disease and/or history of kidney stones, cancer in the previous ten years, eating disorders (anorexia and bulimia), food allergies, changes in the level of physical activity and in the eating habit in the three months preceding the study. Subjects using vitamin supplements, diuretics or drugs that affect food intake and/or the metabolism of nutrients, pacemaker and/or prosthetic limbs users, elite athletes and subjects who were not physically active (number of steps < 10,000)[16] were also excluded.

The study was conducted according to the Declaration of Helsinki guidelines and all procedures involving human subjects were approved by the Ethics Committee in Human Research of the Federal University of Viçosa (Reference nº 069/2010). Written informed consent was obtained from all participants of the study.

Dietary intake assessment

A food frequency questionnaire (FFQ), developed for the Brazilian population, was used to assess the usual dietary intake of the participants[17]. Daily food consumption was estimated as frequency x portion x size for each consumed food item. Nutrient intake was assessed using the software Dietpro® version 5.5i (AS Systems, Viçosa, Brazil), using mainly two Brazilian nutritional composition tables or an international composition table when the needed nutritional information was not described in these tables conforming described previously[18].

The GI values for most foods listed on the FFQ were obtained from the University of Sydney GI data base website[19]. The GI of foods not listed in that database was estimated considering the GI of foods that had similar nutritional composition and methods of preparation. Dietary GI and GL were calculated using the formulas described by Levitan et al.:[11] [Dietary GI $= \sum_{foods}$ Carbohydrate (g) in a serving of food $\times$ Frequency of consumed food $\times$ GI $\sum_{foods}$ Carbohy-

drate (g) in a serving of food × Frequency of consumed food; Dietary GL = $\sum_{foods}$ Carbohydrate (g) in a serving of food × Frequency of consumed food × GI/100]

It should be noted, however, that we considered only the available carbohydrate for these calculations.

Anthropometric and body composition assessments

Body weight, height and waist circumference were taken using standard measurement procedures, as previously described[15]. Body mass index was calculated as weight (kg) divided by height squared (m²). The cut-off used for central obesity was waist circumference ≥ 94 cm[20].

Total body fat percentage was determined by total body scanning with a dual energy X-ray absorptiometry (GE/Lunar, Madison, WI, USA; enCORE software version 13.31).

Lifestyle co-variables

The participants were asked about their current smoking status (yes/no) and habitual quantity/frequency of alcohol consumption. Excessive alcohol consumption was defined as a daily ingestion above 21 units per week[21]. So, in the statistical analysis, were considered excessive alcohol consumer (yes) or no alcohol consumer (no).

The habitual physical activity was estimated by the mean number of daily steps (7 consecutive days) measured by Digi-Walker SW-200 pedometer (Yamax Corporation, Tokyo, Japan), according to the instructions previously described[15]. A minimum of 10,000 steps/day was considered the cut-off point to classify individuals as active[16].

Sample collection and analysis

Blood samples were collected from the antecubital vein after 12-hour overnight fasting. The serum was separated from whole blood by centrifugation at 2.225 *g* for 15 min at room temperature (2-3 Sigma, Sigma Laborzentrifuzen, Osterodeam Harz, Germany) and was immediately frozen at -80° C until analysis.

Glucose, insulin and the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) index were assessed by protocol previously described[15].

Free fatty acids (FFA) were determined by a kinetic spectrophotometry method using the kit EnzyChrom Free Fatty Acid Assay (Bioassay Systems, Hayward, CA). Serum HDL-C and triglyceride (TG) were determined by the enzymatic colorimetric method (Cobas Mira Plus - Roche Diagnostics GmbH, Montclair, NJ, USA). The atherogenic index was calculated by the ratio between triglycerides and HDL-C (TG/HDL-C ratio)[22].

