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Cross-sectional relationship between dietary carbohydrate, glycaemic index, glycaemic load and risk of the metabolic syndrome in a Korean population

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Little is known about the effect of dietary carbohydrate, glycaemic index (GI) and glycaemic load (GL) on the risk of the metabolic syndrome, especially in populations with white rice as the staple food. The study examined the cross-sectional relationship between carbohydrate, GI, GL and risk of the metabolic syndrome. There were a total of 910 middle-aged Korean adults. Dietary carbohydrate, GI and GL were determined by an interview-administered FFQ. The metabolic syndrome was defined using the modified criteria published in the Third Report of the National Cholesterol Education Program Adult Treatment Panel III. The risk of developing the metabolic syndrome was positively related to dietary carbohydrate (P for trend=0·03), GI (P for trend=0·03) and GL intakes (P for trend=0·02) in women after adjusting for potential confounding variables. Among the components of developing the metabolic syndrome, the risk of high TAG and low HDL-cholesterol were positively related to high GI and GL intakes in women. The risk of developing the metabolic syndrome was considerably higher in the highest quintiles of carbohydrate (OR 6·44; 95 % CI 2·16, 19·2), GI (OR 10·4; 95 % CI 3·24, 33·3) and GL intakes (OR 6·68; 95 % CI 2·30, 19·4) than in the lowest quintiles among women with a BMI $\geq 25\,kg/m^2$. However, there was no difference in risk across quintiles of carbohydrate, GI and GL among women with a BMI $< 25\,kg/m^2$. In conclusion, both the quantity and quality of carbohydrate intake has a positive relationship with the risk of the metabolic syndrome in women but this relationship was dependent on the BMI level.

Dietary carbohydrate: Glycaemic index: Glycaemic load: Metabolic syndrome

Diets high in carbohydrates have been known to influence adverse changes in blood lipid and lipoprotein concentrations[1–5] and also cause the aggravation of glucose intolerance[6,7]. Given that the quality as well as the amounts of carbohydrate-containing foods have been addressed, the concept of glycaemic index (GI) and glycaemic load (GL) has been proposed. As an indicator of carbohydrate quality, the GI measures how much each carbohydrate-containing food raises blood glucose levels by comparing it with the same amount of either glucose or white bread[8]. The GL (GI × amount of carbohydrate available in each food item) is an indicator that reflects both the quantity and quality of the carbohydrates[9].

Previous studies related to carbohydrates, GI and GL have mostly focused on the effects that they may have on CVD- and diabetes-related risk factors[4,10–14]. The relationship between carbohydrate, GI and GL intake and the metabolic syndrome is important to consider because this syndrome is an important precursor of CVD and diabetes[15] as a clustering phenomenon of metabolic phenotypes such as dyslipidaemia, high resting blood pressure, high fasting glucose and abdominal obesity. However, all but one previous study[4] did not examine the metabolic syndrome itself. Moreover, most findings related to the quantity and quality of carbohydrates (carbohydrate, GI and GL) are based only on Western populations[4,10–12].

In the Korean population, the average proportion of total energy intake from carbohydrates is 65·6 % and that of total carbohydrate consumption from white rice is 55·6 %[16]. This is more than the Japanese, where only 43 % of the total carbohydrates are from white rice[17]. Thus, the high carbohydrate levels consumed by Koreans may lead to the high prevalence of the metabolic syndrome, which is 22·1 % in men and 27·8 % in women according to the waist circumference criteria for Asians (90 cm for men; 80 cm for women)[18]. However, the mortality of CVD for Koreans is relatively low compared with Western populations[19] and the BMI (23·2 kg/m^2 in men and 23·4 kg/m^2 in women) is also lower than that of the US population (26·6 kg/m^2 in men and 26·3 kg/m^2 in women)[18]. Therefore, the present study examined whether carbohydrate had an effect on the metabolic syndrome and its components and whether the effects were dependent on the weight status of a population whose staple food is white rice.

Methods

Subjects

Subjects who were aged at least 20 years old were recruited for a study on CVD risk factors from February 2004 to August 2005 in Yangpyeong, a city located in the GyeongGi-Do province

Abbreviations: GI, glycaemic index; GL, glycaemic load; HDL-C, HDL-cholesterol.

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of Korea. To recruit subjects, we selected fourteen villages in four towns using multistage cluster sampling and advertised the study to residents after surveying the list of households. Residents on the list of households were encouraged to participate in the study by district leaders (for example, the heads of villages and representatives for women or young adults). All subjects agreed to participate in the present study.

Subjects were excluded if they did not complete the FFQ, reported implausibly low or high energy intakes (less than 2092 kJ/d (500 kcal/d) or greater than 16 736 KJ/d (4000 kcal/d)), or did not provide blood samples. Of the 1292 recruited subjects that met these criteria, additional subjects were excluded if they had a past medical history of myocardial infarction, stroke, heart failure, breast cancer or other cancer (*n* 66), and if they had ever taken medicine for hypertension, diabetes or dyslipidaemia (*n* 316) because of the high probability that they have changed their dieting habits due to their medical condition. Thus, the total number of study subjects used for the analysis was 910 (340 men and 570 women). The Institutional Review Board of the Hanyang University Medical Center approved the present study and written informed consent was obtained from each participant.

General characteristics, anthropometrics and biochemical variables

All of the health examinations and interviews, including the dietary interview, were performed in the Hanyang University Yangpyeong Community Center. Study coordinators and team leaders trained both the interviewers and the technicians with the same protocol consisting of anthropometry, clinical examinations, blood sampling, conducting interviews on lifestyle factors, and dietary assessment, at least one time before and then throughout the survey period.

