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Sweetened beverage consumption and increased risk of metabolic syndrome in Mexican adults

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Abstract

Objective: To examine the relationship between sweetened beverage consumption and components of the metabolic syndrome in a Mexican population.

Design: We performed a cross-sectional analysis of data from selected adults participating in the baseline assessment of the Health Workers Cohort Study. Information on participants' sociodemographic characteristics, dietary patterns and physical activity were collected via self-administered questionnaires. Sweetened beverage consumption was evaluated through a validated semi-quantitative FFQ. Anthropometric and clinical measures were assessed with standardized procedures. The definition of metabolic syndrome was determined using criteria from the National Cholesterol Education Program Adult Treatment Panel III. The associations of interest were evaluated by means of linear and logistic regression models.

Setting: The Mexican states of Morelos and Mexico.

Subjects: A total of 5240 individuals aged 20 to 70 years (mean 39·4 (SD 11·5) years) were evaluated.

Results: Overweight/obesity prevalence was 56·6%. The prevalence of metabolic syndrome in this sample was 26·6%. We found that for each additional daily sweetened beverage serving consumed, participants experienced an average increase of 0·49 mmol/l in TAG and a decrease in HDL cholesterol of 0·31 mmol/l. Subjects consuming more than two servings of sweetened beverages daily were at 2·0 times greater risk of metabolic syndrome than those who did not consume sweetened beverages. We also observed that higher sweetened beverage consumption increased the risk of all components of the metabolic syndrome.

Conclusions: Our data support the hypothesis that sweetened beverage consumption increases the risk of metabolic syndrome in Mexican adults, possibly by providing excess energy and large amounts of rapidly absorbable sugars.

Keywords
Sweetened beverages
Metabolic syndrome
Mexican population

Metabolic syndrome (MS) comprises of a cluster of risk factors for CVD that includes central obesity, dyslipidaemia, hyperglycaemia and hypertension[1,2]. The prevalence of MS is increasing and it now affects 27% of the US population[3]. This epidemic correlates with pronounced changes in the environment, behaviours and lifestyles, and is considered one of the main threats to human health worldwide[4]. Underdeveloped countries are also facing high levels of MS; 26% of the adult Mexican population suffers from this syndrome[5]. Mexico is also facing epidemic levels of CVD and type 2 diabetes mellitus (T2DM), illnesses associated with MS. Ford and colleagues have estimated that the population-attributable fraction associated with MS is ~6–7% for all-cause mortality, 12–17% for CVD and 30–52% for T2DM[6].

Several public health studies have linked sweetened beverage intake and negative health outcomes[7], including serious metabolic disorders such as obesity[8–11], T2DM[8,12], CVD[12] and hypertension[13]. Since Mexico has the third highest rate of sweetened beverage consumption in the world, and 20% of total energy intake comes from sweetened beverages[14], this sweetened beverage consumption seems likely to be related to the elevated national prevalence of MS. However, MS and its risk factors are understudied in Mexico. We thus used data from the Health Workers Cohort Study[11] to examine the relationship

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between sweetened beverage consumption and the prevalence of MS components in Mexican adults.

Experimental methods

Study population

We performed a cross-sectional analysis of data from adults participating in the baseline assessment of the Health Workers Cohort Study in the Mexican states of Morelos and Mexico. The present analysis was performed on data from healthy employees and their relatives from three different health and academic institutions: (i) Instituto Mexicano del Seguro Social (IMSS) and (ii) Instituto Nacional de Salud Pública (INSP), both located in Cuernavaca, Morelos State; and workers at (iii) Universidad Autónoma del Estado de México (UAEM) in Toluca, Mexico State. Subjects recruited for the study were participating in the first stage of an ongoing, long-term cohort study focusing on lifestyle and health. The specifics of the study design, methodology and participants' baseline characteristics have been detailed elsewhere[15–17]. The ethics committees of all participating institutions approved the study protocol and consent forms. Out of a total population of 13 275 study candidates identified between March and April 2006, 9467 employees were invited to participate in the cohort study and a total of 8307 adults were formally enrolled.

For the present analysis we excluded participants with diagnoses of diabetes, hypertension, dyslipidaemia, gout, rheumatoid arthritis and degenerative arthritis (n 2320), and those who had values of plasma glucose ≥7·08 mmol/l (n 628) because this is a criterion for the diagnosis of diabetes. We also excluded subjects who did not satisfy the a priori criterion of a daily energy intake between 2510·4 and 29 288 kJ, and those providing incomplete information on their sweetened beverage consumption (n 119). The remaining 5240 participants were included in our analysis.

Data collection

Demographic characteristics were evaluated by means of self-administered questionnaires. Participants were asked about their physical activity during leisure time, at work and during housework. Each activity was given a value in metabolic equivalent tasks (METS) and total daily METS were computed[18].