The urine samples were collected after 12-hour overnight fasting and frozen at -80° C until analysis. The concentrations of 8-iso-prostaglandin F2α (8-iso-PGF2α) (Oxford, MI, USA) and 8-hydroxydeoxyguanosine (8-OHdG) (Cayman, MI, USA) were determined by competitive ELISA according to manufacturer's instructions. Although Cayman's kit recognizes the 8-OHdG from DNA, the ELISA values are always higher than LC/MS inasmuch as this method also detects 8-hydroxyguanosine and 8-hydroxyguanine from either DNA or RNA. The values for urinary 8-iso-PGF2α and 8-OHdG were normalized by milligrams of urinary creatinine which was measured by a kinetic colorimetric method using a Bioclin commercial kit (Cobas Mira Plus - Roche Diagnostics GmbH, Montclair, NJ, USA).

Statistical analysis

Normal distribution of data was assessed by the Shapiro-Wilk test. Non-normally distributed variables were log-transformed before statistical analysis. The nutrients, dietary GI, and dietary GL were energy adjusted using the residual method as previously applied[11,23]. The comparison of nutrients consumption among lower or higher dietary GL (estimated by median) was performed by t test. Such methodological procedure of dividing participants into groups of risk has been used in epidemiologic study previously[8].

To evaluate the associations of dietary GL, available carbohydrate and dietary GI with FFA and with TG/HDL-c ratio we used linear multiple regression controlled by the occurrence of central obesity (waist circumference ≥ 94 cm), red meat consumption (g/d), age (years) and energy intake (kcal/d). To evaluate the associations of dietary GL, available carbohydrate and dietary GI with 8-iso-PGF2α and with 8-OHdG we used linear multiple regression controlled by previous confounding factors plus others important confounding factors for oxidative stress such as excessive alcohol consumption (yes/no), daily iron intake (mg/d) and current smoking status (yes/no).

In addition, the Spearman correlation coefficient was used to investigate the correlation of HOMA-IR with FFA and with TG/HDL as well as that of dietary GL with available carbohydrate and with dietary GI.

Data processing and analysis were performed using the software STATA version 9.1 (Stata Corp., College Station, TX, USA). The P-value <0.05 was considered as statistically significant.

Results

Anthropometric, clinical and lifestyle characteristics of the participants are shown in table I. The mean BMI was equivalent to 25.5 kg/m² and the number of steps per day corresponded to 13,591. The central obesity occurrence (waist circumference ≥ 94 cm) was 31.2%.

Table I
Anthropometric, clinical anf lifestyle characteristics of participants

Variables	Values[a]
Age (years)	50.6 ± 5.0
Body mass index (kg/m²)	25.5 ± 3.6
Waist Circumference (cm)	89.1 ± 9.8
Total body fat (%)	21.0 ± 7.5
HDL-C (mg/dL)	47.9 ± 12.6
HOMA-IR	1.29 ± 1.09
Triglycerides (mg/dL)	127.7 ± 86.0
TG/HDL-C ratio	3.0 ± 2.8
Free fatty acid (mmol/L)	0.78 ± 0.30
8-OHdG (ng/mg creatinine)	8.7 ± 3.1
8-iso-PGF2α (ng/mg creatinine)	1.53 ± 1.22
Central obesity n (%)	55 (31.2)
Excessive alcohol consumption n (%)	29 (16.5)
Number of steps: per day	13,591 ± 2,798
Smoker n (%)	26 (14.8)

Abbreviation: HDL-C, high density lipoprotein; TG/HDL-C ratio, ratio between triglycerides and high density lipoprotein; 8-iso-PGF2α, 8-iso-prostaglandin F2α; 8-OHdG, 8-hydroxydeoxyguanosine. [a]Values are mean ± SD of 176 individuals for continuous variables; and number (frequency) for categorical variables.

Regarding the dietary habits, according to the median of dietary GL consumed by participants, those subjects who consumed a diet with lower GL (<105.2 units) also consumed a lower GI diet, lower amount of carbohydrate, as well as higher amount of protein, fat, red meat and iron than those who consumed a diet with higher GL (≥ 105.2 units) (Table II).

