Carbohydrate intake and HDL in a multiethnic population[1–3]

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ABSTRACT

Background: Ethnic differences in serum lipids are not explained by genetics, central adiposity, lifestyle, or diet, possibly because dietary carbohydrate has not been considered.

Objective: The aim was to evaluate the relation between carbohydrate intake and HDL and triacylglycerol concentrations in a multiethnic population.

Design: We conducted a population-based cross-sectional study of 619 Canadians of Aboriginal, South Asian, Chinese, and European origin with no previously diagnosed medical conditions. Energy-adjusted carbohydrate intake was measured by a validated food-frequency questionnaire.

Results: South Asians consumed the most carbohydrate, followed by European, Aboriginal, and Chinese persons. Mean (95% CI) HDL concentrations in the lowest and highest categories of carbohydrate intake after adjustment for age, sex, ethnicity, physical activity, smoking, the waist-to-hip ratio, body mass index, alcohol intake, and intakes of total energy, protein, and fiber were 1.21 mmol/L (1.16, 1.27 mmol/L) and 1.08 mmol/L (1.02, 1.13 mmol/L), respectively, and HDL cholesterol was significantly ($P < 0.01$) higher in the lowest tertile of carbohydrate intake than in the highest tertile. High carbohydrate intake was associated with higher fasting triacylglycerols ($P = 0.04$); the adjusted mean fasting triacylglycerol concentrations in the lowest and highest categories of carbohydrate intake were 1.43 mmol/L (1.28, 1.60 mmol/L) and 1.71 mmol/L (1.57, 1.87 mmol/L), respectively. Fewer servings of sugar-containing soft drinks, juices, and snacks were associated with higher HDL (P for trend = 0.02); the multivariate-adjusted mean HDL in the lowest and highest categories of carbohydrate intake was 1.22 mmol/L (1.17, 1.27 mmol/L) and 1.11 mmol/L (1.06, 1.26 mmol/L), respectively.

Conclusions: Differences in HDL and triacylglycerols observed in different ethnic groups may be due in part to carbohydrate intake. Reducing the frequency of intake of sugar-containing soft drinks, juices, and snacks may be beneficial. *Am J Clin Nutr* 2007;85:225–30.

KEY WORDS Dietary carbohydrates, lipoproteins, HDL, triacylglycerols, ethnic groups, Canada

INTRODUCTION

Low HDL-cholesterol, high triacylglycerol, and elevated LDL-cholesterol concentrations increase cardiovascular disease risk (1, 2), and the prevalence of these abnormalities varies by ethnicity (3). Genetic factors alone do not explain the variation in serum lipids, and it is probable that lifestyle factors (ie, physical activity, diet, smoking, and alcohol intake) have a greater influence on lipid concentrations (4).

In previous investigations, ethnic differences in serum lipids were not explained by genetic factors, central adiposity, lifestyle, or diet (4). Part of the reason may be that most dietary studies comparing ethnic differences in serum lipids were focused on fat consumption (5). Although fat intake is important in relation to serum lipids, if carbohydrates are consumed in place of fats, LDL- and HDL-cholesterol concentrations decrease but triacylglycerols concurrently increase (6). Previously, we reported that protein intake was inversely related to central adiposity (7), and central adiposity is known to adversely affect serum lipids (8). The present analysis evaluated the effect of carbohydrate intake on serum lipids in a multiethnic population.

SUBJECTS AND METHODS

Study population

The study population included Canadians of Aboriginal, South Asian, Chinese, and European origin who took part in 2 concurrent cross-sectional studies of cardiovascular disease risk factors between 1996 and 2000 in the Study of Health Assessment and Risk in Ethnic groups (SHARE) (9) and SHARE-Aboriginal People (SHARE-AP) (10). We randomly selected Canadians of South Asian, Chinese, and European background from the cities of Toronto, Hamilton, and Edmonton, Canada, based on unique last names using a previously validated method (11, 12); we also selected Aboriginal Persons from the Six Nations SHARE-AP master list. The participants were 35–75 y of age and had lived in Canada for ≥5 y at the time of entry into the

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study. We excluded participants with active cancer or other serious chronic diseases, such as renal or liver failure, and Canadians of South Asian and Chinese backgrounds who were born in Canada. The McMaster University Research Ethics Committee and the Six Nations Band Council approved the study design, data collection, and analysis.