Age, sex, years of education, family history of disease (hypertension, diabetes and myocardial infarction), marital status, smoking status (current smoker, past smoker or non-smoker), alcohol intake (g/d) and leisure-time physical activity (metabolic equivalent task (MET)-h/week) were collected by an interview-administered questionnaire. Height was measured with a standard height scale to the nearest 0·1 cm and weight was measured with a metric weight scale to the nearest 0·1 kg. BMI was calculated by taking the weight (kg) and dividing it by the height squared (m^2). The waist was measured at the smallest horizontal trunk circumference. The blood pressure was measured by auscultation using a standard sphygmomanometer and a standard cuff and a second measurement was taken after the subject was allowed to rest for at least 5 min. If these measurements differed by more than 5 mmHg, an additional measurement was performed, and the mean value of all measurements was used. The blood samples were collected to measure TAG, HDL-cholesterol (HDL-C) and fasting blood glucose levels after at least 8 h of fasting. TAG and fasting blood glucose were analysed by the Hitachi 747 Automatic Analyzer (Hitachi, Tokyo, Japan) and the HDL was measured directly (Kyowa Medix, Tokyo, Japan).

Metabolic syndrome

The metabolic syndrome was diagnosed if the subject had three or more risk determinants according to the Third Report of The National Cholesterol Education Program Adult Treatment Panel using the standard waist circumference for Asians[20]. The value used for defining abdominal obesity in men was a waist circumference $\geq$90 cm and $\geq$80 cm in women. Another risk factor used a low HDL-C $<$400 mg/l in men and $<$500 mg/l in women. Additional risk factors for both men and women were defined as having a high blood pressure $\geq$130/85 mmHg, a high fasting glucose $\geq$1100 mg/l, or a hypertriacylglycerolaemia $\geq$1500 mg/l.

Dietary assessment and calculation of glycaemic index and glycaemic load

Well-trained interviewers collected dietary data with a FFQ that asked each participant to provide their usual intake of 121 food items over a period of 12 months. The FFQ was modified from an FFQ that was used in a previous large prospective cohort study (Korean Cancer Research Survey) and was validated by Kim *et al.*[21]. The reliability and validity of the FFQ for the total carbohydrate was acceptable (correlation coefficient with multiple 24 h recalls = 0·47; correlation coefficient between two FFQ = 0·41). The FFQ consisted of nine frequency categories ranging from never or $<$ one serving/month to $>$ six servings/d and an open-ended portion size for each food item. All frequencies were standardised into 'times per day' by using the conversion factors 30·4 d or 4·3 weeks per month. Food intake and nutrient intake per d were calculated by using a weighted frequency per d, a portion size per unit, and the recipe and nutrient database, which was provided by the Korean Nutrition Association (Can Pro 2.0)[22].

The GL was calculated by multiplying the carbohydrate content of each food item by its GI. Each GL value is multiplied by its respective frequency of consumption and then the sum of all of the GL values is added to account for all food items that were consumed. The dietary GL thus represents the quality and quantity of the total intake of carbohydrates. Each unit of dietary GL represents the equivalent of 1 g carbohydrate from glucose. The overall dietary GI was calculated by dividing the average daily GL by the average daily carbohydrate intake. The overall dietary GI represents the GL per unit of carbohydrate and reflects the overall quality of carbohydrate intake. The GI value for each food item was obtained from the international table of GI[9], the GI online database maintained by the University of Sydney[23] and from the publication that lists the GI of Korean foods[24]. The reference of GI values was glucose (GI for glucose = 100). When several GI values were available for a food item, the mean GI value was used for the analysis. For foods for which a GI value had not been determined, a value was assigned based on the most similar food item. Furthermore, food items with very low carbohydrate content were ignored because their GI values cannot be accurately measured. Of the 121 food items listed in the FFQ, the GI and GL values were calculated from sixty-four food items with GI values ranging from 14 to 105. In the present study, the carbohydrate content of these sixty-four food items contributed 94·4 (SD 2·8) % of the total available carbohydrate intake.

Statistical analysis

Nutrient intakes, including carbohydrate, GI and GL, were used as total energy-adjusted values, using the residual

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method[25]. The general characteristics of subjects by sex and across quintile categories of carbohydrate, GI and GL were assessed using both a *t* test and the linear regression for continuous variables. For categorical variables, age-standardised proportion was used and χ^2 tests were performed to test differences between proportions. The OR and their 95 % CI for the metabolic syndrome were estimated using the individuals in the lowest quintile as the referent group for each carbohydrate, GI and GL category after adjusting for age, education years, smoking status (current, past or non-smoker), alcohol intake (g/d), family history of diseases such as hypertension, diabetes and myocardial infarction (yes/no), leisure-time physical activity (metabolic equivalent task (MET)-h/week) and energy intake (kJ/d). Categorised variables were treated as continuous variables assigned with the median value within the category in either the linear or logistic regression models in order to estimate the linear trends across the quintile categories of carbohydrate, GI and GL. In order to assess whether the effects of carbohydrate, GI and GL on the risk of the metabolic syndrome were dependent on the BMI, the BMI was categorised into two groups: $<25\,kg/m^2$ and $\geq 25\,kg/m^2$. TAG was right-skewed, so it was log-transformed in the analysis. All analyses were conducted using SAS software (version 9.1; SAS Institute Inc., Cary, NC, USA).

Results

Table 1 shows selected characteristics of subjects according to quintiles of carbohydrate, GI and GL values. Men with the high dietary carbohydrate, GI and GL tended to be older and more educated, and while they consumed more carbohydrates they drank less alcohol, had less total energy intake, and less of their percentage of energy came from fat. Women with the high dietary carbohydrate, GI and GL were also older and consumed more total energy intake and carbohydrates, but less of their energy percentage came from fat. A significant relationship could be found between dietary carbohydrate, GI and GL intakes and other components of the metabolic syndrome; in particular, a high level of HDL-C was related to high dietary carbohydrate and GL intakes in both men and women. A high level of TAG was also related to a high intake of dietary carbohydrates in both men and women.