Participants were also asked about the weight changes they had experienced within the past year and this information was categorized as no weight change, weight loss or weight gain in the past year (less/more than 5 kg).

Anthropometric and clinical assessment

Weight was measured with a previously calibrated electronic scale (model BC-533; Tanita, Tokyo, Japan), with participants wearing minimal clothing. Height was measured using a conventional stadiometer. Waist circumference was measured at the high point of the iliac crest at the end of normal expiration, to the nearest 0·1 cm, with a steel measuring tape. BMI was calculated as the ratio of weight to the square of height (kg/m^2) from standardized measurements of weight and height; and the proportion of body fat was estimated via the reference technique of dual-energy X-ray absorptiometry performed with a Lunar DPXL whole-body X-ray densitometer (Lunar Radiation Corp., Madison, WI, USA; software version 1·35, fast scan mode).

Blood pressure was measured with an automatic digital blood pressure monitor. Participants were seated with their right arm resting at heart level. Up to three blood pressure measurements were obtained from each participant. All measurement procedures were performed by nurses trained to use standardized procedures (reproducibility was evaluated, resulting in concordance coefficients between 0·83 and 0·90).

A fasting venous blood sample was collected from each participant; fasting time was ≥8 h to be consistent with previous analyses of data from adults participating in the National Health and Nutrition Examination Survey[19]. Plasma glucose was measured with the oxidized glucose method, TAG with a colorimetric method following enzymatic hydrolysis performed with the lipase technique, and HDL cholesterol (HDL-C) by the clearance method. All biomedical assays were performed with a Selectra XL instrument (Randox Laboratories Ltd, Antrim, UK), in concordance with the procedures of the International Federation of Clinical Chemistry and Laboratory Medicine[20,21].

Dietary assessment

A semi-quantitative FFQ validated in a Mexican population[22] was used to assess diet. This questionnaire included data on frequency of consumption of 116 food items during the previous year. Sweetened beverage consumption was estimated by means of this FFQ. This questionnaire gathered information on the consumption of colas, flavoured sodas, flavoured water with sugar (such as lemon or orange water prepared with artificial flavourings) and diet colas, using a standard serving size of 355 ml. Sweetened beverage intake frequency was divided into four consumption categories: (i) 0 servings/d; (ii) <1 serving/d; (iii) 1–2 servings/d; (iv) >2 servings/d. The energy intake derived from this sweetened beverage intake (kJ/d) was estimated by means of a comprehensive database of food composition[23]. Total energy, dietary fat and alcohol intake were also estimated with this questionnaire. Outlier values in energy intake were eliminated using the standard deviation method[24], and all values below 2510·4 kJ/d and above 29 288 kJ/d were excluded from the analysis.

Metabolic syndrome definition

The National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) defines MS as the presence of three or more of the following components: (i) fasting plasma glucose ≥5·6 mmol/l; (ii) serum TAG

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≥1·7 mmol/l; (iii) systolic and/or diastolic blood pressure ≥130 and/or ≥85 mmHg, respectively; (iv) waist circumference (central obesity) ≥102 cm (40 in) for men and ≥88 cm (35 in) for women; and (v) HDL-C ≤1·03 mmol/l for men and ≤1·29 mmol/l for women[25]. As suggested by Norman, we used a cut-off for plasma glucose level of ≥5.6 mmol/l, lower than that in the guidelines, in order to optimize our ability to assess diabetes risk[26].

Statistical analysis

We performed a descriptive analysis of the main characteristics of interest by sex, testing differences between groups with Fisher's exact tests, Student *t* tests or tests for trend as appropriate.

Prevalences and 95 % confidence intervals of MS and its components were computed across sweetened beverage intake categories, and differences between groups that consumed different amounts of sweetened beverages were assessed with tests for linear trend. These tests entail non-parametric tests for trend across ordered groups, which are an extension of the Wilcoxon rank-sum test. A correction for ties is incorporated into the test.

We evaluated the influence of sweetened beverage consumption on the studied components of MS using multivariate regression models, in which these variables were analysed as continuous. In this case the increments of the components of MS were considered for each additional serving (standard portion of 355 ml) of sweetened beverages.

To estimate the magnitude of the association between specific categories of sweetened beverage consumption and MS and its components (central obesity, dyslipidaemia, hyperglycaemia and elevated blood pressure), we computed adjusted odds ratios and 95 % confidence intervals with multiple logistic regression models.

All *P* values presented are two-tailed; $P < 0{\cdot}05$ was considered statistically significant. All statistical analyses were performed using the STATA statistical software package version 9·2 (StataCorp LP, College Station, TX, USA).