Data collection

After obtaining informed consent, we collected and recorded each participant's lifestyle characteristics and medical history using standardized questionnaires and height, weight, waist, and hip measurements using a standardized protocol (9, 13). We evaluated the participants' diets using validated, culture-specific, self-administered, quantitative food-frequency questionnaires (FFQs) (14). In a comparison of dietary intake assessed by the FFQs with 7–14 d diet records to assess the validity for South Asians, Chinese, and Europeans, the energy-adjusted, deattenuated correlation coefficients were 0.45–0.57 for protein, 0.17–0.62 for total fats, 0.31–0.60 for carbohydrates, and 0.63–0.70 for total fiber (14). Similar values were found in the validation of the FFQ for Aboriginal Peoples (LE Kelemen, unpublished observations).

Nutrient analysis

We excluded participants with a history of angina, cancer, diabetes, cardiovascular disease, hypertension, hypercholesterolemia, or kidney or liver disease and those who reported implausible dietary intakes (<800 or >4500 kcal/d) or who reported that they had changed their usual diet. On the FFQs, the participants reported how often, on average, they had consumed selected foods in the previous year. We calculated nutrient intakes by multiplying the average nutrient content of a particular food portion by the number of times it was consumed. We determined nutrient content by analyzing diet records using the Food Processor nutrient analysis software (version 6.11; ESHA, Salem, OR), which incorporated the 1991 Canadian Nutrient File and US Department of Agriculture databases (14). We also calculated the glycemic index (15) and glycemic load (16) for each participant.

We log-transformed all nutrients and then adjusted for total energy by linear regression with the nutrient as the outcome and total energy intake as the predictor (17). The residuals from this model were added to the expected value of the nutrient at average energy intake. Energy-adjusted nutrients calculated in this manner can be interpreted as the composition of the particular nutrient in the diet independent of total energy intake (17). Log-transformed nutrients were exponentiated for ease of interpretation.

Foods

We evaluated the relation between carbohydrate-rich foods and serum lipids because dietary recommendations are more easily understood for foods rather than nutrients. We correlated foods on the FFQs with carbohydrate intake to form groups of foods associated with carbohydrate intake in this population. Based on the correlations, we grouped white bread (including rolls, scones, pancakes, chapati, paratha, naan, dosa, and puri), potatoes, and rice as one group of foods and sugar-containing soft drinks, fruit juices, and snacks (including crackers, cookies, chips, chocolate, candy, pakora, bhajia, papad, and papri) as another. For each of the groups, we summed the average number of daily servings of foods that were reported on the FFQ and calculated tertiles of intake.

Serum lipids

All aliquots were frozen at −70 °C and transferred on dry ice to the core laboratory in Hamilton, Canada for central analysis with the use of standard methodology. All blood analyses were conducted in core laboratories without knowledge of the participant's ethnicity, clinical history, and risk factor status. Total cholesterol and triacylglycerols were measured by enzymatic methods (18–20), and LDL cholesterol was calculated (21). HDL cholesterol was measured after precipitating the VLDL and LDL cholesterol with phosphotungstic acid and magnesium chloride.

