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Glycemic index, glycemic load, and metabolic syndrome in Mexican adolescents: a cross-sectional study from the NHNS-2012

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Abstract

Background: The role of dietary glycemic index (GI) and dietary glycemic load (GL) on metabolic syndrome (MetS) in youth populations remains unclear. The aim of the present study was to evaluate the association among dietary GI, dietary GL, and MetS and its components in Mexican adolescents.

Methods: This study was conducted within the framework of the National Health and Nutrition Survey 2012, a cross-sectional, probabilistic, population-based survey with a multistage stratified cluster sampling design. We analyzed a sample of 1346 subjects aged 12–19 years, representing 13,164,077 adolescents. Dietary habits were assessed through a validated semiquantitative food-frequency questionnaire. We assigned GI values using the International Tables of GI values. We defined MetS according to the International Diabetes Federation criteria developed for adolescents. Multiple logistic regression models were used to estimate odds ratios (ORs) and their 95% confidence intervals (CIs) to evaluate the association between categories of dietary GI and GL and the prevalence of MetS and its components.

Results: We observed no associations between dietary GI or GL and MetS prevalence. Female adolescents in the highest category of dietary GI had higher odds of abnormal blood pressure (OR = 3.66; 95% CI, 1.46–9.22; *P* for trend = 0.012). A high dietary GL was also associated with higher odds of abnormal blood pressure in female adolescents (OR = 5.67; 95% CI, 1.84–17.46; *P* for trend = 0.003).

Conclusions: We found higher odds of abnormal blood pressure for female adolescents with a high dietary GI and dietary GL.

Keywords: Glycemic index, Glycemic load, Metabolic syndrome, Adolescent, Mexico

Background

The prevalence of metabolic syndrome (MetS) is high among children and adolescents with obesity [1, 2]. In Mexico, almost 35% of adolescents are either overweight or obese [3] and the prevalence of MetS oscillates between 6.5% [4] and 19.2% [5]. Therefore, special attention should be given to modifiable risk factors, such as lifestyle and

dietary habits: they play an important role in the development and progression of MetS. Among dietary factors, carbohydrates are the main energy source in the diets of most populations and have a special function in energy metabolism and homeostasis [6]. However, evidence indicates that some carbohydrate sources can be beneficial; others are not, depending on their quality and fiber content [7]. The quality of carbohydrates can be measured using the glycemic index (GI); this is defined as the incremental area under the curve of blood glucose response after eating 50 g of available carbohydrates from a certain food and expressed as a percentage of the glycemic

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response elicited by 50 g of glucose or white bread [8]. Moreover, the glycemic load (GL) considers both the quality and quantity of carbohydrate intake [9, 10].

In adults, evidence from different meta-analysis of randomized controlled trials (RCTs) demonstrated that low-GI or GL diets resulted in lower fasting blood glucose and glycated hemoglobin levels [11] and a greater decrease in total cholesterol and low density lipoprotein cholesterol (LDL-c) compared to control diets [12, 13]. Nevertheless, the latter findings have not been observed in overweight/obese subjects who followed low GI/GL diets [14]. Furthermore, results from RCTs have demonstrated a favorable effect of a low-GI diet on triglyceride levels [15] or concentration of high-density lipoprotein cholesterol (HDL-c) [16]. However, such findings are inconsistent and have not been confirmed by a recent meta-analysis [13].

In children and adolescents, a meta-analysis has demonstrated that low-GI diets might reduce serum triglycerides and homeostasis model assessment index in overweight or obese children and adolescents [17].

The association among GI, GL, and MetS has been mostly studied in prospective studies in adult populations [18, 19] and produced varying results. The evidence for such an association in young people is scarce. Two cross-sectional studies conducted in Australia have identified higher odds of developing MetS for each unit increase in breakfast GL [20] and per 20 unit dietary GL increase [21].

To our knowledge, no evidence is available on the relationship between the quality of carbohydrates and MetS in a Mexican youth population. Therefore, the main objective of this study was to evaluate the association among dietary GI, dietary GL, and MetS and its components in a nationally representative sample of Mexican adolescents.

Methods

Study population

This study was conducted within the framework of the National Health and Nutrition Survey 2012 (NHNS-2012), a cross-sectional, probabilistic, population-based survey with a multistage stratified cluster sampling design conducted in Mexico. The design and methods of the NHNS-2012 have been described elsewhere [22]. The main objective of the NHNS-2012 was to quantify the frequency, distribution, and trends in health and nutrition conditions and their determinants in the Mexican population [22]. Data were collected by computer-assisted interviews at participants' homes. Child interviewees under the age of 14 years were assisted in their responses by a relative.

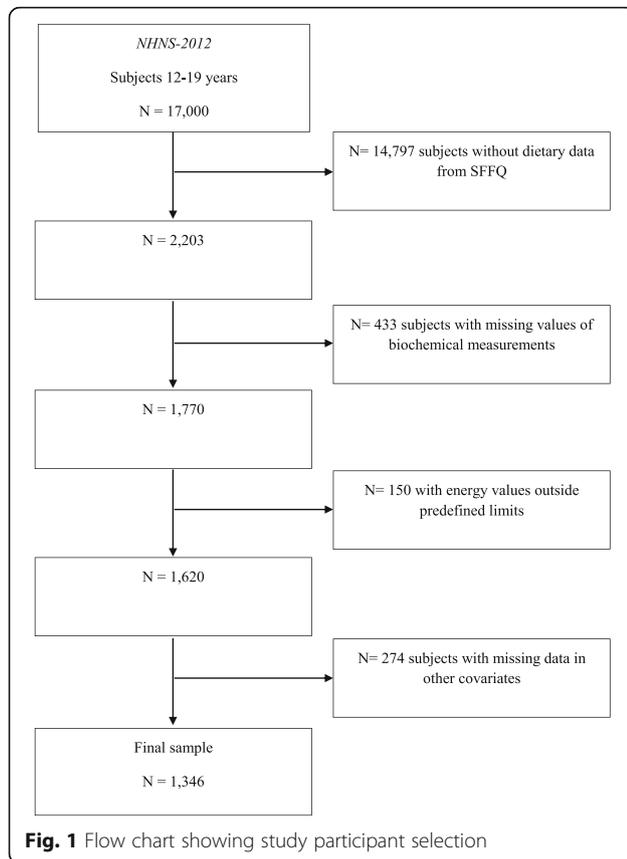
In the NHNS-2012 an original probabilistic sample of 17,000 adolescents was drawn. For the present study, we used the NHNS-2012 subsample of 2203 adolescents aged 12–19 years evaluated by means of a validated

semiquantitative food-frequency questionnaire (SFFQ) to assess dietary habits [23]. We excluded subjects with missing values for biochemical measurements (19.7%) or other covariates used in the statistical analyses (12.4%). Furthermore, we excluded subjects with energy values outside predefined limits (6.8%). The methodology for cleaning dietary data has been broadly described elsewhere [24]. First, the weight in grams of food consumed by each study subject was evaluated according to age-group. We excluded from the analysis subjects who consumed above three standard deviations (SDs) of one or more food items. The biological plausibility of food intake and the percentage contribution of each food to total dietary intake was used to verify data identified as high values. Second, we estimated very high values of energy intake by the ratio of energy intake/estimated energy requirement. The equations of the Institute of Medicine were used as reference [25]. The physical activity level of each subject was considered according to previous studies regarding data of the NHNS-1999 [26]. We excluded very low values of energy intake: under 0.5 of energy intake/basal metabolic rate (BMR). We estimated BMR for adults (≥ 19 years of age) using the Mifflin-St Jeor equations [27]. For subjects under 19 years of age, we used the age- and sex-specific equations of the Food and Agriculture Organization [28]. Accordingly, we included a final sample of 1346 subjects in our analyses, representing a total of 13,164,077 Mexican adolescents (Fig. 1).