The age-adjusted prevalence of the metabolic syndrome including individual components is shown in Table 2. The prevalence of the metabolic syndrome was 23·3 % in men and 34·6 % in women. Of the individual components of the metabolic syndrome, high blood pressure was most prevalent for men (49·4 %) and high waist circumference for women (72·5 %). The increased trends of the prevalence across dietary carbohydrate, GI and GL were shown in HDL-C for both men and women, and for the metabolic syndrome in only the women. The prevalence in the last quintile of carbohydrate intake, GI and GL was more than 40 % in women.

Table 3 shows the relationship between carbohydrate, GI and GL intakes and the risk of the metabolic syndrome and its components by multivariate analysis. For men, among the various components of the metabolic syndrome, the prevalence of low HDL-C increased with high GL intake (*P* for trend=0·03) but no dietary factor showed a significant relationship with the metabolic syndrome. For women, a high risk of low HDL-C was significantly related to increased dietary carbohydrate, GI and GL intakes, and this result was consistent when their intakes were analysed as categorical and continuous variables. Furthermore, the HDL-C level significantly decreased with dietary carbohydrate, GI and GL intakes ($\beta = -5{\cdot}53$, $P=0{\cdot}0005$ per 100 g increment of carbohydrate; $\beta = -2{\cdot}91$, $P=0{\cdot}0267$ per 10-unit increment of GI; and $\beta = -4{\cdot}01$, $P=0{\cdot}0001$ per 50-unit increment of GL) (data not shown).

With regard to hypertriacylglycerolaemia risk, a significant increasing trend was not shown when dietary factors were treated as categorical variables but was shown when they were treated as continuous variables in women. The OR of hypertriacylglycerolaemia risk was 1·86 (95 % CI 1·09, 3·18) per 10-unit increment of GI and 1·60 (95 % CI 1·02, 2·55) per 50-unit increment of GL. In addition, the level of TAG significantly increased by 1·3 mg/l per 100 g increment of dietary carbohydrate and by 1·0 mg/l per 50-unit increment of GL (data not shown).

The prevalence of the metabolic syndrome increased with increased dietary carbohydrate (*P* for trend=0·03), GI (*P* for trend=0·03) and GL (*P* for trend=0·02) intakes in women. The OR of the metabolic syndrome was 2·37 (95 % CI 1·19, 4·74) per 100 g increment of dietary carbohydrate intake and 1·75 (95 % CI 1·12, 2·73) per 50-unit increment of GL intake. The significant relationships of carbohydrate, GI and GL intakes with the risk of low HDL-C, hypertriacylglycerolaemia and the metabolic syndrome were not changed even after additional adjustment for food groups such as green vegetables and fruits.

The relationship according to obesity status (BMI $< 25\,kg/m^2$ and $\geq 25\,kg/m^2$) in women was examined in order to better understand the relationship of the quantity and quality of carbohydrates with the metabolic syndrome (Fig. 1 a–c). Although the interaction effects were not significant (interaction *P* value=0·40 for carbohydrate; 0·16 for GI; 0·13 for GL), the increased risk of the metabolic syndrome from high intakes of carbohydrate, GI and GL was shown among women with a BMI $\geq 25\,kg/m^2$. The OR of the metabolic syndrome for the highest quintiles of intake and BMI $\geq 25\,kg/m^2$ was 6·44 (95 % CI 2·16, 19·2) for carbohydrate, 10·4 (95 % CI 3·24, 33·3) for GI and 6·68 (95 % CI 2·30, 19·4) for GL when the lowest quintiles of intake and BMI $< 25\,kg/m^2$ were used for comparison.

Given that HDL-C is a predominant component of the metabolic syndrome, we also examined the interactive effect of dietary factors and BMI on the risk of low HDL-C in women (Fig. 1 d–f). For women with a BMI $\geq 25\,kg/m^2$, the risk was related to high intakes of carbohydrates, GI and GL. However, a significant relationship was not shown in women with a BMI $< 25\,kg/m^2$. The interactive effects with BMI were significant for all carbohydrate (*P*=0·03), GI (*P*=0·009) and GL (*P*=0·0003) intakes. When HDL-C was additionally adjusted, the prevalence of the metabolic syndrome in women with high intakes of carbohydrates, GI and GL with a BMI $\geq 25\,kg/m^2$ was still significantly high (data not shown).

Discussion

The present study found that the quantity and quality of carbohydrates were positively related to the risk of the metabolic

Table 1. Selected age-adjusted characteristics of subjects according to quintiles (Q) of dietary carbohydrate, glycaemic index and glycaemic load

(Mean values or percentages)