Results

MS as defined by the NCEP ATP III criteria was evident in 26·6 % of our population. On average, participants were middle-aged (mean 39·4 (SD 11·5) years), 71·6 % were women and 56·6 % had BMI ≥ 25 kg/m^2 (mean 26·1 (SD 4·2) kg/m^2). Men consumed more sweetened beverages (1·9 servings/d) than women (1·6 servings/d; $P < 0{\cdot}01$). Men also consumed more energy derived from sweetened beverages (1100·4 kJ/d) than women (907·9 kJ/d; $P < 0{\cdot}001$). However, after adjusting for body weight no differences between men and women were observed. The prevalence of each component of MS was: raised fasting plasma glucose, 14·4 %; raised TAG, 37·5 %; raised blood pressure, 18·3 %; central obesity (higher-than-recommended waist circumference), 38·0 %; and low HDL-C, 76·6 % (Table 1).

Table 1 Distribution of participants according to demographic variables: selected adults (aged 20 to 70 years; *n* 5240) from the baseline assessment of the Health Workers Cohort Study, Mexican states of Morelos and Mexico

Characteristic	Mean	SD
Age (years)	39·4	11·5
BMI (kg/m^2)	26·1	4·2
Weight (kg)	66·8	13·0
Body fat proportion (%fat)	30·4	8·3
Glucose (mmol/l)	4·93	0·58
TAG (mmol/l)	1·71	1·23
HDL-C (mmol/l)	1·02	0·29
WC (cm)	88·7	11·8
DBP (mmHg)	71·7	10·5
SBP (mmHg)	116·3	13·3
SB intake (servings/d)	1·7	1·6
Energy from SB (kJ/d)	963	892
Men	1103**	953
Women	907	860
Energy from SB per kg BW (kJ/kg)	14·7	13·9
Men	14·8††	12·9
Women	14·6	14·2
Energy intake (kJ/d)	9276	3962
Energy intake per kg BW (kJ/kg)	143·4	66·9
Alcohol intake (drinks/d)	2·7	2·2
SFA (% of energy)	9·0	2·6
PUFA (% of energy)	4·3	1·2
Trans fatty acids (% of energy)	0·5	0·3
	%	95 % CI
Low physical activity*	56·0	54·6, 57·3
Current smoking	20·6	19·6, 21·8
Raised glucose†	14·4	13·4, 15·3
Raised TAG‡	37·5	36·2, 38·8
Low HDL-C§	76·6	75·4, 77·7
Central obesity‖	38·0	36·7, 39·3
Raised blood pressure¶	18·3	17·3, 19·3
Metabolic syndrome	26·6	25·4, 27·8

HDL-C, HDL cholesterol; WC, waist circumference; DBP, diastolic blood pressure; SBP, systolic blood pressure; SB, sweetened beverage(s); BW, body weight.
*Subjects with <30 min physical activity/d.
†Fasting plasma glucose ≥ 5·6 mmol/l.
‡Plasma TAG ≥ 1·7 mmol/l.
§Plasma HDL-C ≤ 1·29 mmol/l for women, ≤1·03 mmol/l for men.
‖WC ≥ 88 cm for women, ≥102 cm for men.
¶SBP ≥ 130 mmHg, DBP ≥ 85 mmHg.
Significance of the difference between sexes (Student *t* test): **$P < 0{\cdot}001$, ††$P = 0{\cdot}3$.

Subjects with higher sweetened beverage intake tended to be less physically active, to smoke more and to have higher energy intakes. Intakes of total carbohydrates, sucrose and fructose were higher in subjects with greater sweetened beverage consumption (data not shown). In general, the prevalence of MS and its components increased with higher sweetened beverage intake. Participants in the high intake category had higher plasma glucose than subjects who did not consume sweetened beverages (*P* for trend <0·001). The prevalence of MS was higher among subjects who consumed >2 sweetened beverage servings daily than among non-consumers (*P* for trend <0·001; Table 2).

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Table 2 Characteristics and components of the metabolic syndrome according to sweetened beverage intake in a Mexican population: selected adults (aged 20 to 70 years; *n* 5240) from the baseline assessment of the Health Workers Cohort Study, Mexican states of Morelos and Mexico