Exposure assessment

Dietary assessment

Trained personnel applied a validated SFFQ to evaluate dietary habits during the 7 days before the interview date [23, 24]. For each food item, the questionnaire measured the frequency of intake according to set categories: the range was “never” to “six times a day.” Participants also designated the food portion sizes, using defined categories and number of servings consumed during that week. We first converted the data to number of times a day, and we then estimated the daily portion size. To calculate the consumption of energy (kcal/day) and daily nutrient intakes, we multiplied the daily frequency of consumption (portions/day) of each food by the amount of energy and nutrients in a standard serving or portion size of that food. For that purpose, we used the food composition tables compiled by the National Institute of Public Health of Mexico (INSP: Databases of the nutritional value of food. Compilation of the National Institute of Public Health, unpublished). We totaled the contributions of all foods using Microsoft Visual FoxPro 7.0 (Microsoft Corporation, Seattle, WA, USA). The average Pearson correlation coefficient, between SFFQ and two 24-h dietary recalls, for absolute nutrient intake was 0.374 for



adolescents. The unadjusted, adjusted and deattenuated Pearson correlation coefficients for carbohydrate intake in adolescent population were 0.51, 0.25 and 0.36 respectively [23]. The intake of carbohydrate, protein, fat, and dietary fiber was sex-specific adjusted for total energy intake using the residual method proposed by Willett et al. [29].

Dietary GI and dietary GL assessment

We used the protocol of Louie et al. [30] to assign a GI value to each food item in the SFFQ. We obtained the GI values from available studies conducted in normal subjects, using glucose as reference food [31, 32]. We calculated the dietary GI of each subject by summing the products of the available carbohydrate content per serving for each food multiplied by the average number of daily servings of that food multiplied by its GI; we then divided this by the total amount of daily carbohydrate intake [10, 33]. In a similar manner but without dividing by the total amount of carbohydrate, we estimated dietary GL [10]. Dietary GL was energy-adjusted using sex-specific residuals [29] owing to a high correlation with energy intake ($r = 0.880, P < 0.001$). Finally, we categorized dietary GI and energy-adjusted dietary GL into sex-specific tertiles.

Outcome assessment

Anthropometric assessment

Weight and height were measured using electronic scales and wall stadiometers, respectively. We calculated the BMI as weight (kg) divided by height squared (m^2). We used the BMI z-score (number of SDs by which a child differs from the mean BMI of children of the same age and sex) to classify subjects according to weight status as underweight, normal, overweight, or obese according to the World Health Organization (WHO) growth reference values for adolescents [34]. We measured waist circumference (WC) midway between the lowest rib and the iliac crest using an anthropometric tape parallel to the floor. Blood pressure was measured twice by a trained nurse in the dominant arm by means of a mercury sphygmomanometer [35]. The first reading was conducted after at least 5 min of seated rest. The second reading was taken 5 min after the first. The first Korotkoff sound was used as a measure for systolic blood pressure and the fifth sound for diastolic blood pressure.

Biochemical measurements

Fasting blood samples were collected by trained personnel of the NHNS-2012. The day before blood collection, subjects were instructed to avoid eating any solid or liquid food prior to collection. Blood was drawn from an antecubital vein and collected in tubes without anticoagulant. The blood was centrifuged in situ at 3000 g. For subjects who reported a previous diagnosis of type 2 diabetes mellitus (T2D), a second sample was collected in heparinized tubes. Serum aliquots were stored in cryovials and frozen in liquid nitrogen. Samples were transported to the Mexican National Institute of Public Health and stored at $-70\text{ }^\circ\text{C}$ for posterior analyses in the biochemistry laboratory.

We measured serum glucose concentrations using the glucose oxidase method through chemiluminescence with an automated analyzer (Architect ci8200, Abbott Diagnostics, Wiesbaden, Germany). To verify the accuracy and precision of the procedure, the 965 material of the National Institute of Standards and Technology was measured simultaneously. We determined serum triglyceride levels after lipase hydrolysis in an automatic analyzer (Architect ci8200, Abbott Diagnostics, Wiesbaden, Germany). HDL-c was measured using an enzymatic colorimetric direct method after eliminating chylomicrons, very-low-density lipoproteins (VLDL), and low-density lipoproteins by enzymatic digestion. To assure the precision and accuracy of these measurements, the concentrations of HDL-c and triglycerides were measured simultaneously at a second laboratory (Lipids Laboratory, National Institute of Medical Science and Nutrition Salvador Zubiran of Mexico).

Metabolic syndrome

The presence of MetS was identified according to the International Diabetes Federation (IDF) definition of MetS for children and adolescents [36, 37]. For adolescents aged 12–16 years, MetS was defined according to the following criteria: (1) presence of abdominal obesity (WC \geq 90th percentile for age and sex or adult cutoff if lower); and (2) the presence of two or more other conditions among triglycerides \geq 150 mg/dL, HDL-c $<$ 40 mg/dL, systolic blood pressure \geq 130 or diastolic blood pressure \geq 85 mmHg, fasting plasma glucose \geq 100 mg/dL, and known T2D. Adult IDF criteria were used for subjects aged 16 years or older: central obesity (defined as WC \geq 90 cm for male and \geq 80 cm for female adolescents); and at least two of the following factors: triglycerides \geq 150 mg/dL or specific treatment for high triglycerides; HDL-c $<$ 40 mg/dL in males and $<$ 50 mg/dL in females or specific treatment for these lipid abnormalities; systolic blood pressure \geq 130, diastolic blood pressure \geq 85 mmHg, or treatment of previously diagnosed hypertension; fasting plasma glucose \geq 100 mg/dL; or previously diagnosed T2D.

Covariates

We used specific questionnaires to assess sociodemographic characteristics, medical history, and lifestyle habits. Socioeconomic status (SES) information was based on well-being. Using these data, we calculated an index (well-being index) by principal-components analysis, which included home conditions and presence in the home of household appliances, goods, and services. The continuous variable was categorized into tertiles and used as a proxy for low, medium, and high SES levels.

To collect information on physical activity and sedentary lifestyle in the 12- to 14-year age-group, we used a questionnaire of eight items [38]. The questions included hours of sleep, screen time, means of transportation to school, and formal physical activity (e.g., skating, dancing, and soccer) over the previous year. We also identified the means of transportation and length of time spent on the home-to-school route and vice versa. Furthermore, we categorized formal or competitive physical activities performed in the previous year according to the following criteria: (1) inactive; (2) one or two activities; and (3) three or more activities.

We assessed physical activity in adolescents aged 15–19 years using the short version of the International Physical Activity Questionnaire [39]. In addition, participants were asked about their usual hours of sleep, inactive transport time, and usual screen time [40, 41]. The evaluation comprised 14 questions and allowed us to differentiate the activity during the week and on weekends. Finally, in agreement with WHO criteria, we classified physical activity into three categories: active, moderately active, and inactive [42].

Statistical analyses

The sample design characteristics (sample weights, cluster, and strata variables) were considered for all the analyses. We estimated the baseline characteristics of the population and dietary intake according to sex-specific tertiles of dietary GI and energy-adjusted dietary GL. To explore differences across categories of dietary GI and energy-adjusted dietary GL, we used linear regression models and design-based Wald statistics for quantitative variables; we employed the design-based F statistic (corrected, weighted Pearson chi-square statistic) for categorical data.

We used multiple logistic regression models to estimate odds ratios (ORs) and their 95% confidence intervals (CIs) to evaluate the association between categories of dietary GI and GL and the prevalence of MetS. The first model was adjusted for age (years). The second multivariate model further included the following: SES (low, middle, high); geographic regions of Mexico (north, central, south, metropolitan area) and dietary fiber intake (continuous, energy-adjusted). To examine the associations between categories of dietary GI and GL and the prevalence of MetS components (elevated WC, abnormal blood pressure, elevated fasting serum triglycerides, low HDL-c, elevated fasting serum glucose concentrations), we fitted logistic regression models with the same covariates as those used for the main analyses. We selected covariates using a hypothesis-based analysis. The addition of potential confounders, such as physical activity levels or screen time as covariates in the multivariate models, did not change the magnitude or effect of our results; thus, we did not use those factors in the final models. We took the lowest categories of dietary GI and GL as references in all the models. The tests of the linear trend across increasing categories of dietary GI and GL were conducted by assigning the sex-specific median value within each category. We treated those variables as continuous in the logistic regression models.