	Carbohydrate				Glycaemic index				Glycaemic load			
	Q1	Q3	Q5	*P* for trend	Q1	Q3	Q5	*P* for trend	Q1	Q3	Q5	*P* for trend
Men												
Median	263·5	320·6	365·8		54·3	58·5	62·6		154·0	193·5	227·8	
Age (years)	53·6	57·9	61·2	<0·001	56·9	56·8	61·7	0·03	52·6	58·2	62·3	<0·001
Education (total years)	9·8	10·4	10·7	0·04	10·0	9·9	10·5	0·44	9·7	10·1	10·7	0·01
Exercise (MET-h/week)	22·2	27·4	15·0	0·35	18·0	26·2	25·0	0·28	25·3	21·4	21·9	0·65
Family history (%)*	20·6	26·0	25·5	0·66	31·3	22·9	37·2	0·42	22·2	23·2	25·1	0·22
Current smoker (%)	36·8	35·3	34·0	0·13	41·1	40·0	31·2	0·44	42·3	43·0	31·3	0·08
Alcohol (g/d)	37·2	27·4	25·8	0·07	37·8	29·1	21·6	0·005	41·4	35·9	22·4	0·01
BMI (kg/m^2)	23·7	23·8	23·8	0·82	23·5	23·4	24·2	0·07	23·6	23·5	24·2	0·16
Daily dietary intake (per d)†												
Total energy (kJ)	10 447·9	8921·5	9540·4	0·02	9762·5	9611·1	9166·3	0·53	10 670·0	9086·4	9608·6	0·01
Carbohydrate (% energy)	58·0	67·5	74·5	<0·001	63·2	67·1	69·2	<0·001	58·3	67·6	73·5	<0·001
Fat (% energy)	26·7	19·0	13·6	<0·001	21·9	19·6	17·5	<0·001	26·4	19·0	14·2	<0·001
Total fibre (g)	8·2	9·0	8·2	0·91	10·6	8·1	7·4	<0·001	8·4	8·8	7·4	0·005
Carbohydrate (g)	257·9	320·8	366·3	<0·001	299·3	323·1	329·5	<0·001	260·3	322·3	358·9	<0·001
Glycaemic index	57·5	59·3	59·8	<0·001	54·0	58·5	64·5	<0·001	56·0	58·4	63·0	<0·001
Glycaemic load	148·1	190·1	219·0	<0·001	161·6	189·0	212·4	<0·001	145·4	187·9	225·1	<0·001
Waist circumference (cm)	85·9	86·3	86·3	0·78	85·6	84·8	87·5	0·08	85·7	86·0	86·8	0·53
TAG (mg)	115·4	130·0	139·7	<0·001	120·2	140·0	125·9	0·97	114·6	151·9	131·2	0·17
HDL-cholesterol (mg)	50·3	49·8	45·6	0·01	49·9	48·1	48·9	0·55	50·5	48·4	45·9	0·02
Systolic blood pressure (mmHg)	125·2	124·1	129·2	0·05	125·4	127·0	125·6	0·78	24·9	124·6	127·7	0·20
Diastolic blood pressure (mmHg)	81·2	81·1	85·5	0·01	82·1	82·1	82·2	0·79	81·2	81·2	83·5	0·09
Fasting blood glucose (mg)	84·8	95·8	96·1	0·37	99·2	98·7	97·4	0·88	94·8	98·3	96·3	0·46
Women												
Median	265·7	319·8	360·0		54·0	58·6	63·1		151·6	193·5	223·4	
Age (years)	52·8	53·7	61·2	<0·001	51·4	54·3	56·6	<0·001	51·4	54·1	61·2	<0·001
Education (total years)	9·4	9·3	8·9	0·26	9·5	9·3	9·0	0·29	9·5	9·4	9·2	0·25
Exercise (MET-h/week)	17·4	12·5	12·9	0·13	16·9	17·5	14·0	0·63	16·5	18·9	14·3	0·45
Family history (%)*	33·0	23·7	34·3	0·15	32·2	28·2	28·7	0·30	37·2	26·1	25·3	0·05
Current smoker (%)	7·1	2·9	2·6	0·06	6·8	3·1	2·4	0·44	8·1	3·9	2·2	0·03
Alcohol (g/d)	2·5	2·6	2·7	0·85	2·6	2·6	1·3	0·06	2·6	2·6	1·9	0·63
Height (cm)	154·0	153·1	153·5	0·67	154·1	153·1	153·1	0·07	153·8	153·8	153·3	0·41
BMI (kg/m^2)	24·6	24·3	24·6	0·89	24·7	24·6	24·8	0·67	24·4	24·1	24·6	0·77
Daily dietary intake (per d)†												
Total energy (kJ)	7443·8	7698·1	8744·1	0·001	8061·7	7489·4	8189·3	0·87	7500·7	7733·7	8783·9	0·001
Carbohydrate (% energy)	61·6	68·7	74·2	<0·001	63·8	69·9	68·2	<0·001	61·2	69·2	72·7	<0·001
Protein (% energy)	16·4	14·7	13·2	<0·001	16·5	14·3	14·5	<0·001	16·8	14·6	13·2	<0·001
Fat (% energy)	24·1	18·1	13·8	<0·001	22·4	17·1	18·5	<0·001	24·3	17·7	15·1	<0·001
Total fibre (g)	8·1	8·5	8·9	0·04	10·8	8·3	7·5	<0·001	9·2	8·6	8·0	<0·001
Carbohydrate (g)	259·2	319·2	364·0	<0·001	296·5	324·2	315·6	<0·001	264·1	323·1	355·8	<0·001
Glycaemic index	57·4	59·5	59·1	<0·001	53·2	58·6	65·0	<0·001	55·0	58·3	62·9	<0·001
Glycaemic load	149·2	189·9	215·3	<0·001	158·6	190·1	205·2	<0·001	144·9	188·2	222·7	<0·001
Waist circumference (cm)	85·6	84·3	85·6	0·83	85·7	84·8	86·0	0·84	85·0	84·4	85·7	0·79
TAG (mg)	105·6	116·5	117·4	0·03	107·9	115·8	108·8	0·86	103·7	108·0	117·7	0·12
HDL-cholesterol (mg)	52·8	50·0	47·6	<0·001	51·9	49·6	48·5	0·02	53·3	49·3	47·6	<0·001
Systolic blood pressure (mmHg)	124·5	124·9	121·6	0·15	123·3	122·1	126·3	0·11	123·7	120·7	122·3	0·33
Diastolic blood pressure (mmHg)	81·1	80·7	79·5	0·12	80·9	80·0	81·6	0·61	80·5	78·5	79·3	0·17
Fasting blood glucose (mg)	95·2	101·9	93·3	0·72	93·1	96·8	98·3	0·09	93·7	97·5	95·1	0·66

MET, metabolic equivalent task.

* Family history of hypertension, diabetes, myocardial infarction.

† All nutrients were energy adjusted except for total energy intake.