	Sweetened beverage intake								
	0 servings/d (*n* 992)		<1 serving/d (*n* 2773)		1–2 servings/d (*n* 794)		>2 servings/d (*n* 681)		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	*P* for linear trend¤
Energy intake (kJ/d)	8556	3782	9636	4368	10 673	3916	13 840	4607	<0·01
Alcohol intake (drinks/d)	3·3	9·7	3·4	13·3	9·2	18·8	9·3	19·4	<0·01
	%	95 % CI	%	95 % CI	%	95 % CI	%	95 % CI	
Low physical activity*	54·1	51·1, 57·3	56·0	53·7, 57·1	58·3	55·1, 62·3	60·0	57·4, 64·2	<0·01
Current smoking	16·0	13·8, 18·1	16·6	15·3, 18·0	25·7	23·2, 29·7	37·5	26·2, 47·7	<0·01
Overweight/obese†	50·4	48·0, 53·0	56·4	53·8, 59·0	60·2	57·2, 63·2	61·2	58·6, 63·9	<0·001
Men	57·9	52·3, 63·7	61·6	56·5, 66·8	67·7	62·6, 72·7	65·8	61·7, 69·9	0·04
Women	48·6	45·9, 51·4	54·7	51·7, 57·7	56·6	52·8, 60·3	58·2	54·7, 61·7	<0·001
Raised glucose‡	10·5	8·6, 12·5	14·7	13·5, 15·9	17·4	14·7, 20·0	22·2	13·0, 31·0	<0·001¤
Men	19·4	13·3, 25·7	22·1	19·6, 24·8	21·2	16·6, 25·7	25·0	10·1, 39·9	0·03
Women	8·8	6·9, 10·8	11·7	10·4, 13·0	14·9	11·7, 18·1	20·0	7·8, 32·2	<0·01
Raised TAG§	30·5	27·7, 33·4	38·5	36·9, 40·1	41·4	38·0, 44·9	43·2	32·2, 54·2	<0·01
Men	50·9	43·1, 58·8	56·8	53·7, 59·9	57·8	52·2, 63·2	61·1	44·4, 77·8	0·03
Women	26·6	23·6, 29·7	31·3	28·1, 34·1	30·0	26·0, 32·8	31·6	28·3, 34·5	0·2
Low HDL-C‖	72·6	69·9, 75·4	76·4	74·9, 77·8	81·6	78·9, 84·3	84·0	75·8, 92·1	<0·001
Men	57·2	49·5, 65·0	62·6	59·6, 65·6	69·8	64·8, 75·0	75·0	60·1, 89·9	<0·01
Women	75·6	72·7, 78·5	82·0	80·5, 83·5	89·2	86·4, 91·9	91·1	82·5, 99·7	<0·001
Central obesity¶	34·8	31·8, 37·7	38·8	37·2, 40·5	38·3	34·9, 41·7	40·7	29·8, 51·7	0·01
Men	11·9	6·9, 17·0	16·4	14·1, 18·7	20·5	16·0, 25·0	27·8	12·4, 43·1	0·03
Women	39·1	35·8, 42·3	48·1	46·1, 50·1	49·8	45·3, 54·3	51·1	35·9, 66·3	<0·001
Raised blood pressure**	14·8	6·9, 22·7	18·0	15·3, 20·7	18·7	17·4, 20·1	17·3	14·9, 19·7	0·03
Men	25·0	10·1, 39·9	28·5	23·5, 33·5	33·6	30·6, 36·6	31·4	24·1, 38·7	0·1
Women	6·6	9·0, 14·2	11·2	8·3, 14·0	12·7	11·3, 14·0	14·6	12·2, 17·1	0·01
Metabolic syndrome	22·3	20·2, 24·3	27·2	24·9, 29·6	27·9	25·1, 30·7	30·1	27·3, 32·3	<0·01
Men	26·7	21·6, 31·9	28·7	24·0, 33·6	31·7	26·7, 36·7	32·0	27·2, 35·1	0·01
Women	21·3	19·0, 23·6	26·7	24·1, 29·5	26·0	22·7, 29·3	29·0	25·7, 32·1	<0·01

HDL-C, HDL cholesterol.
*Subjects with <30 min physical activity/d.
†BMI ≥ 25·0 kg/m^2.
‡Fasting plasma glucose ≥5·6 mmol/l.
§Plasma TAG ≥ 1·7 mmol/l.
‖Plasma HDL-C ≤ 1·29 mmol/l for women, ≤1·03 mmol/l for men.
¶Waist circumference ≥88 cm for women, ≥102 cm for men.
**Systolic blood pressure ≥130 mmHg, diastolic blood pressure ≥85 mmHg.

Table 3 shows the results of a multivariate regression analysis evaluating the effects of sweetened beverage intake on components of MS. After adjusting for demographic characteristics, energy intake and consumption of SFA, PUFA, *trans* fatty acids and alcohol, we found that for each additional serving of sweetened beverage, subjects' glucose and TAG levels were increased. Furthermore, we observed that for each additional serving of sweetened beverages, subjects' HDL-C concentrations decreased. The β coefficients did not change substantially after we controlled for several previously reported risk factors for MS. Age, sex, BMI and weight gain in the past year were positively associated with the five components of MS studied (data not shown).