To examine a possible interaction between dietary GI and GL and age (under and over 16 years), and weight status (underweight/normal, overweight/obese), we introduced the product terms in the different multivariable models; we considered $P < 0.05$ in the likelihood ratio test as statistically significant. All statistical analyses were performed using Stata 12.0 (StataCorp, College Station, TX, USA), and the significance level was set at $P < 0.05$.

Results

In this study, the mean (SD) dietary GI and GL of adolescents in the NHNS-2012 was 51.8 (5.3) and 150.0 (27.3), respectively. The MetS prevalence in the overall sample was 8.8%, with a higher proportion among female (12.0%) than male adolescents (6.4%; $P = 0.019$).

Tables 1 and 2 present the main characteristics of the sample according to sex-specific tertiles of dietary GI

Table 1 General characteristics of the sample according to sex-specific categories of dietary glycemic index^a

Characteristics	Dietary glycemic index ^b													
	Female adolescents						Male adolescents							
	Low		Moderate		High		P value	Low		Moderate		High		P value
Mean	SD	Mean	SD	Mean	SD	Mean		SD	Mean	SD	Mean	SD		
Dietary GI (units)	46.8	0.3	51.0	0.1	56.6	0.4	<0.001	47.6	0.3	51.9	0.1	57.0	0.3	<0.001
Age (years)	15.5	0.2	16.2	0.3	16.0	0.2	0.123	15.4	0.2	16.0	0.2	15.6	0.2	0.185
Socioeconomic status (%)							0.549							0.117
Low	30.0		25.6		30.9			27.7		32.6		31.2		
Medium	26.8		34.3		35.6			37.3		28.1		35.9		
High	43.3		40.1		33.5			35.0		39.3		32.9		
Geographic region (%)							0.186							0.515
North	24.4		12.2		20.8			19.5		14.4		25.0		
Central	25.1		25.2		35.4			31.0		28.9		31.6		
Metropolitan area	24.5		30.3		15.4			14.3		19.8		12.0		
South	26.1		32.3		28.4			35.2		37.0		31.4		
Weight status (%)							0.379							0.001
Underweight	1.3		1.0		1.9			1.2		0.9		0.7		
Normal	64.2		65.9		50.9			71.8		66.3		69.7		
Overweight	21.3		23.4		34.5			13.1		28.0		13.3		
Obese	13.3		9.7		12.7			13.9		4.9		16.3		
Screen time (computer, TV, and video) (%)							0.131							0.011
≤ 2 h/day	29.6		43.8		28.9			33.7		39.8		29.4		
2–4 h/day	35.0		29.1		45.7			29.3		39.5		37.1		
≥ 4 h/day	34.1		25.3		24.5			36.8		17.8		33.5		
No data available	13		1.7		1.0			0.3		2.9		0.0		
Physical activity (% age 12–14 years)							0.174							0.034
Sedentary	76.3		77.7		57.7			51.6		40.2		55.9		
1–2 activities	18.9		20.0		36.4			44.9		50.5		41.1		
≥ 3 activities	2.1		0.5		2.4			2.7		0.6		2.5		
No data available	2.7		1.8		3.4			0.7		8.6		0.5		
Physical activity (% age 15–19 years)							0.239							0.131
Sedentary	28.2		29.7		26.7			17.7		17.9		14.0		
Moderately active	6.1		22.2		16.7			9.4		19.8		19.4		
Active	65.8		46.0		56.7			72.9		62.3		66.6		
No data available	0.0		2.1		0.0			0.0		0.0		0.0		
Dietary intake														
Total energy intake (kcal/d)	1795	53	1747	73	1828	58	0.707	2033	53	2121	61	2215	69	0.118
Carbohydrate intake (g/d) ^c	246.0	3.3	271.5	4.2	269.7	3.3	<0.001	298.0	3.6	315.8	4.3	311.5	4.1	0.002
Carbohydrate intake (% energy)	54.9	0.7	61.5	1.2	60.8	0.8	<0.001	55.8	0.7	59.5	0.8	59.1	0.7	<0.001
Protein intake (g/d) ^c	58.5	1.0	52.7	1.2	51.9	0.8	<0.001	67.9	1.1	63.8	1.0	60.5	1.2	<0.001
Protein intake (% energy)	13.2	0.2	11.8	0.3	11.5	0.2	<0.001	13.0	0.2	12.1	0.2	11.4	0.2	<0.001

Table 1 General characteristics of the sample according to sex-specific categories of dietary glycemic index^a (Continued)

Fat intake (g/d) ^c	67.2	1.1	58.8	1.3	59.1	1.3	<0.001	77.5	1.3	70.5	1.6	69.5	1.5	<0.001
Fat intake (% energy)	33.9	0.5	28.8	0.9	29.4	0.7	<0.001	33.2	0.5	29.8	0.7	29.6	0.5	<0.001
MUFA (g/d) ^c	22.6	0.5	19.7	0.5	20.5	0.6	<0.001	25.9	0.6	23.6	0.5	24.7	0.6	0.020
PUFA (g/d) ^c	14.3	0.4	14.1	0.4	14.5	0.4	0.750	17.4	0.4	17.5	0.7	16.8	0.4	0.424
SFA (g/d) ^c	25.8	0.5	22.1	0.6	22.7	0.7	<0.001	29.4	0.7	26.2	0.7	26.5	0.7	0.002
Trans fatty acids (g/d) ^c	0.5	0.0	0.5	0.0	0.5	0.0	0.054	0.5	0.0	0.5	0.0	0.6	0.0	0.692
Dietary fiber intake (g/d) ^c	21.6	0.5	22.7	1.3	21.3	0.7	0.683	25.8	0.7	27.0	1.0	22.8	0.8	0.003
Dietary sugar intake (g/d)	94.1	4.9	109.9	7.2	112.3	4.4	0.021	108.4	4.1	116.2	6.4	134.6	4.4	<0.001
WC (cm)	76.8	1.0	76.8	1.2	78.3	1.4	0.640	77.0	1.5	77.5	1.0	78.7	1.2	0.660
Triglycerides (mg/dL)	116.9	7.5	135.3	9.7	113.5	5.6	0.142	113.2	6.6	113.3	6.6	132.1	9.4	0.212
HDL-c (mg/dL)	45.1	0.8	48.7	2.0	43.0	1.4	0.075	43.3	0.9	41.3	0.9	43.0	0.8	0.231
Systolic blood pressure (mmHg)	107.1	0.9	108.9	1.1	110.0	1.2	0.169	111.2	1.5	110.9	1.0	113.3	1.0	0.219
Diastolic blood pressure (mmHg)	70.0	0.8	72.0	1.1	73.2	1.0	0.050	70.3	1.1	71.1	0.9	73.3	0.8	0.051
Fasting serum glucose (mg/dL)	80.4	1.0	79.0	1.2	77.6	1.0	0.172	81.3	0.8	80.2	1.3	81.5	1.4	0.733
MetS prevalence (%) ^d	9.5		9.7		16.9		0.234	6.9		3.8		8.4		0.344

Abbreviations: *GI* Glycemic index, *GL* glycemic load, *kcal/d* kilocalories per day, grams per day (g/d), *MUFA* monounsaturated fatty acids, *PUFA* polyunsaturated fatty acids, *SFA* saturated fatty acids, *WC* waist circumference, *HDL-c* high-density lipoprotein cholesterol, *MetS* metabolic syndrome

^aValues are expressed as means and standard deviations (SD) for continuous variables, and data from categorical variables are shown as percentages

^bCategories based on sex-specific tertiles of dietary GI. ^cValues were adjusted for energy intake using sex-specific residuals. ^dThe age-specific International Diabetes Foundation definition of the metabolic syndrome was used [36, 37]

and energy-adjusted dietary GL. Participants in the highest category of dietary GI had higher carbohydrate and sugar intake and lower values of protein and total fat, than subjects in the lowest category of dietary GI. Similar characteristics were found across categories of dietary GL, in addition, we observed a higher dietary fiber intake in the top tertile of dietary GL compared with those in the lowest tertile. We found no differences in the prevalence of MetS or the mean of its components across dietary GL categories.