Table 2. Prevalence of individual components of the metabolic syndrome according to carbohydrate (CHO), glycaemic index (GI) and glycaemic load (GL) quintile (Q) categories*

		Quintiles of CHO, GI and GL					
	Total	Q1	Q2	Q3	Q4	Q5	*P* for trend
Men							
CHO†		263·5	303·6	320·6	339·8	365·8	
WC	32·7	40·7	34·5	30·6	21·5	39·1	0·55
TG	39·7	39·7	44·4	39·7	43·9	40·1	0·61
HDL-C	25·0	20·9	16·0	24·5	25·6	39·0	0·01
BP	49·4	48·9	40·9	41·1	56·9	63·9	0·02
FBG	13·8	12·3	18·3	14·3	19·8	6·9	0·52
MS	23·3	24·2	17·0	22·8	23·0	33·0	0·11
GI†		54·3	56·9	58·5	59·9	62·6	
WC	32·7	33·1	26·6	31·4	32·2	41·2	0·26
TG	39·7	35·6	46·7	44·2	36·8	32·8	0·59
HDL-C	25·0	11·1	29·8	27·8	30·7	23·9	0·07
BP	49·4	53·4	53·1	51·7	42·5	48·1	0·47
FBG	13·8	19·4	11·8	14·8	10·3	14·0	0·37
MS	23·3	17·7	25·7	26·9	22·0	23·3	0·47
GL†		154·0	178·5	193·5	206·9	227·8	
WC	32·7	39·6	29·8	24·8	30·5	40·7	0·67
TG	39·7	40·3	37·2	50·6	43	35·5	0·78
HDL-C	25·0	19·2	15·0	26·0	30·3	37·9	<0·001
BP	49·4	49·9	42·3	40·0	60·3	57·7	0·1
FBG	13·8	8·6	17·3	16·0	16·9	8·5	0·81
MS	23·3	22·5	16·3	20·4	31·3	30·4	0·04
Women							
CHO†		265·7	299·6	319·8	336·4	360·0	
WC	72·5	76·4	69·9	67·1	75·5	81·8	0·53
TG	25·3	25·6	18·8	26·9	30·0	23·2	0·38
HDL-C	52·8	40·2	48·9	52·9	58·4	65·8	<0·001
BP	45·1	44·4	48·8	48·0	37·6	46·3	0·58
FBG	11·2	11·4	5·9	18·4	13·1	7·0	0·53
MS	34·6	29·4	26·0	38·5	36·4	42·7	0·03
GI†		54·0	56·7	58·6	60·4	63·1	
WC	72·5	72·9	71·1	70·2	75·8	73·5	0·55
TG	25·3	26·0	25·7	23·7	26·5	24·7	0·92
HDL-C	52·8	44·5	50·5	58·0	52·6	59·9	0·03
BP	45·1	44·9	41·4	44·9	43·4	52·1	0·25
FBG	11·2	5·6	10·8	14·1	10·2	15·6	0·06
MS	34·6	30·0	33·3	34·4	36·9	40·9	0·06
GL†		151·6	179·5	193·3	204·6	223·4	
WC	72·5	72·7	71·8	68·4	76·5	74·4	0·61
TG	25·3	26·6	24·6	24·3	25·5	28	0·69
HDL-C	52·8	40	47·6	54·1	59·6	61·9	<0·001
BP	45·1	43·4	53·1	42·8	39·2	51·1	0·59
FBG	11·2	8·1	12·8	12·8	12·8	12·1	0·75
MS	34·6	28·6	32·9	34·8	36·1	43·6	0·05

WC, high waist circumferences (≥90 cm in men and ≥80 cm in women); TG, hypertriacylglycerolaemia (≥1500 mg/l); HDL-C, low HDL-cholesterol (<400 mg/l in men, <500 mg/l in women); BP, high blood pressure (≥130/85 mmHg); FBG, high fasting glucose (≥1100 mg/l); MS, metabolic syndrome (≥three each components of MS).

* All values are age adjusted. In addition, dietary carbohydrate, GI and GL were energy adjusted.

† Median values for each quintile are presented.

syndrome, as well as the risk of individual metabolic phenotypes in women, but not in men. In addition, their adverse effects on metabolic syndrome risk were clearly evident in obese women, but not in women with a normal weight. Of the individual metabolic phenotypes studied, the HDL-C may be the key component mediating the risk of metabolic syndrome and the quantity and quality of carbohydrate intake.

This study population had considerably higher carbohydrate intakes than Caucasians and even the Japanese[10–14,26–30]. In the present study, the food item that contributed most to total carbohydrate intake was rice (contributing about 60 %, and its GI value was 64 when the reference of the GI value was glucose). Instant noodles and sugar also contributed to carbohydrate intake (12 and 8 %, respectively), and their GI were high (68 for sugar and 46 for instant noodles). Foods with low GI values contributing to total carbohydrate intake included soya beans (GI = 18) and barley (GI = 25).

The prevalence of the metabolic syndrome in men and women of the present study (23·3 % in men, 34·6 % in women) was similar to that in middle-aged Korean men (20–26 %) and women (30–40 %) of other studies[31–34]. In the present study, the findings that high blood pressure was prevalent in men while high waist circumference and low HDL-C were prevalent in women were consistent with the data published in the Korean National Health and Nutrition Survey[18,32].