In the adjusted model, we found that subjects consuming >2 servings of sweetened beverages daily had a greater risk of low HDL-C compared with those who did not consume sweetened beverages. The subjects consuming >2 sweetened beverage servings daily had a greater risk of central obesity compared with non-consumers. The odds for MS were 2·0 for subjects consuming >2 sweetened beverage servings daily compared with those who did not consume sweetened beverages (Table 4).

Discussion

After controlling for known risk factors, we found that sweetened beverage consumption is associated with risk of MS in a Mexican population.

The high prevalence of MS observed in our study population (26·6 %) is similar to that found by other studies using the NCEP ATP III criteria in Mexico (26 %)[5] and the USA (26·7 %)[3]. As expected, we found that MS occurrence was related to increased BMI and proportion of body fat.

Table 3 Multivariate regression model for evaluating the effect of sweetened beverages on components of the metabolic syndrome in a Mexican population: selected adults (aged 20 to 70 years; *n* 5240) from the baseline assessment of the Health Workers Cohort Study, Mexican states of Morelos and Mexico

	Model 1*		Model 2†		Model 3‡	
	Sweetened beverage intake (per serving)		Sweetened beverage intake (per serving)		Sweetened beverage intake (per serving)	
	β	95 % CI	β	95 % CI	β	95 % CI
Glucose (mmol/l)	0·68	0·50, 0·85	0·57	0·39, 0·74	0·56	0·38, 0·74
TAG (mmol/l)	0·74	0·34, 1·13	0·51	0·11, 0·90	0·49	0·08, 0·89
HDL-C (mmol/l)	−0·36	−0·46, −0·27	−0·33	−0·42, −0·23	−0·31	−0·41, −0·22
WC (cm)	14·8	10·9, 18·0	8·1	5·3, 10·9	7·7	4·9, 10·6
DBP (mmHg)	3·3	−0·2, 6·8	5·0	1·7, 8·3	5·6	2·3, 8·9
SBP (mmHg)	7·3	2·9, 10·8	8·60	4·7, 12·7	8·5	4·3, 12·5

HDL-C, HDL cholesterol; WC, waist circumference; DBP, diastolic blood pressure; SBP, systolic blood pressure.
*Model 1 adjusted for age (continuous) and sex.
†Model 2 adjusted for variables in Model 1 plus BMI (two categories: <25·0 and ≥25·0 kg/m^2).
‡Model 3 adjusted for variables in Models 1 and 2 plus weight change within past year (no weight change, weight gain, weight loss), physical activity, total energy intake (kJ/d), alcohol intake (drinks/d), SFA intake (% of energy), PUFA intake (% of energy), *trans* fatty acid intake (% of energy), cigarette smoking (never, past, current) and place of residence.

Table 4 Odds ratio for components of the metabolic syndrome according to sweetened beverage intake: selected adults (aged 20 to 70 years; *n* 5240) from the baseline assessment of the Health Workers Cohort Study, Mexican states of Morelos and Mexico

	Sweetened beverage intake							
	0 servings/d		<1 serving/d		1–2 servings/d		>2 servings/d	
	OR	95 % CI	OR*	95 % CI	OR*	95 % CI	OR*	95 % CI
Raised glucose†	1·0	ref	1·4	1·1, 1·7	1·5	1·2, 2·0	2·1	1·2, 3·8
Raised TAG‡	1·0	ref	1·3	1·2, 1·6	1·5	1·2, 1·8	1·8	1·1, 2·7
Low HDL-C§	1·0	ref	1·4	1·1, 1·6	2·1	1·6, 2·6	2·6	1·3, 4·7
Central obesity‖	1·0	ref	1·4	1·2, 1·7	1·5	1·1, 1·9	2·2	1·2, 3·8
Raised blood pressure¶	1·0	ref	1·9	1·3, 2·7	2·2	1·7, 2·9	2·9	1·3, 6·7
Metabolic syndrome	1·0	ref	1·4	1·2, 1·7	1·7	1·3, 2·0	2·0	1·1, 3·1

HDL-C, HDL cholesterol; ref, referent category.
*Adjusted for age (continuous), sex, BMI (two categories: <25·0 and ≥25·0 kg/m^2), weight change within past year (no weight change, weight gain, weight loss), physical activity, total energy intake (kJ/d), alcohol intake (drinks/d), SFA intake (% of energy), PUFA intake (% of energy), *trans* fatty acid intake (% of energy), cigarette smoking (never, past, current) and place of residence.
†Fasting plasma glucose ≥5·6 mmol/l.
‡Plasma TAG ≥ 1·7 mmol/l.
§Plasma HDL-C ≤ 1·29 mmol/l for women, ≤1·03 mmol/l for men.
‖Waist circumference ≥88 cm for women, ≥102 cm for men.
¶Systolic blood pressure ≥130 mmHg, diastolic blood pressure ≥85 mmHg.