Multiple linear regression models were applied to assess the relationship of dietary GL, dietary GI and available carbohydrate with blood lipid profile (FFA concentrations and TG/HDL-C ratio) and with the concentrations of oxidative stress markers. Interestingly, TG/HDL-C ratio and concentrations of FFA and of 8-OHdG, a marker of oxidative DNA damage were positively associated with dietary GL, regardless of interfering variables (Table III). Moreover, there was a

positive association of TG/HDL-C ratio and 8-OHdG concentrations with the dietary available carbohydrate, regardless of interfering variables. However, there were no associations between dietary GI and the evaluated variables (Table III).

Finally, was verified a positive correlation of blood lipid profile (TG/HDL-C ratio and FFA concentrations) with HOMA-IR (Fig. 1) as well as of dietary GL with available carbohydrate and dietary GI (Fig. 2).

Discussion

This cross-sectional study was carried out to evaluate the association of habitual dietary GL with cardiometabolic risk factors in physically active Brazilian middle-aged men.

An important finding was the positive association of dietary GL with the TG/HDL-C ratio in this population, after adjustment for the confounding variables. The increased concentrations of TG and the reduced concentrations of HDL-C promoted by the consumption of high-carbohydrate diets may be related to decreased clearance of TG-rich particles. The clearance of TG occurs through the action of lipoprotein lipase (LPL) both in the TG stored in adipose tissue or skeletal muscle. In the skeletal muscle LPL is also involved in TG oxidation. However, the competition between chylomicron from enterocytes and VLDL-C particles (from liver) by LPL leads to its accumulation in plasma, which stimulates cholesterol ester transfer from HDL-C and LDL-C to those TG-rich lipoproteins. Subsequently, the content of TG from HDL-C particles could be hydrolyzed by hepatic lipase forming small particles of such lipoprotein that are rapidly removed via circulation[24].

Similar results were reported in the Health Workers Cohort Study[7] with men and women aged 20 to 70 years and by other[11] in women (≥ 45 years) without diabetes/DCV showing a positive association of dietary GL with TG concentrations and a negative association with HDL-C concentrations. In contrast, a

Table II
Food and nutrient consumption, according to median energy-adjusted glycemic load intake

	Lower Glycemic Load < 105.2 units	Higher Glycemic Load ≥ 105.2 units	P-value[a]
Energy intake (kcal/d)	1,421 ± 455.5	1,518 ± 496.7	0.174
Glycemic Index	62.8 ± 3.7	67.8 ± 6.1*	<0.001
Carbohydrate (g/d)	176.2 ± 23.4	220.6 ± 25.3*	<0.001
Protein (g/d)	73.8 ± 12.3	60.9 ± 9.6*	<0.001
Fat (g/d)	50.7 ± 9.1	36.6 ± 10.0*	<0.001
Fiber (g/d)	21.9 ± 5.7	23.7 ± 6.5	0.063
Iron (mg/d)	7.3 ± 1.2	6.8 ± 1.2*	0.002
Red meat (g/d)	82.1 ± 39.7	58.5 ± 22.0*	<0.001

Values are mean ± SD of 176 individuals. [a]P-value from Student *t*-test. *denotes statistical difference from diet with lower glycemic load.

Table III
Multiple linear regression models with glycemic load, available carbohydrate and glycemic index as main independent variable

Dependent variables	Main independent variable	β	CI 95%	R²	P-value
Free fatty acid (mmol/L)[a]	Glycemic load	0.311	0.02354 ; 0.5992	0.13	0.034[*]
	Available carbohydrate	0.328	-0.003749 ; 0.09513	0.12	0.078
	Glycemic index	0.473	-0.1368 ; 1.08449	0.11	0.127
TG/HDL-C ratio[a]	Glycemic load	0.598	0.06646 ; 1.13069	0.19	0.028[*]
	Available carbohydrate	0.747	0.0730 ; 1.4223	0.19	0.030[*]
	Glycemic index	0.591	-0.54334 ; 1.7256	0.17	0.305
8-iso-PGF2α (ng/mg creatinine)[b]	Glycemic load	0.407	-0.312702 ; 1.12710	0.12	0.266
	Available carbohydrate	0.293	-0.615624 ; 1.202685	0.12	0.525
	Glycemic index	0.964	-0.5336651 ; 2.46290	0.12	0.205
8-OHdG (ng/mg creatinine)[b]	Glycemic load	0.432	0.1382791 ; 0.726494	0.11	0.004[*]
	Available carbohydrate	0.614	0.244211 ; 0.98540	0.12	0.001[*]
	Glycemic index	0.222	-0.4102 ; 0.85448	0.03	0.489