Table 3. Risk for individual components of the metabolic syndrome by carbohydrate (CHO), glycaemic index (GI) and glycaemic load (GL) quintile (Q) category* (Odds ratios and 95 % confidence intervals)

	Quintiles of dietary CHO, GI and GL										
	Q1	Q2		Q3		Q4		Q5		Continuous variables	
	OR	OR	95 % CI	OR	95 % CI	OR	95 % CI	OR	95 % CI	*P* for trend	OR	95 % CI
Men												
CHO†	263·5	303·6		320·6		339·8		365·8			per 100 g increment	
WC	1·00	1·24	0·37, 4·21	0·80	0·22, 2·91	0·22	0·05, 0·85	0·82	0·24, 2·73	0·32	0·64	0·23, 1·78
TG	1·00	1·37	0·60, 3·10	1·63	0·69, 3·83	1·60	0·68, 3·77	1·49	0·64, 3·46	0·31	1·45	0·71, 2·95
HDL-C	1·00	0·54	0·19, 1·52	0·97	0·35, 2·68	0·99	0·36, 2·69	1·76	0·68, 4·57	0·17	1·82	0·76, 4·36
BP	1·00	0·53	0·24, 1·16	0·77	0·34, 1·74	0·98	0·43, 2·24	1·53	0·67, 3·49	0·21	1·60	0·80, 3·19
FBG	1·00	2·92	0·88, 9·69	1·97	0·57, 6·85	3·05	0·91, 10·19	0·73	0·17, 3·11	0·96	0·89	0·33, 2·35
MS	1·00	0·61	0·21, 1·80	1·32	0·46, 3·81	1·08	0·37, 3·10	2·11	0·77, 5·75	0·11	1·71	0·70, 4·20
GI†	54·3	56·9		58·5		59·9		62·6			per 10-unit increment	
WC	1·00	1·29	0·35, 4·73	0·98	0·24, 3·95	1·05	0·27, 3·99	1·64	0·43, 6·25	0·54	1·24	0·51, 3·04
TG	1·00	1·77	0·74, 4·19	1·71	0·71, 4·08	0·86	0·34, 2·16	0·91	0·36, 2·29	0·44	0·64	0·30, 1·35
HDL-C	1·00	4·24	1·28, 14·0	5·15	1·54, 17·2	4·74	1·41, 15·8	3·91	1·13, 13·4	0·08	1·51	0·70, 3·24
BP	1·00	0·97	0·42, 2·23	0·91	0·40, 2·09	0·65	0·27, 1·54	0·67	0·27, 1·62	0·26	0·50	0·25, 1·01
FBG	1·00	0·73	0·24, 2·21	0·79	0·26, 2·42	0·50	0·14, 1·78	0·70	0·22, 2·25	0·49	0·68	0·25, 1·79
MS	1·00	2·95	0·97, 8·96	3·19	1·02, 9·92	1·41	0·43, 4·58	1·60	0·49, 5·19	0·98	0·60	0·24, 1·46
GL†	154·0	178·5		193·5		206·9		227·8			per 50-unit increment	
WC	1·00	1·01	0·27, 3·72	0·94	0·29, 3·10	0·43	0·13, 1·46	0·91	0·27, 3·05	0·59	0·89	0·46, 1·71
TG	1·00	1·30	0·56, 3·00	2·08	0·90, 4·78	2·05	0·87, 4·80	1·16	0·47, 2·82	0·4	1·06	0·66, 1·68
HDL-C	1·00	0·69	0·24, 1·98	1·09	0·39, 2·98	1·34	0·49, 3·60	2·60	0·96, 7·03	0·03	1·55	0·88, 2·71
BP	1·00	0·83	0·37, 1·82	0·54	0·24, 1·22	1·58	0·69, 3·60	0·97	0·42, 2·23	0·72	1·04	0·66, 1·64
FBG	1·00	2·87	0·86, 9·59	2·77	0·83, 9·27	2·68	0·79, 9·04	0·57	0·11, 2·75	0·84	0·81	0·43, 1·53
MS	1·00	0·85	0·29, 2·48	1·09	0·38, 3·12	2·18	0·79, 5·99	1·70	0·59, 4·88	0·15	1·11	0·63, 1·96
Women												
CHO†	265·7	299·6		319·8		336·4		360·0			per 100 g increment	
WC	1·00	0·37	0·14, 0·98	0·57	0·21, 1·53	0·67	0·23, 1·91	0·71	0·23, 2·16	0·54	0·78	0·31, 1·96
TG	1·00	0·44	0·18, 1·08	1·08	0·51, 2·28	1·43	0·68, 3·03	0·83	0·36, 1·92	0·58	1·45	0·71, 2·94
HDL-C	1·00	0·98	0·52, 1·84	1·49	0·79, 2·79	1·96	1·02, 3·79	3·54	1·69, 7·40	<0·001	2·96	1·60, 5·45
BP	1·00	1·82	0·90, 3·67	1·26	0·63, 2·52	0·62	0·29, 1·30	1·20	0·57, 2·54	0·63	0·92	0·50, 1·70
FBG	1·00	0·40	0·11, 1·41	1·58	0·61, 4·08	1·11	0·39, 3·14	0·49	0·14, 1·67	0·72	0·87	0·36, 2·08
MS	1·00	0·62	0·27, 1·42	1·57	0·73, 3·37	1·38	0·63, 3·02	2·05	0·92, 4·56	0·03	2·37	1·19, 4·74
GI†	54·0	56·7		58·6		60·4		63·1			per 10-unit increment	
WC	1·00	1·40	0·52, 3·75	1·21	0·43, 3·40	1·59	0·50, 5·06	0·94	0·32, 2·76	0·86	0·57	0·27, 1·22
TG	1·00	1·19	0·51, 2·76	1·17	0·50, 2·73	1·60	0·63, 4·07	1·85	0·78, 4·36	0·12	1·86	1·09, 3·18
HDL-C	1·00	2·09	1·09, 4·01	2·19	1·13, 4·25	2·26	1·06, 4·82	2·64	1·29, 5·37	0·01	1·77	1·06, 2·93
BP	1·00	0·86	0·42, 1·76	0·97	0·48, 1·97	0·93	0·41, 2·13	1·03	0·48, 2·21	0·84	0·86	0·50, 1·45
FBG	1·00	1·37	0·40, 4·68	1·87	0·58, 6·00	1·71	0·44, 6·50	2·56	0·79, 8·22	0·09	1·38	0·68, 2·78
MS	1·00	1·54	0·66, 3·56	2·08	0·92, 4·70	2·54	1·00, 6·45	2·40	1·01, 5·66	0·03	1·37	0·79, 2·34
GL†	151·6	179·5		193·3		204·6		223·4			per 50-unit increment	
WC	1·00	0·66	0·24, 1·80	0·65	0·25, 1·71	1·27	0·46, 3·50	0·5	0·17, 1·48	0·5	0·68	0·37, 1·25
TG	1·00	0·79	0·34, 1·84	0·97	0·44, 2·13	1·32	0·60, 2·90	1·52	0·67, 3·44	0·23	1·60	1·02, 2·55
HDL-C	1·00	1·58	0·83, 2·99	1·83	0·98, 3·41	3·13	1·59, 6·18	3·67	1·77, 7·61	<0·001	2·21	1·47, 3·33
BP	1·00	2·16	1·06, 4·39	1·16	0·58, 2·30	0·61	0·29, 1·28	1·06	0·5, 2·24	0·45	0·89	0·59, 1·34
FBG	1·00	1·89	0·60, 5·91	1·89	0·61, 5·84	1·44	0·43, 4·74	1·71	0·54, 5·41	0·46	1·09	0·61, 1·93
MS	1·00	1·40	0·61, 3·23	2·15	0·98, 4·71	2·16	0·96, 4·84	2·44	1·07, 5·55	0·02	1·75	1·12, 2·73

