Consumption of Added Sugars and Indicators of Cardiovascular Disease Risk Among US Adolescents

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Background—Whereas increased carbohydrate and sugar consumption has been associated with higher cardiovascular disease risk among adults, little is known about the impact of high consumption of added sugars (caloric sweeteners) among US adolescents.

Methods and Results—In a cross-sectional study of 2157 US adolescents in the National Health and Nutrition Examination Survey (NHANES) 1999 to 2004, dietary data from one 24-hour recall were merged with added sugar content data from the US Department of Agriculture MyPyramid Equivalents databases. Measures of cardiovascular disease risk were estimated by added sugar consumption level (<10%, 10 to <15%, 15 to <20%, 20 to <25%, 25 to <30%, and ≥30% of total energy). Multivariable means were weighted to be representative of US adolescents and variances adjusted for the complex sampling methods. Daily consumption of added sugars averaged 21.4% of total energy. Added sugars intake was inversely correlated with mean high-density lipoprotein cholesterol levels (mmol/L) which were 1.40 (95% confidence interval [CI] 1.36 to 1.44) among the lowest consumers and 1.28 (95% CI 1.23 to 1.33) among the highest (P trend =0.001). Added sugars were positively correlated with low-density lipoproteins (P trend =0.01) and geometric mean triglycerides (P trend =0.05). Among the lowest and highest consumers, respectively, low-density lipoproteins (mmol/L) were 2.24 (95% CI 2.12 to 2.37) and 2.44 (95% CI 2.34 to 2.53), and triglycerides (mmol/L) were 0.81 (95% CI 0.74, 0.88) and 0.89 (95% CI 0.83 to 0.96). Among those overweight/obese (≥85th percentile body-mass-index), added sugars were positively correlated with the homeostasis model assessment (P linear trend =0.004).

Conclusion—Consumption of added sugars among US adolescents is positively associated with multiple measures known to increase cardiovascular disease risk. **(*Circulation*. 2011;123:249-257.)**

Key Words: Sugars ■ cardiovascular diseases ■ risk factors ■ lipids ■ triglycerides ■ diabetes mellitus

Cardiovascular disease (CVD) is a leading cause of morbidity and mortality among US adults. Whereas atherosclerosis and CVD occur later in life, their risk factors, including lipid disorders,[1] diabetes mellitus,[2] and obesity are increasingly identified among adolescents and even children. Currently 32% of US children and adolescents aged 2 to 18 years are overweight or obese.[3] Though CVD among children is rare, an increase in risk factors at younger ages and their apparent tendency to track into adulthood highlights the need for early and effective prevention efforts.[4–6]

Lifestyle changes, including dietary change, have long been a central focus of efforts to reduce CVD risk. Since the 1950s Americans have been advised to reduce their consumption of fats and cholesterol and replace them with complex carbohydrates.[7] It appears that, in part, Americans have followed this advice. But whereas food disappearance data suggest that fat consumption has decreased, it is refined rather than complex carbohydrates that have increased. Although the overall health impact of this trend is unclear, several studies have shown a positive correlation between the consumption of carbohydrates, particularly some sugars, and the presence of CVD risk factors.[8–10] A recent longitudinal study among women demonstrated that the incidence of CVD was increased among higher consumers of sugar-sweetened beverages,[11] the largest contributor of added sugars in the US diet.[12] Studies comparing the impact of different sugars have demonstrated that the monosaccharide fructose, but not glucose, raises triglyceride levels and lowers high-density lipoprotein (HDL) levels, suggesting that the metabolic impact may differ substantially by sugar type.[10,13]

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Added sugars are refined calorie-containing sweeteners added to foods and beverages during processing or prepara-

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tion. Consumption of these sugars has increased substantially in recent decades. Sugars used to sweeten soft drinks have become the largest single source of calories in the US diet.[14] In 1994 to 1996, Americans >2 years of age obtained nearly 16% of their total energy from added sugars; adolescents, the highest consumers, obtained >20% of their energy from these sugars.[15] Today in the US, the most commonly consumed added sugars are refined beet or cane sugar (sucrose) and high fructose corn syrup,[16] both of which contribute fructose and glucose in approximately equal amounts to the diet. Added sugars are estimated to contribute 74% to 80% of the dietary fructose consumed.[17,18]

Given the high consumption of added sugars among adolescents and the potential for long-term health risks associated with early diet, it is important to understand the impact of this dietary trend. The purpose of our study was to determine if there is an association between the consumption of added sugars and indicators of CVD risk among US adolescents and to determine if body weight modifies this association.

Methods

Study Design and Subjects

Data for our study come from the National Health and Nutrition Examination Survey (NHANES). NHANES is a sequential series of cross-sectional surveys of the US civilian noninstitutionalized population designed to obtain nationally representative estimates on diet and health indicators. A description of the complex sampling methodology is described elsewhere.[19] The study sample consists of adolescents ages 12 to18 year living in the US between 1999 and 2004 (n=2485) who were randomly selected to provide a fasting blood sample for NHANES 1999 to 2000, NHANES 2001 to 2002, or NHANES 2003 to 2004. Excluded from the sample (in order of exclusion) were those with unreliable[20] or implausible (<600 or >4500 kcal/d) dietary data (n=159), those pregnant (n=33), those with extreme triglyceride levels (>300 mg/dL; n=23), those with previously diagnosed diabetes mellitus (n=9), and those with missing covariate data (n=104). After exclusions, the total sample for this study included 2157 adolescents. Study protocols for NHANES 1999 to 2004 were approved by the institutional review board at the National Center for Health Statistics.[21] Signed informed consent was obtained from all participants and their parent/guardian.