Statistical analyses

To characterize the participants by carbohydrate intake, we calculated tertiles of intake based on the total sample and then compared persons across those categories with respect to personal, lifestyle, and dietary factors. To evaluate the relation between serum lipids and carbohydrate intake, we used analysis of covariance (ANCOVA) (22). In the multivariate model, serum lipids were the outcome and tertiles of carbohydrate intake the predictor, with adjustment for age (in y), intakes of total energy (kcal), height (cm), physical activity score, waist-to-hip ratio, body mass index (BMI, in kg/m^2; continuous variable), sex (dichotomous), smoking (never, past, or current), alcohol intake (never or <1 drink/mo, 1 drink/mo to 5 drinks/wk, or >5 times/wk), and ethnicity (Aboriginal, South Asian, Chinese, or European). We calculated the physical activity index by summing ordinal categories of intensity of physical activity levels at work, sports, and leisure time (7). We included variables in the multivariate model that may be related to both serum lipid levels and carbohydrate intake based on pervious studies. To evaluate possible confounding by other dietary variables, we further adjusted the analyses separately for intakes of protein, total fat, polyunsaturated fat, monounsaturated fat, *trans* fat, sugar, total fiber, soluble fiber, insoluble fiber, glycemic index, and glycemic load.

We evaluated the relation between carbohydrate intake and HDL in subgroups by age (<45 or ≥45 y), sex, BMI (< 25 or ≥ 25), smoking (never, past, or current), alcohol intake (never or <1 drink/mo or >1drink/mo), and physical activity (less than median of physical activity index, median, or more). To assess interaction, multiplicative terms we computed between the stratifying categories and each nutrient (eg, sex × protein intake) and evaluated in ANCOVA models by the Wald test after adjustment for confounders.

We further examined the relation between carbohydrate intake and HDL using the nutrient residual model (17). We included energy-adjusted carbohydrate intake divided by 100, total fat, and total energy with adjustment for age (in y), height (cm), physical activity score, waist-to-hip ratio, BMI (continuous variable), sex (dichotomous), smoking (never, past, or current), alcohol intake (never or <1 drink/mo, 1 drink/mo to 5 drink/wk, or >5 drinks/wk), and ethnicity (Aboriginal, South Asian, Chinese, or European). The β coefficient for carbohydrate in this model could be interpreted as the change in HDL resulting from a 100 g/d increase in carbohydrate intake instead of an equivalent amount of protein (17). To evaluate the relation between ethnicity and HDL without accounting for dietary factors, we reran the model excluding the dietary variables.

In the assessment of foods, we first calculated partial correlations with adjustment for age and sex between carbohydrate intake and 2 food groups: the first containing white bread, rice, and potatoes and the second consisting of sugar-containing soft drinks and juices and snacks. We then related tertiles of intakes of these food groups separately in ANCOVA models with HDL as the outcome after adjustment for age (in y), total energy (kcal), height (cm), physical activity score, waist-to-hip ratio (continuous variable), sex (dichotomous), smoking (never, past, or current), alcohol intake (never or <1 drink/mo, 1 drink/mo to 5 drinks/wk, or >5 drinks/wk), and ethnicity (Aboriginal, South Asian, Chinese, or European). We repeated these analyses with additional adjustment for protein and fiber intakes. We used SAS version 9 (SAS Institute, Cary, NC) for the analyses.

RESULTS

Of the 1286 participants who completed all aspects of the clinic assessment, we excluded 560 persons because of prevalent health conditions or reports of altered diet and 107 persons because of implausible dietary data (those reporting total energy intake of <800 or >4500 kcal/d), leaving 619 participants for the present analysis (n = 92 Aboriginal Peoples, 163 South Asians, 168 Chinese, and 186 Europeans). We excluded people with diagnosed comorbidities because they may have altered their diet or lifestyle as a consequence of their medical condition. Persons who did not report comorbidities had an average (±SD) energy intake of 1957 ± 710 kcal/d, a mean BMI of 26 ± 5.0, and a mean physical activity score of 7.7 ± 2.7 (the higher the score the more physically active); in contrast, those who reported comorbidity had a mean energy intake of 1919 ± 765 kcal/d, a mean BMI of 28 ± 5.5, and a mean physical activity score of 7.2 ± 3.7.