Table 3 shows the ORs and 95% CI for MetS and its components according to sex-specific categories of dietary GI. We observed no association of MetS with either dietary GI or dietary GL. However, when MetS components were analyzed separately, a direct association between the highest dietary GI and abnormal blood pressure was evident in female adolescents (Model 1: OR = 3.66; 95% CI, 1.59–8.39; *P* for trend = 0.009). This association remained statistically significant after multivariate adjustment. Table 4 shows the ORs and 95% CI for MetS and its components according to sex-specific categories of energy-adjusted dietary GL. Our results from the multivariate model also indicated that female adolescents with the highest dietary GL had higher odds of abnormal blood pressure (OR = 5.67; 95% CI, 1.84–17.46); there was a significant trend across categories of dietary GL (*P* for trend = 0.003). Among males, no

statistically significant associations were found between dietary GI or dietary GL and abnormal BP. We found no statistically significant associations for the remaining MetS criteria with dietary GI or GL.

None of the interactions assessed was statistically significant in the association between dietary GI and GL and MetS (*P* for interaction >0.05)

Discussion

In this cross-sectional study, we found no associations between dietary GI or GL and MetS. However, in an analysis of MetS components, high dietary GI and GL were associated with higher odds of abnormal blood pressure in female adolescents.

We found no associations between dietary GI or GL and MetS. Similar results were observed in a clinical trial performed in European children and adolescents (5–18 years) did not reveal an association between a low-GI diet and MetS [43]. A cross-sectional study conducted in 516 Australian adolescents found no association between overall dietary GI or dietary GL and MetS [20]. In that study, however, breakfast GL was found to be predictive of MetS in female, but not male, adolescents. In the present study, we used SFFQ to assess dietary intake, and we were unable to estimate dietary GI or GL at

Table 2 General characteristics of the sample according to sex-specific categories of energy-adjusted dietary glycemic load^a

Characteristics	Energy-adjusted dietary glycemic load ^{b,c}													
	Female adolescents						Male adolescents							
	Low		Moderate		High		P value	Low		Moderate		High		P value
Mean	SD	Mean	SD	Mean	SD	Mean		SD	Mean	SD	Mean	SD		
Dietary GL ^c (units)	110.0	1.3	135.0	0.6	162.9	1.4	<0.001	132.1	1.5	159.9	0.7	192.5	1.4	<0.001
Age (years)	15.9	0.3	15.9	0.2	15.9	0.3	0.998	15.8	0.3	15.9	0.2	15.3	0.2	0.112
Socioeconomic status (%)							0.013							<0.001
Low	21.3		30.6		34.7			16.9		29.9		44.8		
Medium	24.9		40.4		31.4			31.3		33.2		36.7		
High	53.8		29.0		33.9			51.8		37.0		18.5		
Geographic region (%)							0.030							0.065
North	26.8		19.3		11.3			23.2		22.7		12.9		
Central	29.6		26.7		29.4			26.2		29.4		35.9		
Metropolitan area	28.1		15.6		26.5			23.1		12.5		10.6		
South	15.6		38.4		32.8			27.6		35.4		40.7		
Weight status (%)							0.678							0.283
Underweight	1.3		1.8		1.2			0.7		0.9		1.1		
Normal	63.7		55.0		62.3			72.1		61.5		74.1		
Overweight	20.4		31.1		27.6			15.0		24.3		15.3		
Obese	14.6		12.1		9.0			12.2		13.3		9.5		
Screen time (computer, TV, and video) (%)							0.868							<0.001
≤ 2 h/day	35.8		32.8		33.7			35.9		27.6		39.6		
2–4 h/day	32.7		37.1		40.1			23.5		47.9		34.6		
≥ 4 h/day	31.2		28.1		24.6			40.3		24.3		23.2		
No data available	0.4		2.0		1.6			0.3		0.3		2.6		
Physical activity (% age 12–14 years)							0.039							0.171
Sedentary	79.3		69.3		61.7			56.4		50.5		44.0		
1–2 activities	16.6		24.3		35.6			42.3		46.6		46.5		
≥ 3 activities	3.2		0.0		2.2			0.6		2.0		3.2		
No data available	1.0		6.4		0.5			0.7		0.9		6.3		
Physical activity (% age 15–19 years)							0.255							0.355
Sedentary	37.7		26.3		21.2			14.1		20.4		15.1		
Moderately active	11.5		12.2		22.9			8.0		23.0		19.3		
Active	50.8		61.5		53.7			77.9		56.6		65.7		
No data available	0.0		0.0		2.3			0.0		0.0		0.0		
Dietary intake														
Total energy intake (kcal/d)	1844	58	1697	59	1828	62	0.190	2169	53	2019	65	2179	60	0.141
Carbohydrate intake (g/d) ^c	228.2	2.1	263.0	1.8	296.5	2.3	<0.001	266.1	2.8	308.6	2.0	350.9	2.9	<0.001
Carbohydrate intake (% energy)	50.8	0.5	59.5	0.4	67.0	0.7	<0.001	50.2	0.5	58.2	0.4	66.1	0.5	<0.001
Protein intake (g/d) ^c	61.2	0.8	53.4	0.7	48.5	1.0	<0.001	71.4	1.2	64.1	0.8	56.8	0.9	<0.001
Protein intake (% energy)	13.8	0.2	12.0	0.2	10.8	0.2	<0.001	13.6	0.2	12.2	0.2	10.7	0.2	<0.001

Table 2 General characteristics of the sample according to sex-specific categories of energy-adjusted dietary glycemic load^a (Continued)

Fat intake (g/d) ^c	73.0	0.9	61.9	0.8	50.1	0.9	<0.001	85.6	1.2	72.8	0.7	59.1	1.1	<0.001
Fat intake (% energy)	36.9	0.4	30.6	0.4	24.6	0.6	<0.001	36.5	0.5	30.9	0.4	25.1	0.4	<0.001
MUFA (g/d) ^c	25.0	0.5	21.0	0.4	16.8	0.4	<0.001	28.8	0.6	25.1	0.3	20.2	0.5	<0.001
PUFA (g/d) ^c	14.9	0.4	14.3	0.4	13.8	0.4	0.130	19.0	0.7	16.6	0.3	16.1	0.4	0.002
SFA (g/d) ^c	28.6	0.6	23.7	0.4	18.3	0.4	<0.001	32.8	0.6	28.0	0.4	21.3	0.6	<0.001
Trans fatty acids (g/d) ^c	0.6	0.0	0.5	0.0	0.4	0.0	<0.001	0.7	0.0	0.5	0.0	0.4	0.0	<0.001
Dietary fiber intake (g/d) ^c	18.7	0.7	21.0	0.5	25.9	1.1	<0.001	20.9	0.5	24.6	0.7	30.2	0.9	<0.001
Dietary sugar intake (g/d)	94.4	6.0	102.5	5.5	119.6	6.2	0.014	111.7	3.9	117.3	5.6	129.9	5.2	0.015
WC (cm)	77.5	1.2	78.1	1.1	76.3	1.4	0.592	78.1	1.5	79.1	1.3	76.0	1.1	0.133
Triglycerides (mg/dL)	117.5	6.6	115.0	8.2	133.3	9.4	0.300	114.0	7.7	121.5	6.9	122.8	8.4	0.711
HDL-c (mg/dL)	47.8	2.0	44.1	1.0	44.9	1.5	0.233	43.0	0.9	41.6	0.8	43.1	0.9	0.302
Systolic blood pressure (mmHg)	108.2	0.9	108.6	1.0	109.3	1.3	0.794	111.5	1.6	112.0	0.9	111.8	0.9	0.955
Diastolic blood pressure (mmHg)	71.3	0.9	71.3	1.0	72.5	1.1	0.653	70.5	1.1	71.5	0.9	72.7	0.8	0.231
Fasting serum glucose (mg/dL)	80.4	1.2	79.9	1.2	76.7	0.9	0.018	81.4	1.0	81.5	1.4	80.1	1.2	0.620
MetS prevalence (%) ^d	9.1		13.0		13.9		0.605	8.7		5.2		5.2		0.461