Abbreviation: β, beta coefficient; CI, confidence interval; TG/HDL-C ratio, ratio between triglycerides and high density lipoprotein; 8-iso-PGF2α, 8-iso-prostaglandin F2α; 8-OHdG, 8-hydroxydeoxyguanosine. [a]P-value from the linear regression model adjusted for occurrence of central obesity (waist circumference ≥ 94 cm), red meat intake (g/d), age (years) and energy intake (kcal/d). [b]P-value from the linear regression model adjusted for occurrence of central obesity (waist circumference ≥ 94 cm), red meat intake (g/d), age (years), energy intake (kcal/d), excessive alcohol consumption (yes/no), iron intake (mg/d) and current smoking status (yes/no). [*]denotes significant relationship.
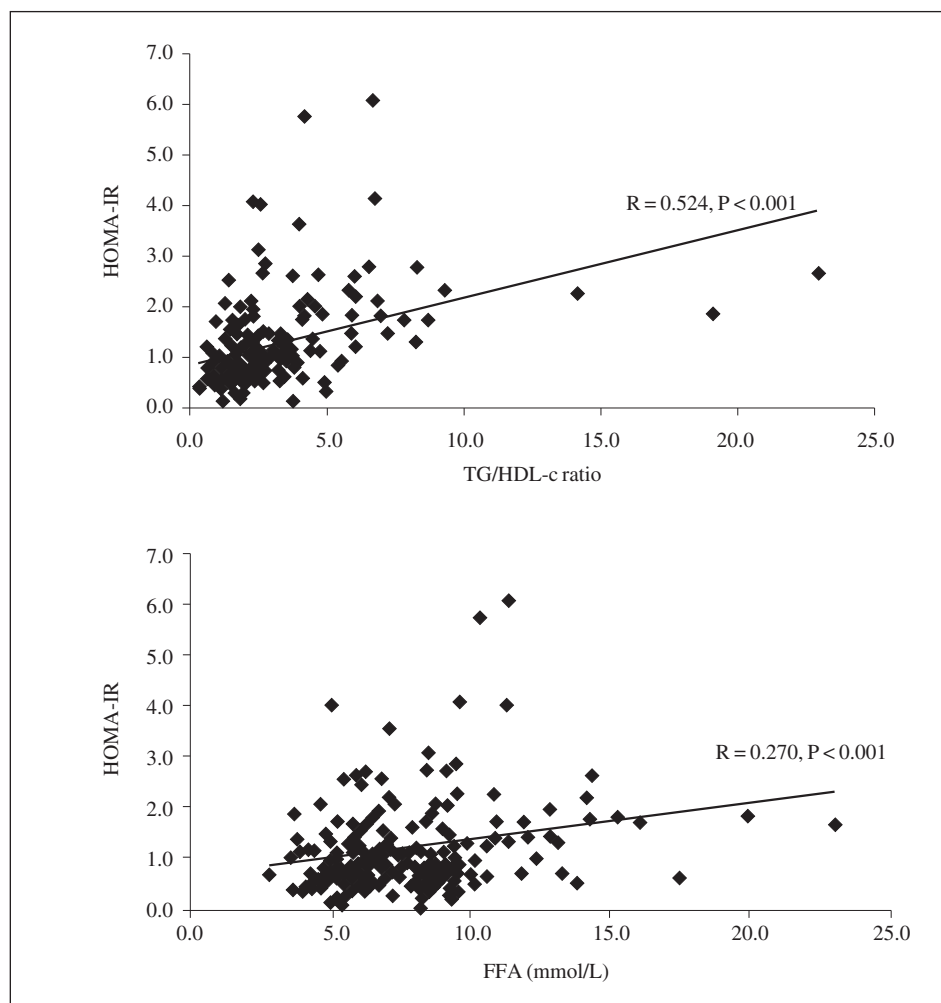