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The MS component most altered was low HDL-C. Low levels of HDL-C are common in the Mexican population, which has one of the highest rates of low HDL-C worldwide[27]. In our study the prevalence of low HDL-C was 76·6 %, and it was more prevalent in women (81·6 %) than in men (63·9 %).

We observed significant differences in the amount of sweetened beverage consumption by sex: men consumed about 15 % more sweetened beverages than women (1·9 *v.* 1·6 servings/d). Our study population, particularly the men, derived a considerable proportion of their energy intake from sweetened beverages (10·5 %). These results are consistent with previous national reports of daily energy derived from sweetened beverage intake in Mexican adult males, who derived ~10 % of their daily energy intake from sweetened beverages[14].

Study participants with high sweetened beverage consumption also had relatively high levels of energy intake from other foods, indicating that sweetened beverages may induce hunger and food intake. Nevertheless, this might be the result of a possible report bias and cannot be interpreted as a causal relationship. In addition, experimental data on sweetened beverage consumption and food intake have not provided support for this hypothesis[28,29].

Our findings suggest that subjects who consume >2 servings of sweetened beverages daily are at 2·0 times greater risk for MS than those who do not consume sweetened beverages. This finding is in accordance with a previous study to evaluate the relationship between soft drink intake and risk of MS, which reported that individuals consuming ≥1 soft drink/d had a higher prevalence of MS (OR = 1·48; 95 % CI 1·30, 1·69) than those consuming <1 drink/d[30].

Our analyses showed that each serving of sweetened beverages is associated with an increase in plasma glucose of 0·56 mmol/l ($P < 0·001$). This means that for subjects who do not consume sweetened beverages, increasing their consumption to a daily serving would elevate their plasma glucose from 5·09 to 5·66 mmol/l, a level that would be classified by our criteria as raised plasma glucose. We found that subjects consuming >2 servings of sweetened beverages daily are at 2·1 times greater risk of raised fasting plasma glucose compared with non-consumers. The ability of sweetened beverage consumption to raise fasting plasma glucose has been attributed the high amount of rapidly absorbable carbohydrates that these beverages provide[8,31,32]. Sweetened beverages therefore increase consumers' dietary glycaemic index, and diets with a high glycaemic index have been found to be a risk factor for diabetes in some cohort studies[8,33–36].

Though a similar biological pathway, sweetened beverages might also increase the risk of raised plasma TAG and low plasma HDL-C. We observed that subjects consuming >2 servings of sweetened beverage daily are at 1·8 times greater risk of raised plasma TAG and 2·6 times greater risk of low plasma HDL-C compared with non-consumers. These results are consistent with those of a previous study, which reported that consumption of ≥1 soft drink/d was associated with hypertriacylglycerolaemia (OR = 1·25; 95 % CI 1·04, 1·51) and low HDL-C (OR = 1·32; 95 % CI 1·06, 1·64)[30]. Our analyses showed that each serving of sweetened beverage is associated with a 0·49 mmol/l increase in plasma TAG concentrations ($P < 0·001$). This means that for subjects who do not consume sweetened beverages, increasing their consumption by a serving daily would elevate their plasma TAG concentrations from 1·67 to 2·15 mmol/l, classifying them as having raised plasma TAG according to our criteria. Since an experimental study in adults has shown that consumption of fructose-sweetened beverages rapidly increases and peaks plasma TAG concentrations[37], the large amounts of free fructose and fructose combined in sucrose derived from sweetened beverages may be a precursor to hepatic lipogenesis, since they provide a relatively unregulated source of carbon[38,39].

We also found that increased sweetened beverage consumption is associated with increases in systolic and diastolic blood pressure. Each serving of sweetened beverage was associated with an increase of 8·5 mmHg in systolic blood pressure ($P < 0·001$) and an increase of 5·6 mmHg in diastolic blood pressure ($P < 0·01$). Experiments have shown that high-fructose sweetened beverages induce hypertension in animals[40,41] and that adults consuming sucrose-sweetened beverages exhibit an increase in both systolic and diastolic blood pressure[42]. The mechanism by which fructose-sweetened beverages induce hypertension is not well understood, but it may involve uric acid production, hyperinsulinaemia, aldehyde formation and/or altered vascular reactivity[12,40].

The present study's cross-sectional design made it difficult to examine the potential causal relationship between sweetened beverage intake and MS occurrence, since the temporal relationship of these events could not be established. Further, this cohort contained adults from a specific segment of the Mexican population: working, seemingly healthy individuals. While these adults cannot be considered representative of the Mexican adult population as a whole, they may be considered representative of middle- to low-income adults residing in the urban areas of central Mexico. Even in light of these limitations, our results provide important information about the association between sweetened beverage intake and risk of MS in our population. However, MS is a multifactorial disorder, and diet plays an important role in its development. Dietary intake can be considered in terms of particular dietary patterns. This perspective accounts for the effect of dietary patterns as a whole and thus may provide insight beyond the effects described for single nutrients or foods[43,44].