South Asians consumed the most carbohydrate, followed by Europeans, Aboriginal Peoples, and Chinese, who consumed the least (**Table 1**). Persons who consumed more carbohydrate were less likely to smoke and to consume >5 drinks of alcohol/d, protein, and fats than were those who ate less carbohydrate (Table 1). Persons who ate more carbohydrate were more physically active and ate more fiber and sugar than did those who ate less carbohydrate (Table 1).

HDL cholesterol was significantly higher ($P < 0.01$) in the lowest tertile of carbohydrate intake than in the highest tertile; mean (95% CI) HDL concentrations in the lowest and highest categories of carbohydrate intake after adjustment for age, sex, ethnicity, physical activity, smoking, the waist-to-hip ratio, BMI, alcohol intake, and intakes of total energy, protein, and fiber were 1.21 mmol/L (1.16, 1.27 mmol/L) and 1.08 mmol/L (1.02, 1.13 mmol/L), respectively (**Table 2**). High carbohydrate intake was associated with a lower HDL-to-LDL ratio ($P = 0.04$); the adjusted mean HDL-to-LDL ratios of the lowest and the highest category of carbohydrate intake were 0.45 (0.41, 0.48) and 0.37 (0.33, 0.40), respectively. High carbohydrate intake was also associated with higher fasting triacylglycerols ($P = 0.04$); the adjusted mean fasting triacylglycerol concentrations in the lowest and highest categories of carbohydrate intake were 1.43 mmol/L (1.28, 1.60 mmol/L) and 1.71 mmol/L (1.57, 1.87 mmol/L), respectively. These associations remained significant after adjustment for glycemic index, glycemic load, saturated fat, *trans*-fat, and total fat intakes. No significant relation was observed between carbohydrate intake and LDL and fasting free fatty acids (Table 2).

TABLE 1
Characteristics of the participants by carbohydrate intake[1]

	Carbohydrate intake		
	Tertile 1	Tertile 2	Tertile 3
Age (y)	47.4 ± 0.6[2]	47.8 ± 0.7	48.2 ± 0.6
Men [*n* (%)]	95 (32.7)	93 (32.0)	103 (35.4)
Ethnicity[3]			
Aboriginal [*n* (%)]	42 (45.7)	29 (31.5)	21 (22.8)
South Asian [*n* (%)]	10 (5.8)	48 (27.8)	115 (66.5)
Chinese [*n* (%)]	101 (60.1)	49 (29.2)	18 (10.7)
European [*n* (%)]	51 (27.4)	76 (40.9)	59 (31.7)
Smoking status[3]			
Never smoker [*n* (%)]	113 (55.4)	127 (62.9)	159 (74.7)
Past smoker [*n* (%)]	46 (22.6)	41 (20.3)	34 (16.0)
Current smoker [*n* (%)]	45 (22.1)	34 (16.8)	20 (9.4)
Drinking status[3]			
Never drinker [*n* (%)]	82 (40.2)	77 (38.1)	103 (48.4)
Moderate drinker [*n* (%)]	94 (46.1)	98 (48.5)	97 (45.5)
Heavy drinker [*n* (%)]	28 (13.7)	27 (33.9)	13 (6.1)
BMI (kg/m^2)	26.0 ± 0.4	26.3 ± 0.3	26.3 ± 0.3
Waist (cm)	87.8 ± 1.1	88.9 ± 1.0	88.8 ± 0.9
Waist-to-hip ratio	0.865 ± 0.016	0.866 ± 0.016	0.868 ± 0.016
Physical activity index[3]	7.5 ± 0.1	7.7 ± 0.1	8.1 ± 0.1
Total energy intake (kcal/d)	2039.9 ± 55.8	1871.5 ± 45.7	1959.7 ± 46.0
Protein (g/d)[3]	97.7 ± 1.4	81.0 ± 0.9	69.2 ± 0.7
Total fat (g/d)[3]	76.3 ± 0.8	64.5 ± 0.5	53.6 ± 0.5
Saturated fat (g/d)[3]	23.2 ± 0.5	21.3 ± 0.4	17.3 ± 0.3
Polyunsaturated fat (g/d)[3]	14.8 ± 0.3	11.3 ± 0.2	10.1 ± 0.2
Monounsaturated fat (g/d)[3]	29.3 ± 0.3	24.2 ± 0.3	19.6 ± 0.2
trans Fat (g/d)[3]	0.47 ± 0.05	0.47 ± 0.03	0.36 ± 0.03
Total fiber (g/d)[3]	13.6 ± 0.3	15.8 ± 0.3	20.6 ± 0.4
Sugar (g/d)[3]	6.8 ± 0.2	8.4 ± 0.3	10.7 ± 0.4