Fig. 1.—Spearman's correlation coefficients (R) for HOMA-IR and lipid profile (TG/HDL-C ratio, ratio between triglycerides and high density lipoprotein; FFA, free fatty acids concentrations).
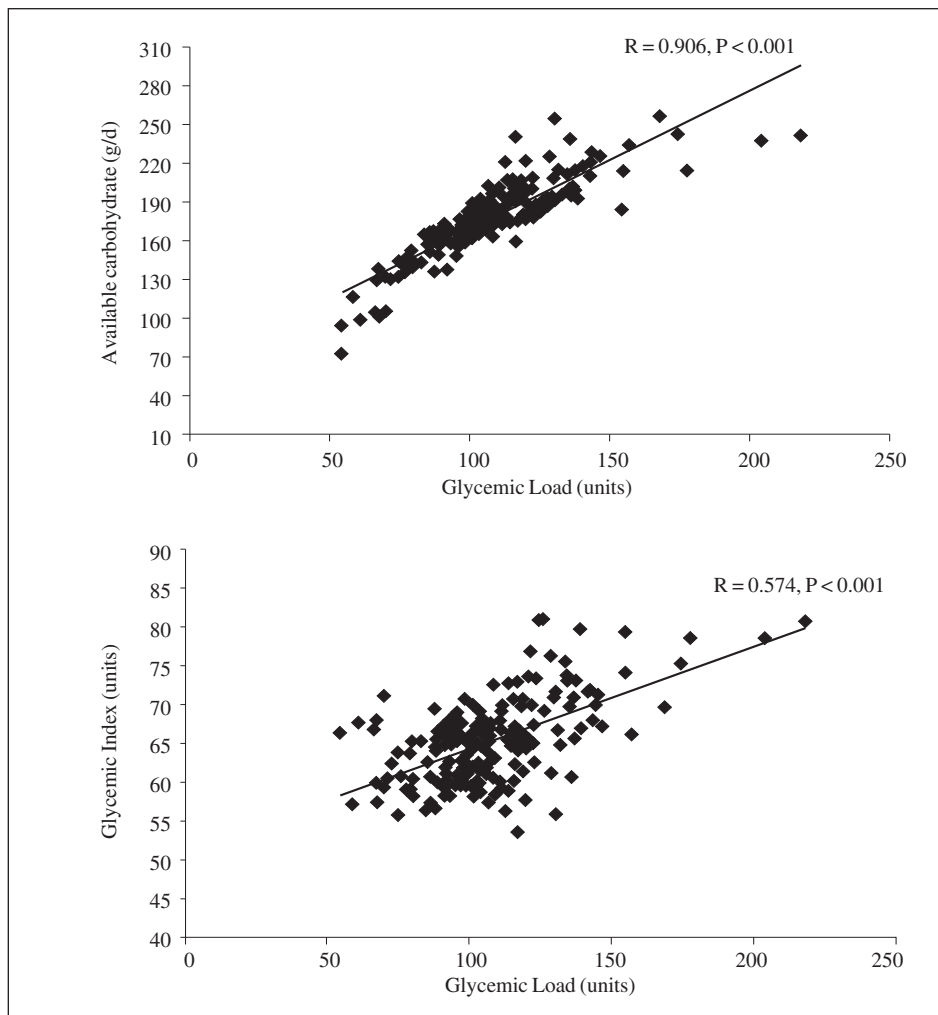
Fig. 2.—Spearman's correlation coefficients (R) for dietary glycemic load and available carbohydrate and dietary glycemic index.

randomized clinical trial demonstrated that despite the reduction in dietary GL, no association of this change with improvements in TG and HDL-C concentrations was found in middle-aged men and women[23]. It is possible that the results reported by Lin et al.[23] reflects the impact of the food intake under-reporting on GL classification and on its association with risk factors.