Our findings support the need to develop public health strategies that will discourage sweetened beverage consumption in the Mexican population. All sectors of

society, including private and governmental institutions, the health-care system and especially nutrition professionals, have important roles to play in reducing the population's sweetened beverage consumption. Nutrition and health professionals should educate our population about the potential adverse effects of excessive sweetened beverage consumption. These professionals could also suggest strategies for limiting sweetened beverage intake, including limiting the availability of sweetened beverages in homes and workplaces and recommending smaller portion sizes. They could also promote healthier alternatives, particularly non-caloric beverages like water.

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References

1. Raven GM (1988) Banting Lecture: Role of insulin resistance in human disease. *Diabetes* **37**, 1595–1607.
2. Alberti KG, Zimmet P & Shaw J (2005) The metabolic syndrome: a new worldwide definition. *Lancet* **366**, 1059–1062.
3. Ford ES, Giles WH & Dietz WH (2002) Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *JAMA* **287**, 356–359.
4. Eckel RH, Scott MG & Zimmet P (2005) The metabolic syndrome. *Lancet* **365**, 1415–1428.
5. Aguilar-Salinas CA, Rojas R, Gómez-Pérez FJ *et al.* (2004) High prevalence of metabolic syndrome in Mexico. *Arch Med Res* **35**, 76–81.
6. Ford ES (2005) Risk for all-cause mortality, cardiovascular disease, and diabetes associated with the metabolic syndrome. *Diabetes Care* **28**, 1769–1778.
7. Vartanian LR, Schartz MB & Brownell KD (2007) Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *Am J Public Health* **97**, 667–675.
8. Schulze MB, Manson JE, Ludwig DS *et al.* (2004) Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. *JAMA* **292**, 927–934.
9. Malik VS, Schulze MB & Hu FB (2006) Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr* **84**, 274–288.
10. Bray GA, Nielsen SJ & Popkin BM (2004) Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *Am J Clin Nutr* **79**, 537–543.
11. Denova-Gutiérrez E, Jiménez-Aguilar A, Halley-Castillo E *et al.* (2008) Association between sweetened beverage consumption and obesity, proportion of body fat and body fat distribution in Mexican adolescents. *Ann Nutr Metab* **53**, 245–251.
12. Johnson RJ & Segal MS (2007) Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease, and cardiovascular disease. *Am J Clin Nutr* **86**, 899–906.
13. Yoo S, Nicklas T, Baranowski T *et al.* (2004) Comparison of dietary intakes associated with metabolic syndrome risk factors in young adults: the Bogalusa Heart Study. *Am J Clin Nutr* **80**, 841–848.
14. Rivera JA, Muñoz-Hernández O, Rosas-Peralta M *et al.* (2008) Consumo de bebidas para una vida saludable: recomendaciones para la población mexicana. *Salud Publica Mex* **50**, 173–195.
15. Salmeron-Castro J & Arillo-Santillán E (2002) Tabaquismo en profesionales de la salud del Instituto Mexicano del Seguro Social, Morelos. *Salud Publica Mex* **44**, Suppl. 1, S67–S75.
16. López-Caudana AE, Téllez-Rojo Solís MM, Hernández-Avila M *et al.* (2005) Predictors of bone mineral density in female workers in Morelos State, Mexico. *Arch Med Res* **35**, 172–180.
17. Tamayo J, Lazcano-Ponce E, Muñoz MC *et al.* (2009) Reference values for areal bone mineral density among a healthy Mexican population. *Salud Publica Mex* **51**, Suppl. 1, S56–S83.
18. Ainsworth BE, Haskell WL, Whitt MC *et al.* (2000) Compendium of physical activities: an update of activity codes and MET intensities. *Med Sci Sports Exerc* **32**, Suppl. 9, S498–S516.
19. Ford ES & Liu S (2001) Glycemic index and serum high-density lipoprotein cholesterol concentration among US adults. *Arch Intern Med* **161**, 572–576.
20. Tate JR, Rifai N, Berg K *et al.* (1999) International Federation of Clinical Chemistry and Laboratory Medicine (IFCC) Standardization Project for the Measurement of Lipoprotein(a). Phase 2: selection and properties of a proposed secondary reference material for lipoprotein(a). *Clin Chem Lab Med* **37**, 949–958.
21. Halley Castillo E, Borges G, Talavera JO *et al.* (2007) Body mass index and the prevalence of metabolic syndrome among children and adolescents in two Mexican populations. *J Adolesc Health* **40**, 521–526.
22. Hernández-Avila M, Romieu I, Parra S *et al.* (1998) Validity and reproducibility of a food frequency questionnaire to assess dietary intake of women living in Mexico City. *Salud Publica Mex* **40**, 133–140.
23. Hernández-Avila M, Resoles M, Parra S *et al.* (2000) *Sistema de evaluación de hábitos nutricionales y consumo de nutrimentos (SNUT)*. Cuernavaca, Mexico: INSP.
24. Rosner B (1983) Percentage points for a generalized ESD many-outlier procedure. *Technometrics* **25**, 165–172.
25. National Cholesterol Education Program, National Heart, Lung and Blood Institute & National Institutes of Health (2001) *Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on: Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III), Executive Summary. NIH Publication* no. 10-3670, p. 16. Washington, DC: US Department of Health and Human Services.