Abbreviations: GI Glycemic index, GL glycemic load, kcal/d kilocalories per day, g/d grams per day, MUFA monounsaturated fatty acids, PUFA polyunsaturated fatty acids, SFA saturated fatty acids, WC waist circumference, HDL-c high-density lipoprotein cholesterol, MetS metabolic syndrome

^aValues are expressed as means and standard deviations (SD) for continuous variables, and data from categorical variables are shown as percentages

^bCategories based on sex-specific tertiles of dietary GL. ^cValues were adjusted for energy intake using sex-specific residuals. ^dThe age-specific International Diabetes Foundation definition of the metabolic syndrome was used [36, 37]

different mealtimes. Thus, it was not possible for us to confirm the results of that Australian study.

Our results also contrast with those of a cross-sectional study, in which dietary GL was associated with a higher prevalence of MetS in 769 adolescents (13–15 years) [21]. The variance with our results may be explained by the different methods used for dietary assessment. The 3-day food record used in that study may in fact have assessed GI more accurately than the SFFQ used in ours: food records give a more precise indication of the types and portions of food consumed than the SFFQ.

We identified an association between the highest dietary GI and GL and abnormal blood pressure among female adolescents. In contrast to our findings, those of a clinical trial that included 50 overweight or obese female adolescents did not indicate a decrease in blood pressure after a 10-weeks intervention with a low-GI diet [44]. The discrepancy between our results and theirs could be explained by the study design. Our cross-sectional study did not allow an assessment of causality; therefore, more prospective studies and clinical trials are needed to confirm the observed association. On the other hand, similar results were observed in a prospective investigation conducted among 858 Australian adolescents followed up for 5 years [45]. The authors found a direct association among female adolescents: for each 1-SD increment in dietary GI and GL, mean systolic blood pressure rose by

2.3 and 4 mmHg, respectively. In that study, no significant associations were observed between carbohydrate quality and blood pressure among male adolescents.

In the present work, no evidence was found concerning an association among dietary GI, dietary GL, and the remaining METs components (elevated WC, elevated triglycerides, low HDL-c, elevated fasting serum glucose). Results from a recent systematic review did not show an association between low/high GI diets and body mass index, waist circumference, hip circumference, waist-to-hip ratio, total cholesterol, LDL-c, HDL-c, diastolic and systolic blood pressure, fasting serum glucose, fasting serum insulin, glycosylated hemoglobin and C-reactive protein. However, the latter meta-analyses demonstrated that low GI protocols resulted in more pronounced decreases in triglycerides and HOMA-index [17].

Nevertheless, recent intervention studies determined that low-GI diets led to a significantly greater reduction in WC [46, 47] compared with controls. Also, a clinical trial have demonstrated that blood glucose total area under the curve was 13% greater with a high-GI than low-GI breakfast among overweight female adolescents and 4% higher in non-overweight female adolescents [48]. Moreover, a dietary intervention with low GI was observed to improve serum glucose levels in children and adolescents with type 1 diabetes mellitus [49]. Similarly, a low-GL dietary intervention for 6 weeks among

Table 3 Association between metabolic syndrome and sex-specific categories of dietary glycaemic index

	Dietary glycaemic index ^a							
	Female adolescents				Male adolescents			
	Low	Moderate	High	P trend	Low	Moderate	High	P trend
MetS								
Model 1 ^b OR (95% CI)	1	0.92 (0.35–2.40)	1.78 (0.70–4.55)	0.192	1	0.50 (0.15–1.63)	1.21 (0.50–3.19)	0.673
Model 2 ^c OR (95% CI)	1	0.81 (0.30–2.19)	1.60 (0.62–4.15)	0.275	1	0.56 (0.18–1.77)	1.25 (0.48–3.31)	0.641
Elevated WC								
Model 1 ^b OR (95% CI)	1	1.22 (0.58–2.54)	1.24 (0.70–2.22)	0.486	1	0.84 (0.40–1.74)	1.13 (0.58–2.20)	0.696
Model 2 ^c OR (95% CI)	1	1.16 (0.56–2.42)	1.33 (0.72–2.45)	0.361	1	0.87 (0.43–1.76)	1.25 (0.64–2.46)	0.513
Elevated triglycerides								
Model 1 ^b OR (95% CI)	1	1.41 (0.64–3.12)	0.98 (0.51–1.88)	0.840	1	0.80 (0.38–1.66)	1.06 (0.53–2.12)	0.850
Model 2 ^c OR (95% CI)	1	1.25 (0.58–2.68)	0.99 (0.52–1.88)	0.911	1	0.78 (0.35–1.70)	1.15 (0.56–2.35)	0.714
Low HDL-c								
Model 1 ^b OR (95% CI)	1	0.69 (0.37–1.29)	1.65 (0.93–2.94)	0.058	1	1.58 (0.91–2.76)	1.26 (0.76–2.09)	0.396
Model 2 ^c OR (95% CI)	1	0.67 (0.36–1.26)	1.56 (0.82–2.95)	0.126	1	1.71 (1.00–2.92)	1.29 (0.78–2.13)	0.321
Abnormal blood pressure								
Model 1 ^b OR (95% CI)	1	2.22 (0.76–6.43)	3.66 (1.59–8.39)	0.009	1	0.48 (0.18–1.28)	1.67 (0.82–3.40)	0.139
Model 2 ^c OR (95% CI)	1	2.02 (0.60–6.75)	3.66 (1.46–9.22)	0.012	1	0.53 (0.20–1.41)	1.66 (0.83–3.32)	0.143
Elevated fasting serum glucose								
Model 1 ^b OR (95% CI)	1	0.71 (0.13–3.76)	0.24 (0.05–1.32)	0.114	1	1.25 (0.23–6.63)	2.30 (0.47–11.22)	0.278
Model 2 ^c OR (95% CI)	1	1.07 (0.23–5.11)	0.24 (0.05–1.22)	0.068	1	1.21 (0.24–6.17)	2.72 (0.43–17.08)	0.289

Abbreviations: OR Odds ratio, CI confidence interval, MetS metabolic syndrome, WC waist circumference, HDL-c high-density lipoprotein cholesterol. ^aCategories based on sex-specific tertiles of dietary GI. ^bModel adjusted for age (years). ^cMultivariate model adjusted for age (years), socioeconomic level (low, middle, or high), geographic region (north, central, south, or metropolitan area) and dietary fiber intake (continuous, energy-adjusted)

overweight and obese 11-year-old children showed a reduction in fasting glucose [50]. However, clinical trials have been conducted in specific population groups: this fact—along with dietary intervention—could explain the differences from our results.

In our study, mean dietary GI was 51.5 among female adolescents and 52.1 in male adolescents, and dietary GL was higher among male adolescents (161.4) than female adolescents (135.8). The GI values of our sample were lower than those found in Australian, Canadian, British or Japanese adolescents (around 56 to 64 units) and mean dietary GL of our study was in agreement to previous studies conducted in adolescents (range from 128 to 168 units) [20, 51–54]. Thus it is still necessary and urgent to elucidate the role that low GI or GL diets exert on MetS onset in youth population worldwide, since individuals with MetS have a 2-fold risk of developing cardiovascular disease [55] and higher risk of T2D compared with people without this syndrome [56].