[1] n = 619: 92 Aboriginal Peoples, 163 South Asians, 168 Chinese, and 186 Europeans. The medians (ranges) of carbohydrate intake (in g/d) were 222 (121–251) for tertile 1, 268 (251–289) for tertile 2, and 310 (289–380) for tertile 3.
[2] $\bar{x}$ ± SEM (all such values).
[3] Overall $P < 0.05$ (Cochran-Mantel-Haenszel test for categorical variables and F statistic for continuous data).

Glycemic index, glycemic load, and intakes of protein, fat (total and subtypes), and fiber were not significantly related to HDL cholesterol or fasting triacylglycerols in these persons (data not shown). We did not detect any significant change in the relation between carbohydrate intake and HDL in subgroups by age, sex, smoking, alcohol intake, BMI, and physical activity. We observed no relation between carbohydrate intake and HDL stratified by ethnicity because there were few South Asians in the low carbohydrate intake group and few Chinese in the high carbohydrate intake group.

In the multivariate nutrient residual model (**Table 3**), every 100-g/d increment of carbohydrate (approximately the difference between the top and bottom tertiles) was associated with 0.15-mmol/L less HDL after adjustment for intakes of fat, total energy, and alcohol and age, sex, smoking, physical activity, height, the waist-to-hip ratio, BMI, and ethnicity. Ethnicity remained significantly related to HDL after accounting for lifestyle

TABLE 2
Adjusted mean (95% CI) lipid concentration by tertiles of carbohydrate intake[1]

	Carbohydrate intake			
	Tertile 1	Tertile 2	Tertile 3	P^2
HDL (mmol/L)	1.21 (1.16, 1.27)	1.22 (1.17, 1.26)	1.08 (1.02, 1.13)	<0.01
LDL (mmol/L)	3.07 (2.95, 3.20)	3.06 (2.97, 3.17)	3.18 (3.06, 3.31)	0.37
HDL:LDL	0.45 (0.41, 0.48)	0.44 (0.41, 0.47)	0.37 (0.33, 0.40)	<0.01
Triacylglycerols (mmol/L)	1.43 (1.28, 1.60)	1.46 (1.34, 1.60)	1.71 (1.57, 1.87)	0.04
Free fatty acids (μmol/L)	512.73 (478.24, 549.55)	481.18 (453.83, 510.87)	476.00 (442.18, 512.15)	0.35

[1] $n = 619$ [291 M (47%), 328 F (53%)]: 92 Aboriginal Peoples, 163 South Asians, 168 Chinese, and 186 Europeans. The medians (ranges) of carbohydrate intake (in g/d) were 222 (121–251) for tertile 1, 268 (251–289) for tertile 2, and 310 (289–380) for tertile 3. Values were adjusted for age, total energy intake (kcal/d), protein (g/d), fiber (g/d), waist-to-hip ratio, BMI (kg/m^2), height (cm), and physical activity (index) as continuous variables and for sex, smoking (never, past, or current smoker), alcohol intake (never or <1 drink/mo, ≤5 drinks/wk, or >5 drinks/wk), and ethnicity (Aboriginal, South Asian, Chinese, or European).
[2] F statistic from ANCOVA.

factors (Table 3). When dietary variables were excluded from this model, the relation between ethnicity and HDL was stronger. HDL was, on average, 0.04 mmol/L lower in South Asians, and 0.06 mmol/L higher in Chinese (P for differences in HDL by ethncity = 0.02). A similar trend was observed for the HDL-to-LDL ratio and triacylglycerols. Higher waist-to-hip ratio, higher BMI, and male sex were associated with lower HDL, whereas age and more physical activity were associated with higher HDL in this analysis (Table 3). LDL, fasting triacylglycerols, and fasting free fatty acids were not significantly different by ethnicity.