WC, high waist circumferences (≥90 cm in men and ≥80 cm in women); TG, hypertriacylglycerolaemia (1500 mg/l); HDL-C, low HDL-cholesterol (<400 mg/l in men, <500 mg/l in women); BP, high blood pressure (≥130/85 mmHg); FBG, high fasting glucose (≥1100 mg/l), MS, metabolic syndrome (≥three each components of MS).

* OR adjusted for age, smoking status (never, past, current), alcohol intake (g), education (years), family history of disease such as hypertension, diabetes and myocardial infarction (yes or no), BMI (kg/m^2), physical activity (metabolic equivalent task-h/week), fibre, total energy intake. All nutrients were energy adjusted except for total energy intake.

† Median values for each quintile are presented.

Given that there has been no observational study, to our knowledge, examining the relationship of GI and GL with all components of the metabolic syndrome, the present study examined the relationship for all components. Findings of previous studies on the relationship for each component were not consistent between GI and GL, or between sexes. Dietary GL has been shown to be inversely related to HDL-C in other cross-sectional studies[11–14,35]. However, the relationship between GI and HDL-C was not consistent. An inverse correlation has been reported in some studies[12,13], but not in others[11,14,30]. The present study found that high GL were related to the increased risk trend of having low HDL-C in

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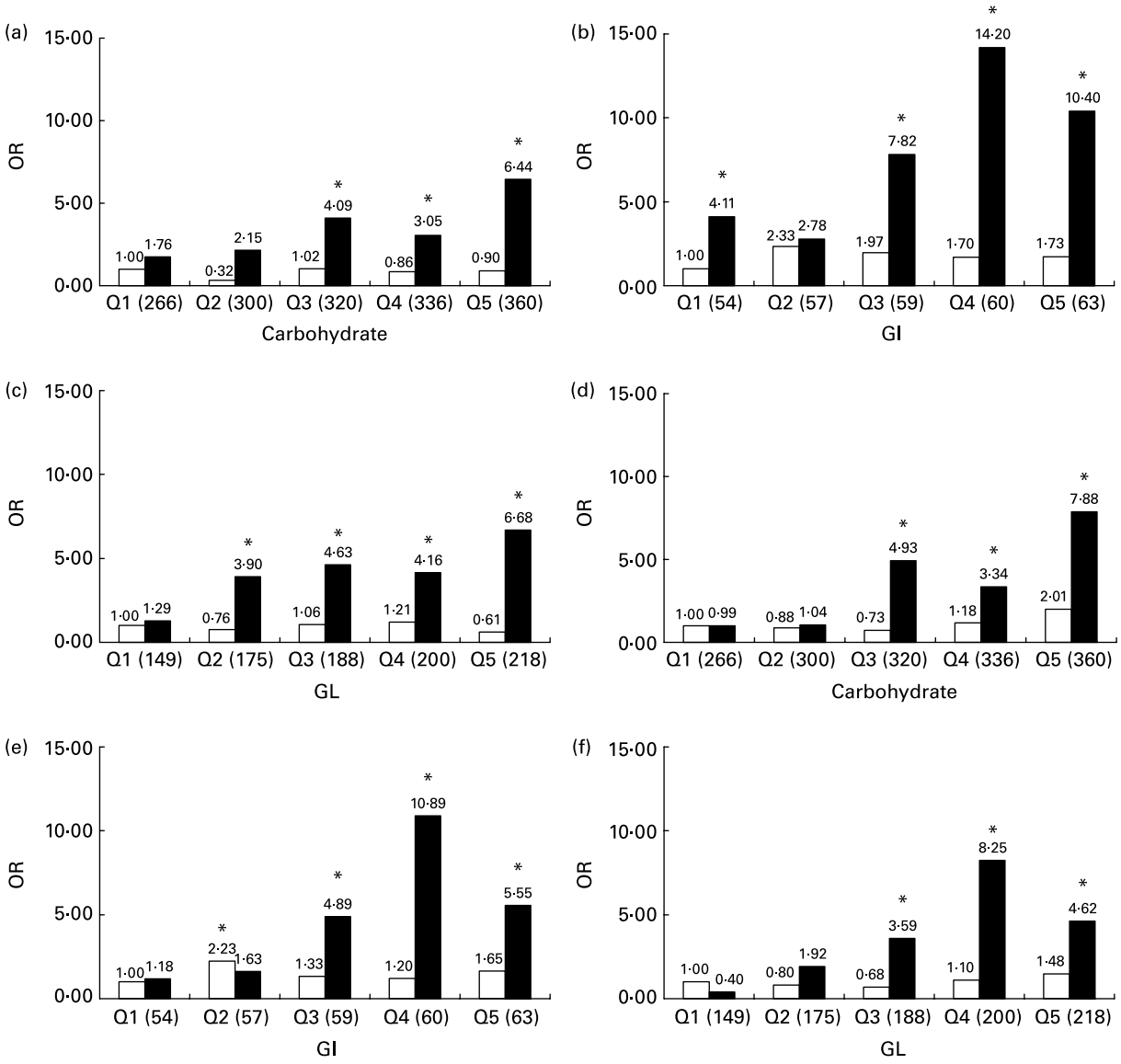