One hypothesized metabolic effect by which high-GI and GL diets increase blood pressure is a postprandial glycaemic response and the consequent hyperinsulinemia elicited after consuming high-GI foods [57]. It has been found that higher dietary GI during puberty is

prospectively associated with greater insulin resistance [58]. Hyperinsulinemia has been associated with abnormal levels of blood pressure through stimulation of the sympathetic nervous system [59], increased sodium retention, and volume expansion [45].

We acknowledge that our study has several limitations. Owing to the cross-sectional design, we cannot make causal inferences. Our findings are specific for Mexican adolescents and cannot therefore be generalized to other population groups. Other limitation is that we were unable to assess the impact of pubertal or hormonal status in our analyses. Puberty could be a confounding variable since transition from Tanner stage I to Tanner stage III has been associated with temporary reduction of insulin sensitivity, increases in fasting glucose and insulin levels and different hormonal changes [60]. In addition, physical activity was not included as a covariate in our analyses due to the lack of significance in our models. However, a recent meta-analysis has found an association between physical activity and MetS in adolescents [61]. We therefore, cannot discard that measurement error might exist since questionnaires used in this study are not validated for estimating physical activity in Mexican adolescents. Also, underreporting could be a source of bias in our study, since evidence in adolescents demonstrated that misreporters showed higher rates

Table 4 Association between metabolic syndrome and sex-specific categories of energy-adjusted dietary glyce mic load

	Energy-adjusted dietary glyce mic load ^{ab}							
	Female adolescents				Male adolescents			
	Low	Moderate	High	P trend	Low	Moderate	High	P trend
MetS								
Model 1 ^c OR (95% CI)	1	1.52 (0.56–4.12)	1.64 (0.60–4.49)	0.338	1	0.57 (0.19–1.68)	0.59 (0.20–1.75)	0.364
Model 2 ^d OR (95% CI)	1	1.48 (0.54–4.04)	1.88 (0.64–5.55)	0.255	1	0.50 (0.15–1.64)	0.55 (0.18–1.67)	0.310
Elevated WC								
Model 1 ^c OR (95% CI)	1	1.19 (0.63–2.25)	1.12 (0.55–2.29)	0.760	1	0.85 (0.40–1.82)	0.93 (0.46–1.89)	0.863
Model 2 ^d OR (95% CI)	1	1.23 (0.65–2.36)	1.07 (0.52–2.20)	0.848	1	0.85 (0.39–1.84)	0.95 (0.45–2.00)	0.906
Elevated triglycerides								
Model 1 ^c OR (95% CI)	1	0.93 (0.45–1.96)	1.61 (0.72–3.59)	0.240	1	1.11 (0.54–2.27)	1.03 (0.50–2.11)	0.954
Model 2 ^d OR (95% CI)	1	0.80 (0.38–1.71)	1.03 (0.46–2.29)	0.909	1	0.92 (0.43–1.94)	0.67 (0.30–1.48)	0.300
Low HDL-c								
Model 1 ^c OR (95% CI)	1	1.80 (0.96–3.40)	1.93 (0.96–3.88)	0.074	1	1.31 (0.76–2.25)	0.94 (0.54–1.64)	0.773
Model 2 ^d OR (95% CI)	1	1.44 (0.78–2.67)	1.73 (0.82–3.64)	0.151	1	1.13 (0.65–1.97)	0.76 (0.41–1.41)	0.351
Abnormal blood pressure								
Model 1 ^c OR (95% CI)	1	1.07 (0.36–3.23)	2.69 (0.93–7.78)	0.073	1	0.66 (0.30–1.42)	1.00 (0.47–2.12)	0.948
Model 2 ^d OR (95% CI)	1	1.42 (0.42–4.79)	5.67 (1.84–17.46)	0.003	1	0.69 (0.32–1.48)	1.30 (0.56–3.03)	0.538
Elevated fasting serum glucose								
Model 1 ^c OR (95% CI)	1	0.52 (0.13–2.16)	0.42 (0.05–3.68)	0.416	1	1.94 (0.42–8.99)	2.20 (0.46–10.42)	0.313
Model 2 ^d OR (95% CI)	1	0.52 (0.13–2.11)	0.62 (0.10–3.83)	0.568	1	2.35 (0.45–12.24)	3.43 (0.38–30.80)	0.260

Abbreviations: OR Odds ratio, CI confidence interval, MetS metabolic syndrome, WC waist circumference, HDL-c high-density lipoprotein cholesterol. ^aCategories based on sex-specific tertiles of dietary GL. ^bValues were adjusted for energy intake using sex-specific residuals. ^cModel adjusted for age (years). ^dMultivariate model adjusted for age (years), socioeconomic level (low, middle, or high), geographic region (north, central, south, or metropolitan area) and dietary fiber intake (continuous, energy-adjusted).

of insufficient intake of carbohydrate [62]. Although in our study subjects with energy values outside predefined limits were excluded, under-reporting bias might still exist and alter the estimation of nutrient intake and the associations between dietary GI or GL and MetS.

Moreover, the SFFQ evaluated consumption of foods during 7 days prior to the date of the interview, thus habitual dietary habits of the population might not be reflected by this assessment. In addition, the SFFQ was not specifically designed to evaluate dietary GI and GL; using this tool could generate bias about dietary GI and GL variation owing to the limited number of food items and restrictions in quantifying individual amounts of food consumed [63]. Nevertheless, the SFFQ used in the NHNS-2012 has been found sufficiently valid for assessing carbohydrate intake in adolescents [23]. Furthermore, published GI values for local foods in Mexico are limited; for that reason, we used reference GI data from other countries. This could be a source of error because GI values of foods may differ according to variety, growing conditions, processing, and cooking [64]. Some degree of misclassification may have occurred in our dietary assessment; however, such misclassification would probably have been more non-differential such that the bias would likely have been toward null.

One of the strengths of this study is the large sample size, allowing us to introduce possible confounders in the models. The use of an established protocol also allowed us to assign the GI values to the SFFQ in a systematic, reproducible manner. Furthermore, to our knowledge, this is the first study conducted among Mexican adolescents to explore the association among dietary GI, dietary GL, and MetS or its components. Nevertheless, further evidence based on prospective studies is necessary to determine the long-term association among dietary GI, dietary GL, and MetS in youth populations.

Conclusions

We observed no association between dietary GI or dietary GL and MetS in a nationally representative sample of Mexican adolescents. However, we found higher odds of abnormal blood pressure among female adolescents with the highest dietary GI and GL. This investigation contributes to the body of evidence about the relationship between the quality of carbohydrates and MetS risk factors in youth populations. However, owing to the cross-sectional study design, our results have to be treated with caution, and further investigations are required to confirm the identified associations.

Abbreviations

BMI: Body mass index; CI: Confidence interval; GI: Glycemic index; GL: Glycemic load; HDL-c: High-density lipoprotein cholesterol; IDF: International Diabetes Federation; MetS: Metabolic syndrome; MUFA: Monounsaturated fatty acids; NHNS-2012: National Health and Nutrition Survey 2012; OR: Odds ratio; PUFA: Polyunsaturated fatty acids; RCTs: Randomized controlled trials; SD: Standard deviation; SES: Socioeconomic status; SFA: Saturated fatty acids; SFFQ: Semi-quantitative food-frequency questionnaire; T2D: Type 2 diabetes mellitus; WC: Waist circumference; WHO: World Health Organization

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Availability of data and materials

Sample was obtained from the Mexican NHNS-2012 dataset, which is freely accessible from the National Public Health Institute of Mexico web site: <http://ensanut.insp.mx/basesdoctos.php#.WOUeHoWcHIV>. In order to analyze data from the NHNS-2012 survey, permission was obtained from the Ethics Review Board of the National Public Health Institute of Mexico. The datasets of the current study are available from the corresponding author on reasonable request.