TABLE 3
Parameter estimates from a multivariate nutrient residual model evaluating carbohydrate intake in relation to HDL[1]

	Parameter estimate (β)	SE	P^2
Intercept	3.0382	0.4989	<0.001
Energy-adjusted carbohydrate intake (g/d)[3]	−0.1478	0.0638	0.02
Energy-adjusted fat intake (g/d)	−0.0021	0.0019	0.27
Total energy intake (kcal/d)	−0.000025	0.000019	0.21
Age (y)	0.0052	0.0015	<0.001
Sex			0.001
Women (reference)	—	—	
Men	−0.1447	0.0443	
Smoking status			0.66
Never smoker (reference)	—	—	
Past smoker	0.0863	0.0372	
Current smoker	0.0204	0.0423	
Alcohol use			0.22
Nondrinker (reference)	—	—	
≤5 drinks/wk	0.0304	0.0306	
>5 drinks/wk	0.0959	0.0552	
Physical activity index	0.0169	0.0082	0.04
Waist-to-hip ratio	−0.6085	0.2175	0.01
BMI (kg/m^2)	−0.0240	0.0032	<.0001
Height (cm)	−0.0028	0.0021	0.19
Ethnicity			0.02
European (reference)	—	—	
South Asian	−0.0431	0.0455	
Chinese	0.0623	0.0483	
Aboriginal People	0.0169	0.0082	

[1] $n = 619$: $n = 92$ Aboriginal Peoples, 163 South Asians, 168 Chinese, and 186 Europeans.
[2] Wald test for continuous and binary variables; F statistic for variables with >2 categories.
[3] β coefficient represents the change in HDL for 100 g/d intake of carbohydrate instead of an equivalent amount of protein after adjustment for fat intake and other potential explanatory factors.

Carbohydrate intake was correlated with servings of white bread, rice, and potatoes and with sugar-containing soft drinks, juices, and snacks (**Table 4**). However, there was no significant correlation between servings of white bread, rice, and potatoes and intake of sugar-containing soft drinks, juices, and snacks (Table 4). Fewer servings of sugar-containing soft drinks, juices, and snacks were associated with higher HDL (P for trend = 0.02); the multivariate-adjusted mean HDL in the lowest and highest categories of carbohydrate intake were 1.22 mmol/L (1.17, 1.27 mmol/L) and 1.11 mmol/L (1.06, 1.26 mmol/L). Total energy intake was not significantly related to HDL in this model. No significant relation ($P = 0.41$) was observed between servings of white bread, rice, and potatoes and HDL; the multivariate-adjusted mean HDL of the extreme categories of intake were 1.15 (1.10, 1.20) and 1.20 (1.15, 1.25) mmol/L.

DISCUSSION

Higher carbohydrate was associated with lower HDL cholesterol and higher triacylglycerols after accounting for personal and lifestyle characteristics in a multiethnic population. Our findings are consistent with those from other studies. HDL cholesterol declined by <0.016 mmol/L and triacylglycerols rose by 0.026 mmol/L for every 1% of total energy from polyunsaturated fats that was replaced by carbohydrates in a meta-analysis of 60 controlled trials (6). With moderate total fat reduction (8.2% after 6 y), there was no effect on HDL and triacylglycerols (23). But when dietary fat content was more extreme, the average difference in HDL was 0.12 mmol/L and in triacylglycerols was −0.25

TABLE 4
Partial correlation coefficients adjusted for age and sex

	Carbohdrate intake	Sugar-containing soft drinks, juices, and snacks
White bread, rice, and potatoes	0.33[1]	0.05
Sugar-containing soft drinks, juices, and snacks	0.26[1]	—

[1] $P < 0.05$.

mmol/L for the high-fat diet group (24). Our results were consistent with results from studies that evaluated HDL and either physical activity (25) or alcohol intake (26). Higher HDL is ascribed to longevity (27); the positive association we observed with age may be due to survival of these persons. Lower HDL in South Asians is ascribed to insulin resistance, but its origins are not known (28) and it is not explained by genetics (5) or fat intake (4). Likewise, the relation between body fat and lipid abnormalities in South Asians is inconsistent (29). None of these studies accounted for carbohydrate intake.