Fig. 1. The interaction effect of BMI and carbohydrate, glycaemic index (GI) and glycaemic load (GL) on the metabolic syndrome and low HDL-cholesterol in women. (a) The effect of BMI and carbohydrate intake on the metabolic syndrome. (b) The effect of BMI and GI on the metabolic syndrome. (c) The effect of BMI and GL on the metabolic syndrome. (d) The effects of BMI and carbohydrate intake on low HDL-cholesterol levels. (e) The effects of BMI and GI on low HDL-cholesterol levels. (f) The effects of BMI and GL on low HDL-cholesterol levels. OR are shown after adjusting for age, smoking status, alcohol intake, education, family history of disease such as hypertension, diabetes and myocardial infarction, BMI, physical activity, fibre and energy intake. Carbohydrate, GI and GL are classified by quintiles (Q). Values in parentheses are median values in each category. All nutrients were energy adjusted, except for total energy intake. * The CI of the values does not include 1·0. (□), BMI $< 25\,kg/m^2$; (■), BMI $\geq 25\,kg/m^2$.

both men and women. In addition, significant adverse effects of dietary carbohydrate intake and GI on these risks were observed in women. In terms of fasting blood glucose, the present study did not find an increased risk of high fasting blood glucose by dietary carbohydrate intake. No association was observed between GI, GL and fasting blood glucose in elderly men in an observational study[30] but a positive relationship was shown in a cross-sectional study for Japanese women[14] and several randomised controlled trials[36,37]. The role of quantity and quality of carbohydrates in abdominal obesity has not yet been investigated. However, randomised clinical trials have shown larger decreases in fat mass in low-GI diets than in high-GI diets[38,39]. The present study did not find a significant relationship between high waist circumferences with GI or GL. Several studies demonstrated that both GI and GL were positively correlated with TAG[11,13,14], but one study for elderly men found that there was no association between GI and TAG[30]. In the present study, high carbohydrate, GI and GL were related to the high risk of hypertriacylglycerolaemia in women only.

An increased risk of the metabolic syndrome across quintiles of GI and GL was identified in women, but not in men. This may partly be explained by the recent suggestion that the risk of the metabolic syndrome may be related to female sex hormones inducing an insulin resistance syndrome, implying a presumably higher susceptibility to the risk of the metabolic syndrome in women than men[40–42]. However, there was no difference when the premenopausal women were

compared with the postmenopausal women in the present study (data not shown). Sex hormones may not be enough to explain the sex difference found in the present study. The interesting finding is that the effects of carbohydrate, GI and GL on the risk of the metabolic syndrome were dependent on BMI level with a cut off point of $\geq 25\,kg/m^2$. The Nurses' Health Study showed that the effect of carbohydrate intake on the risks of some components (TAG and HDL-C) of the metabolic syndrome was stronger in women with high BMI levels[11]. The present study also demonstrated the significant interactive effects of carbohydrate, GI and GL intakes and BMI level on the risk of low-HDL-C level. Thus, we additionally adjusted for HDL-C to examine whether the risks of developing the metabolic syndrome dependent on levels of adiposity is only through HDL-C. The risk of the metabolic syndrome for women with high carbohydrate intakes and high BMI levels remained high (data not shown). This finding suggests that the risks of developing the metabolic syndrome through clustering components may depend on levels of adiposity.

The present study has limitations. The FFQ used in the study was modified in order to account for the ability to assess the quality and quantity of carbohydrate intake from the initial validated FFQ[21] but was not additionally validated with other dietary measurements such as multiple 24 h dietary recalls. In a previous study[11], HDL-C or TAG was suggested to provide the validity of dietary carbohydrate, GI and GL intake as 'alloyed gold standard', although these physiological responses respond to other factors. Thus, the relationships between carbohydrate, GI and GL intake and HDL-C (or TAG in women) shown in the present study may provide a part of validity. However, the assessment of the validity with more accurate measures is still needed. Residual confounding may exist due to collinearity within dietary factors (for example, dietary fat; r −0·6 between carbohydrate and percentage energy from fat), although the apparent relationship between the quantity and quality of the carbohydrates and the metabolic syndrome was shown after the adjustment for covariates. Regardless of these limitations, the present study is the first to report on the effect of both the quality and quantity of carbohydrate intake and metabolic syndrome risk in an Asian population whose staple food is white rice. Moreover, the findings provide valuable insight on CVD and diabetes prevention.

In conclusion, high intakes of carbohydrates, GI or GL were considerably related to a high risk of the metabolic syndrome in women, but the relationship was dependent on the BMI level. The high risk for women with high BMI levels of developing the metabolic syndrome in the present study can be attributed mainly to an increased risk of low HDL-C. These findings suggest that preventing obesity or substituting carbohydrate sources (for example, brown rice instead of white rice) to reduce GL and overall GI may prove a better strategy to decrease the risk of the metabolic syndrome in populations whose total energy is primarily supplied through carbohydrates rather than limiting the amount of total carbohydrate intake.

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