Authors' contributions

IC-Q and SA-E contributed to the study design, data analyses, and interpretation of findings and wrote the manuscript with important input and feedback from all coauthors; AS-V, MDR-L, RA, and LS-M contributed to the study design and to the critical revision of the manuscript; TS-L contributed to the study design, interpretation of findings, and critical revision of the manuscript. All the authors read and approved the final version of the manuscript.

Competing interests

The authors declare that they have no competing interests.

Consent for publication

Not applicable.

Ethics approval and consent to participate

This study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human subjects were approved by the Ethics Review Board of the National Public Health Institute of Mexico. Written informed consent was obtained from all subjects or their legal guardians prior to the study.

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References

1. Evia-Viscarra ML, Rodea-Montero ER, Apolinar-Jiménez E, Quintana-Vargas S. Metabolic syndrome and its components among obese (BMI >=95th) Mexican adolescents. *Endocr Connect*. 2013;2:208–15.
2. Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW, et al. Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med*. 2004;350(23):2362–74.
3. Gutierrez JP, Rivera-Domarco J, Shamah-Levy T, Villalpando-Hernández S, Franco A, Cuevas-Nasu L, et al. National Health and Nutrition Survey 2012. National Results. (Encuesta Nacional de Salud y Nutrición 2012. Resultados Nacionales). 1st ed. Cuernavaca: Instituto Nacional de Salud Pública; 2012.
4. Rodríguez-Moran M, Salazar-Vazquez B, Violante R, Guerrero-Romero F. Metabolic syndrome among children and adolescents aged 10–18 years. *Diabetes Care*. 27. United States ; 2004. p. 2516-2517.
5. Halley Castillo E, Borges G, Talavera JO, Orozco R, Vargas-Alemán C, Huitrón-Bravo G, et al. Body mass index and the prevalence of metabolic syndrome among children and adolescents in two Mexican populations. *J Adolesc Health*. 2007;40:521–6.
6. Mann J, Cummings JH, Englyst HN, Key T, Liu S, Riccardi G, et al. FAO/WHO scientific update on carbohydrates in human nutrition: conclusions. *Eur J Clin Nutr*. 2007;61(Suppl 1):S132–7.
7. Augustin LS, Kendall CW, Jenkins DJ, Willett WC, Astrup A, Barclay AW, et al. Glycemic index, glycemic load and glycemic response: An International Scientific Consensus Summit from the International Carbohydrate Quality Consortium (ICQC). *Nutr Metab Cardiovasc Dis*. 2015; doi: 10.1016/j.numecd.2015.05.005.
8. Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H, Baldwin JM, et al. Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr*. 1981;34:362–6.
9. Jenkins DJ, Kendall CW, Augustin LS, Franceschi S, Hamidi M, Marchie A, et al. Glycemic index: overview of implications in health and disease. *Am J Clin Nutr*. 2002;76:266S–73S.
10. Salmerón J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, Willett WC. Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *JAMA*. 1997;277:472–7.
11. Livesey G, Taylor R, Hulshof T, Howlett J. Glycemic response and health—a systematic review and meta-analysis: relations between dietary glycemic properties and health outcomes. *Am J Clin Nutr*. 2008;87:258S–68S.
12. Thomas DE, Elliott EJ, Baur L. Low glycaemic index or low glycaemic load diets for overweight and obesity. *Cochrane Database Syst Rev*. 2007;3: CD005105.
13. Goff LM, Cowland DE, Hooper L, Frost GS. Low glycaemic index diets and blood lipids: a systematic review and meta-analysis of randomised controlled trials. *Nutr Metab Cardiovasc Dis*. 2013;23:1–10.
14. Schwingshackl L, Hoffmann G. Long-term effects of low glycemic index/load vs. high glycemic index/load diets on parameters of obesity and obesity-associated risks: a systematic review and meta-analysis. *Nutr Metab Cardiovasc Dis*. 2013;23:699–706.
15. Sacks FM, Carey VJ, Anderson CA, Miller ER, Copeland T, Charleston J, et al. Effects of high vs low glycemic index of dietary carbohydrate on cardiovascular disease risk factors and insulin sensitivity: the OmniCarb randomized clinical trial. *JAMA*. 2014;312:2531–41.
16. Jenkins DJ, Kendall CW, McKeown-Eyssen G, Josse RG, Silverberg J, Booth GL, et al. Effect of a low-glycemic index or a high-cereal fiber diet on type 2 diabetes: a randomized trial. *JAMA*. 2008;300:2742–53.
17. Schwingshackl L, Hobl LP, Hoffmann G. Effects of low glycaemic index/high glycaemic load vs. high glycaemic index/high glycaemic load diets on overweight/obesity and associated risk factors in children and adolescents: a systematic review and meta-analysis. *Nutr J*. 2015;14:87.
18. Finley CE, Barlow CE, Halton TL, Haskell WL. Glycemic index, glycemic load, and prevalence of the metabolic syndrome in the cooper center longitudinal study. *J Am Diet Assoc*. 2010;110:1820–9.
19. Juanola-Falgarona M, Salas-Salvadó J, Buil-Cosiales P, Corella D, Estruch R, Ros E, et al. Dietary Glycemic Index and Glycemic Load Are Positively Associated with Risk of Developing Metabolic Syndrome in Middle-Aged and Elderly Adults. *J Am Geriatr Soc*. 2015;63:1991–2000.
20. Nicholl A, du Heaume M, Mori TA, Beilin LJ, Oddy WH, Bremner AP, et al. Higher breakfast glycaemic load is associated with increased