High-carbohydrate diets are hypothesized to increase triacylglycerol concentrations by inducing fatty acid production in the liver and inhibiting the action of lipoprotein lipase (LPL) through increased apolipoprotein CIII production, particularly in the presence of insulin resistance (30). Higher fat intakes may raise circulating chylomicrons, but do not affect LPL and hence do not affect triacylglycerol concentrations. Higher triacylglycerol concentrations are related to smaller-sized LDL particles and lower HDL concentrations, which together are called the lipid triad (31). The effect of carbohydrate intake on serum lipids may be attenuated by factors related to insulin sensitivity, such as the maintenance of optimal body weight, higher lean body mass, and increased physical activity, and may explain why carbohydrate-rich diets are not related to abnormal serum lipids in lean, active populations. Consistent with a recent meta-analysis (32), we did not observe any relation with glycemic index, glycemic load, HDL cholesterol, or fasting triacylglycerols. Possible reasons for this finding may be little variation in the glycemic index of the diets and differences in the combinations in which carbohydrate was consumed. For instance, oil added to rice would slow the rate of absorption from the gut compared with plain rice.

Our data suggest that the differences in HDL and triacylglycerols observed between ethnic groups may in part be due to differences in carbohydrate intake. Sugar-containing soft drinks, juices, and snacks (but not total calories) reduced HDL. No association was observed with intakes of bread, rice, and potatoes. On the basis of these findings, we hypothesize that reducing the servings of sugar and sugared soft drinks, juices, and snacks may increase HDL cholesterol, but this would have to be confirmed in prospective studies.

The present study had some limitations. First, exposure and outcome were measured at the same time. We therefore excluded persons who reported morbidities or attempts at changing their diet because they may have done so as a consequence of their medical condition. Persons who reported comorbidities ate less and were less physically active but weighed more than those who did not report comorbidities, suggesting that they had likely altered their lifestyle as a result of the diagnoses. Because the persons remaining in the present study did not know they had any medical condition, their dietary histories probably reflected their usual diet and minimized the possibility of bias. Second, the sample size was relatively small. HDL has been related to intakes of polyunsaturated, monounsaturated, or saturated fats and fiber, but we did not observe these associations in the present study, most likely because of inadequate power; therefore, our nonsignificant results should be interpreted with caution. Likewise, we were limited in our ability to examine the relation between carbohydrate intake and HDL and triacylglycerols in the subgroups. Third, for the nutrient estimation from the FFQ, we used the average nutrient values of foods, which leads to inevitatble misclassification of exposure; most likely this was random, which would attenuate any associations. Last, there were substantially fewer Aboriginal Peoples in this sample compared with the rest of the ethnic groups. The reasons for this were that the proportion of persons who reported established diseases such as diabetes (29%) and hypertension (26%) was high among the Aboriginal Peoples and they were excluded from the analysis.

The study also has some strengths. The population was randomly sampled, multiethnic, and with varied carbohydrate intake. We collected data on several lifestyle and personal characteristics and were able to account for them in the analyses. We measured diet with validated, culture-specific food-frequency questionnaires. Our results are, therefore, likely to be more generalizable to a multiethnic population without diagnosed morbidity living in the Western world.

In conclusion, differences in HDL and triacylglycerols observed in different ethnic groups may in part be due to carbohydrate intake. Reducing the frequency of intake of sugar-containing soft drinks, juices, and snacks may be beneficial.

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