26. Norman EL (2004) Diabetes mellitus. An update on diagnostic criteria for diabetes and the metabolic syndrome. *Rev Cardiovasc Med* **5**, 182–185.
27. Aguilar-Salinas CA, Olaiz G, Valles V *et al.* (2001) High prevalence of low HDL cholesterol concentrations and mixed hyperlipidemia in a Mexican nationwide survey. *J Lipid Res* **42**, 1298–1307.
28. Almiron-Roig E & Drewnowski A (2003) Hunger, thirst, and energy intakes following consumption of caloric beverages. *Physiol Behav* **79**, 767–773.
29. DiMeglio DP & Mattes RD (2000) Liquid versus solid carbohydrates: effects on food intake and body weight. *Int J Obes Relat Metab Disord* **24**, 794–800.
30. Dhingra R, Sullivan L, Jacques PF *et al.* (2007) Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. *Circulation* **116**, 480–488.
31. Akgun S & Ertel NH (1985) The effects of sucrose, fructose, and high-fructose corn syrup meals on plasma glucose and insulin in non-insulin-dependent diabetic subjects. *Diabetes Care* **8**, 279–283.
32. Drewnowski A & Bellisle F (2007) Liquid calories, sugar and body weight. *Am J Clin Nutr* **85**, 651–661.
33. Willett W, Manson J & Liu S (2002) Glycemic index, glycemic load, and risk of type 2 diabetes. *Am J Clin Nutr* **76**, 274S–280S.
34. Schulze MB, Liu S, Rimm EB *et al.* (2004) Glycemic index, glycemic load, and dietary fiber intake and incidence of type 2 diabetes in younger an middle-aged women. *Am J Clin Nutr* **80**, 348–356.
35. Salmerón J, Ascherio A, Rimm EB *et al.* (1997) Dietary fiber, glycemic load, and risk of NIDDM in men. *Diabetes Care* **20**, 545–550.
36. Salmerón J, Manson JE, Stampfer JM *et al.* (1997) Dietary fiber, glycemic load and risk of non-insulin-dependent diabetes mellitus in women. *JAMA* **277**, 472–477.
37. Teff KL, Elliott SS, Tschöp M *et al.* (2004) Dietary fructose reduces circulating insulin and leptin, attenuates postprandial suppression of ghrelin, and increases triglycerides in women. *J Clin Endrocrinol Metab* **89**, 2963–2972.
38. Petersen KF, Laurent D, Yu C *et al.* (2001) Stimulating effects of low-dose fructose on insulin-stimulated hepatic glycogen synthesis in humans. *Diabetes* **50**, 1263–1268.
39. Basciano H, Federico L & Adeli K (2005) Fructose, insulin resistance, and metabolic dyslipidemia. *Nutr Metab (Lond)* **2**, 5.
40. Elliot S, Keim N, Stern J *et al.* (2002) Fructose, weight gain, and the insulin resistance syndrome. *Am J Clin Nutr* **76**, 911–922.
41. Sánchez-Lozada L, Tapia E, Jímenez A *et al.* (2007) Fructose-induced metabolic syndrome is associated with glomerular hypertension and renal microvascular damage in rats. *Am J Physiol Renal Physiol* **292**, F423–F429.
42. Raben A, Vasilaras TH, Moller AC *et al.* (2002) Sucrose compared with artificial sweeteners: different effects on *ad libitum* food intake, and body weight after 10 weeks of supplementation in overweight subjects. *Am J Clin Nutr* **76**, 721–729.
43. Esmaillzadeh A, Kimiagar M, Mehrabi Y *et al.* (2007) Dietary patterns, insulin resistance, and prevalence of the metabolic syndrome in women. *Am J Clin Nutr* **85**, 910–918.
44. Lutsey PL, Steffen LM & Stevens J (2008) Dietary intake and development of the metabolic syndrome. The Atherosclerosis Risk in Communities study. *Circulation* **117**, 754–761.