- metabolic syndrome risk, including lower HDL-cholesterol concentrations and increased TAG concentrations, in adolescent girls. *Br J Nutr.* 2014;112:1974–83.
21. O'Sullivan TA, Lyons-Wall P, Bremner AP, Ambrosini GL, Huang RC, Beilin LJ, et al. Dietary glycaemic carbohydrate in relation to the metabolic syndrome in adolescents: comparison of different metabolic syndrome definitions. *Diabet Med.* 2010;27:770–8.
 22. Romero-Martinez M, Shamah-Levy T, Franco-Nunez A, Villalpando S, Cuevas-Nasu L, Gutierrez JP, et al. National Health and Nutrition Survey 2012: design and coverage. *Salud Publica Mex.* 2013;55(Suppl 2):S332–40.
 23. Denova-Gutiérrez E, Ramírez-Silva I, Rodríguez-Ramírez S, Jiménez-Aguilar A, Shamah-Levy T, Rivera-Dommarco JA. Validity of a food frequency questionnaire to assess food intake in Mexican adolescent and adult population. *Salud Publica Mex.* 2016;58:617–28.
 24. Ramírez-Silva I, Jiménez-Aguilar A, Valenzuela-Bravo D, Martínez-Tapia B, Rodríguez-Ramírez S, Gaona-Pineda E, et al. Methodology for estimating dietary data from the semi-quantitative food frequency questionnaire of the Mexican National Health and Nutrition Survey 2012. *Salud Publica Mex.* 2016;58:629–38.
 25. Institute of Medicine. Energy, Dietary reference intakes for energy, carbohydrates, fiber, fat, protein and amino acids (macronutrients). Washington, DC: Institute of Medicine, National Academies Press; 2005. p. 107–264.
 26. Hernández B, de Haene J, Barquera S, Montterrubio E, Rivera J, Shamah T, et al. Factores asociados con la actividad física en mujeres mexicanas en edad reproductiva. *Pan Am J Public Health.* 2003;14:235–44.
 27. Frankenfield D, Roth-Yousey L, Compher C. Comparison of predictive equations for resting metabolic rate in healthy nonobese and obese adults: a systematic review. *J Am Diet Assoc.* 2005;105:775–89.
 28. Food and Agriculture Organization of the United Nations, World Health Organization, United Nations University. Human energy requirements: Report of a Joint FAO/WHO/ONU Expert Consultation. Rome: FAO; 2004.
 29. Willett WC, Howe GR, Kushi LH. Adjustment for total energy intake in epidemiologic studies. *Am J Clin Nutr.* 1997;65(Suppl 4):1220S–8S. discussion 9S–31S
 30. Louie JC, Flood V, Turner N, Everingham C, Gwynn J. Methodology for adding glycemic index values to 24-hour recalls. *Nutrition.* 2011;27:59–64.
 31. Atkinson FS, Foster-Powell K, Brand-Miller JC. International tables of glycemic index and glycemic load values: 2008. *Diabetes Care.* 2008;31:2281–3.
 32. The University of Sydney. Sydney University Glycemic Index Research Service 2014. 2016. <http://www.glycemicindex.com/>. Accessed 17 Nov 2015.
 33. Wolever TM, Nguyen PM, Chiasson JL, Hunt JA, Josse RG, Palmason C, et al. Determinants of diet glycemic index calculated retrospectively from diet records of 342 individuals with non-insulin-dependent diabetes mellitus. *Am J Clin Nutr.* 1994;59:1265–9.
 34. de Onis M, Onyango AW, Borghi E, Siyam A, Nishida C, Siekmann J. Development of a WHO growth reference for school-aged children and adolescents. *Bull World Health Organ.* 2007;85:660–7.
 35. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA.* 2003;289:2560–72.
 36. Alberti G, Zimmet P, Kaufman F, Tajima N, Silink M, Arslanian S, et al. The IDF consensus definition of the metabolic syndrome in children and adolescents. International Diabetes Federation: Brussels; 2007.
 37. Zimmet P, Alberti KG, Kaufman F, Tajima N, Silink M, Arslanian S, et al. The metabolic syndrome in children and adolescents - an IDF consensus report. *Pediatr Diabetes.* 2007;8:299–306.
 38. Pereira MA, FitzerGerald SJ, Gregg EW, Joswiak ML, Ryan WJ, Suminski RR, et al. A collection of Physical Activity Questionnaires for health-related research. *Med Sci Sports Exerc.* 1997;29(Suppl 6):S1–205.
 39. Craig CL, Marshall AL, Sjöström M, Bauman AE, Booth ML, Ainsworth BE, et al. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc.* 2003;35:1381–95.
 40. Hernandez B, Gortmaker SL, Colditz GA, Peterson KE, Laird NM, Parra-Cabrera S. Association of obesity with physical activity, television programs and other forms of video viewing among children in Mexico city. *Int J Obes Relat Metab Disord.* 1999;23:845–54.
 41. Pals MA, Peeters PH, Bueno-De-Mesquita HB, Ocké MC, Wentink CA, Kemper HC, et al. Validity and repeatability of a modified Baecke questionnaire on physical activity. *Int J Epidemiol.* 1995;24:381–8.
 42. WHO. Global Recommendations on Physical Activity for Health. Geneva, Switzerland: World Health Organization; 2010.
 43. Damsgaard CT, Papadaki A, Jensen SM, Ritz C, Dalskov SM, Hlavaty P, et al. Higher protein diets consumed ad libitum improve cardiovascular risk markers in children of overweight parents from eight European countries. *J Nutr.* 2013;143:810–7.
 44. Rouhani MH, Kelishadi R, Hashemipour M, Esmailzadeh A, Azadbakht L. The effect of low glycemic index diet on body weight status and blood pressure in overweight adolescent girls: a randomized clinical trial. *Nutr Res Pract.* 2013;7:385–92.
 45. Gopinath B, Flood VM, Rochtchina E, Baur LA, Smith W, Mitchell P. Influence of high glycemic index and glycemic load diets on blood pressure during adolescence. *Hypertension.* 2012;59:1272–7.
 46. Kong AP, Choi KC, Chan RS, Lok K, Ozaki R, Li AM, et al. A randomized controlled trial to investigate the impact of a low glycemic index (GI) diet on body mass index in obese adolescents. *BMC Public Health.* 2014;14:180.
 47. Mirza NM, Palmer MG, Sinclair KB, McCarter R, He J, Ebbeling CB, et al. Effects of a low glycemic load or a low-fat dietary intervention on body weight in obese Hispanic American children and adolescents: a randomized controlled trial. *Am J Clin Nutr.* 2013;97:276–85.
 48. Zakrzewski JK, Stevenson EJ, Tolfrey K. Effect of breakfast glycemic index on metabolic responses during rest and exercise in overweight and non-overweight adolescent girls. *Eur J Clin Nutr.* 2012;66:436–42.
 49. Rovner AJ, Nansel TR, Gellar L. The effect of a low-glycemic diet vs a standard diet on blood glucose levels and macronutrient intake in children with type 1 diabetes. *J Am Diet Assoc.* 2009;109:303–7.
 50. Fajcsak Z, Gabor A, Kovacs V, Martos E. The effects of 6-week low glycemic load diet based on low glycemic index foods in overweight/obese children—pilot study. *J Am Coll Nutr.* 2008;27:12–21.
 51. Jones M, Barclay AW, Brand-Miller JC, Louie JC. Dietary glycaemic index and glycaemic load among Australian children and adolescents: results from the 2011–2012 Australian Health Survey. *Br J Nutr.* 2016;116:178–87.
 52. Forbes LE, Storey KE, Fraser SN, Spence JC, Plotnikoff RC, Raine KD, et al. Dietary patterns associated with glycemic index and glycaemic load among Alberta adolescents. *Appl Physiol Nutr Metab.* 2009;34:648–58.
 53. Murakami K, McCaffrey TA, Livingstone MB. Dietary glycaemic index and glycaemic load in relation to food and nutrient intake and indices of body fatness in British children and adolescents. *Br J Nutr.* 2013;110:1512–23.
 54. Murakami K, Miyake Y, Sasaki S, Tanaka K, Arakawa M. Dietary glycaemic index and glycaemic load in relation to risk of overweight in Japanese children and adolescents: the Ryukyus Child Health Study. *Int J Obes.* 2011;35:925–36.
 55. Mottillo S, Filion KB, Genest J, Joseph L, Pilote L, Poirier P, et al. The metabolic syndrome and cardiovascular risk a systematic review and meta-analysis. *J Am Coll Cardiol.* 2010;28(56):1113–32.
 56. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JJ, Donato KA, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation.* 2009;120:1640–5.
 57. Wolever T. Physiological mechanisms and observed health impacts related to the glycaemic index: some observations. *Int J Obes.* 2006;30:S72–S8.
 58. Goletzke J, Herder C, Joslowski G, Bolzenius K, Remer T, Wudy SA, et al. Habitually higher dietary glycemic index during puberty is prospectively related to increased risk markers of type 2 diabetes in younger adulthood. *Diabetes Care.* 2013;36:1870–6.
 59. Landsberg L. Insulin-mediated sympathetic stimulation: role in the pathogenesis of obesity-related hypertension (or, how insulin affects blood pressure, and why). *J Hypertens.* 2001;19:523–8.
 60. Goran MI, Gower BA. Longitudinal study on pubertal insulin resistance. *Diabetes.* 2001;50:2444–50.
 61. Oliveira RG, Guedes DP. Physical Activity, Sedentary Behavior, Cardiorespiratory Fitness and Metabolic Syndrome in Adolescents: Systematic Review and Meta-Analysis of Observational Evidence. *PLoS One.* 2016;11:e0168503.
 62. Serra-Majem L, Ribas L, Pérez-Rodrigo C, García-Closas R, Peña-Quintana L, Aranceta J. Determinants of nutrient intake among children and adolescents: results from the enKid Study. *Ann Nutr Metab.* 2002;46(Suppl 1):31–8.
 63. Hare-Bruun H, Nielsen BM, Grau K, Oxlund AL, Heitmann BL. Should glycemic index and glycemic load be considered in dietary recommendations? *Nutr Rev.* 2008;66:569–90.
 64. Wolever TM. Is glycaemic index (GI) a valid measure of carbohydrate quality? *Eur J Clin Nutr.* 2013;67:522–31.