Hypertriglyceridemia induced by high-carbohydrate diets can also be triggered by insulin resistance due to stimulation of lipolysis and consequently an endogenous overproduction of FFA and VLDL-C[25]. Moreover, the glucose and insulin concentrations increase considerably in the period immediately after consumption of high GI[26]/GL[5] diet while in the period 4 to 6 hours postprandial there is glycaemia reduction and possibility hypoglycemia, which leads to increased release of counter-regulatory hormones, and consequently the increase in glycaemia and FFA levels[27]. In fact our results show a positive association of FFA levels with dietary GL, as well as, a positive correlation of insulin resistance (HOMA-IR) with FFA levels and with TG/HDL-C ratio.

The inverse relationship between HDL-C and CVD risk is well established inasmuch as HDL-C exerts antioxidant, anticoagulant, antiplatelet and antiathero-genic functions[28]. Moreover, high concentrations of FFA[29] and TG/HDL-C ratio[30] may favor the increase of CVD risk. Thus, our results indicate that the increase in dietary GL is a possible risk factor for CVD, even in physically active individuals.

The practice of walking (metabolic equivalent hours per day walk) was associated with lower CVD risk in a cohort study that included the participation of men and women[14]. In addition, daily walking can attenuate the fasting TG concentrations and remnants particles in response to high-carbohydrate intake[31]. However, dietary GL was not assessed by Koutsari et al.[31] and in other study[14] the association of physical activity with dietary intake was not investigated. Thus, it is still not yet clear whether the walking (directly related to number of steps) may actually attenuate the deleterious effects of dietary high GL on cardiometabolic risk factors manifestation.

Postprandial hyperglycemia also has been related to oxidative stress[3,2] possibly due to increased production of free radicals by the non-enzimatic glycation; auto-oxidation of glucose; and intracellular activation of the polyol pathway, which produces an imbalance in the NADH/NAD+ ratio[33]. In this context, another important result obtained in this study was the positive asso-

ciation between dietary GL and urinary 8-OHdG, an oxidative DNA damage marker.

Besides postprandial hyperglycemia, oxidative stress may be a consequence of a reduction in the antioxidant activity[34]. However, since HDL-C has antioxidant function[35] and a negative association of dietary GL with this lipoprotein was demonstrated in the present study, we performed complementary analysis and observed a negative correlation between 8-OHdG and HDL-C (R = -0.153, P-value = 0.04). This outcome indicates that the pro-oxidant effect of dietary GL could also be related to reduced HDL-C concentrations.

Our results showed no association between dietary GL and the lipid peroxidation marker, 8-iso-PGF2 . In concert, a crossover study showed no significant changes in the fasting urinary isoprostane concentrations in young men (29.4 ± 4.4 years) after the consumption of high or low GI[36]. Another cross-sectional study reported that plasma malondialdehyde concentrations, but not isoprostane, increased linearly with the increase in GL in healthy men and women (46.7 ± 13.5 years)[9]. In view of these results, investigations are needed to further test the relationship between dietary GL and changes in the concentrations of 8-iso-PGF2α.

It is noteworthy that in the present study, we observed a strong correlation of dietary GL with available carbohydrate consumption and a moderate correlation between dietary GI and dietary GL. Similar results were obtained in the Women's Health Study conducted in postmenopausal women without diagnosis of CVD or cancer[11] and in another study involving healthy men and women (18-75 years)[9]. These outcomes suggest that the available carbohydrate but not the dietary GI explains better the statistical results obtained for dietary GL.

Some limitations of the current study should be noted. First, since this is a cross-sectional, the results showed here must be cautiously considered as we cannot assure that the observed associations show a cause/effect relationship, although we controlled potential interfering variables. Second, further studies involving larger numbers of physically active individuals and/or with longitudinal design should be conducted before considering the application of these results at the population level.

In summary, the data presented in this cross-sectional study showed that dietary GL was positively associated with blood lipid profile (TG/HDL-C ratio and FFA concentrations) and with oxidative stress marker (8-OHdG) in physically active men. These results indicate the potentially harmful influence of the consumption of higher GL diets on cardiometabolic risk factors, even in physically active subjects.

Acknowledgments

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