Added Sugars and Other Dietary Intake

In NHANES 1999 to 2000 and NHANES 2001 to 2002, one 24-hour dietary recall was used to assess dietary intake from all participants. In NHANES 2003 to 2004, a second 24-hour recall was collected by phone from all respondents. For consistency, we used only the first dietary recall to assess intake for all participants in the primary analysis. In addition, a sensitivity analysis was performed using the mean added sugars intake for each of the respondents in NHANES 2003 to 2004. Nutrient content of the foods consumed was determined by NHANES using the Food and Nutrient Database for Dietary Studies, which uses food composition data from the US Department of Agriculture National Nutrient Database for Standard Reference.[22] Because the Standard Reference database does not include information on the added sugar content of many foods, we merged the individual food files from NHANES with the most recently released MyPyramid Equivalents database (MPED) files, those for 1999 to 2000, 2001 to 2002, and 2003 to 2004.[23] The MPED database provides standard serving size information for the major food categories found on the US Department of Agriculture Food Guide Pyramid (grains, meat, dairy, fruits, vegetables, and beans), as well as for added sugars and discretionary fats. A description of the MPED database[24] and the methods used to calculate the sugar content of foods can be found elsewhere.[25]

To determine the amount of added sugars consumed in each food and beverage, we multiplied the total amount consumed in grams (as provided in the NHANES database) by the amount of added sugars in each of these foods (teaspoons/100 g; as provided in the MPED database). The results for each food consumed were summed to obtain the total added sugars intake in teaspoons and converted to grams by multiplying by 4.2 g/teaspoon.[26] This result was multiplied by 4 kcal to obtain the total energy from added sugars. Finally, the total energy from added sugars (kcal) was divided by total energy intake (kcal/d) to obtain the percentage of total energy from added sugars.

Indicators of CVD Risk

Biological indicators known to be associated with CVD[1,27,28] were measured in NHANES using standardized laboratory procedures that have been described elsewhere.[29] Measured lipids include fasting serum or plasma: HDL cholesterol, total cholesterol (TC), and triglycerides. Measured indicators of glucose metabolism include fasting insulin and glucose. Anthropometric measures (height, weight, and waist circumference) and blood pressure were measured by trained interviewers using standardized equipment and protocols. Body-mass-index (BMI) was calculated from measured weight and height as kg/m^2, and BMI was converted to age- and sex-standardized percentiles and z-scores on the basis of Centers for Disease Control and Prevention 2000 growth charts.[30]

Low-density lipoprotein (LDL) levels were calculated using the Friedewald formula: LDL cholesterol (mmol/L)=TC−HDL cholesterol−triglycerides/2.19.[31] The homeostasis model assessment (HOMA-IR) is an estimate of insulin resistance derived from fasting glucose and insulin levels, with higher levels representing greater degrees of insulin resistance.[32] HOMA-IR was calculated using the formula developed by Mathews et al: fasting insulin (pmol/L)·fasting glucose (mmol/L)/22.5.[33]

Covariates

Variables previously shown to be associated with carbohydrate intake and with any of the CVD risk indicators specified above were included as covariates. These covariates include: measured waist circumference and BMI, as well as self-reported demographic data (participant's age [in years], sex, income, and race/ethnicity [% non-Hispanic white, non-Hispanic black, Hispanic, and other]). Given the small sample size, Mexican-American and other Hispanic were combined into a single category entitled "Hispanic" for analyses. Because education, when compared to income and occupation, has been shown to be the only measure of socioeconomic status significantly associated with measures of CVD risk,[34] we included educational level of lease or mortgage holding parent/guardian (greater than high-school [yes or no]) in our models. Because of the high number of missing values (7.5% of sample), we elected to not include income as a second measure of socioeconomic status. As a measure of physical activity, respondents were asked to provide a list all of the moderate or vigorous leisure activities they engaged in over the previous month and to provide the frequency and the usual duration of these activities. MET (metabolic equivalent) minutes were then calculated as the sum of the following for each reported activity: duration in minutes·frequency·metabolic equivalent intensity level (MET score).

The values for dietary covariates were determined using data from one 24-hour dietary recall and included total energy intake and the total energy-adjusted nutrient residuals for fiber, other carbohydrates (excluding added sugars and fiber), saturated fats (SFAs), polyunsaturated fatty acids (PUFAs), and monounsaturated fatty acids (MUFAs), proteins, fiber, sodium, and cholesterol. These nutrient residuals were calculated using linear regression models with total calorie intake as the predictor and the absolute intake of each nutrient of interest (in grams) as the outcome in order to separate the nutrient effect from that of the calories consumed.[35]

Data Analysis

Statistical Analysis Software version 9.2 (SAS Institute, Cary, NC) was used for all analyses. Procedures that account for the complex

sampling methods used in NHANES were applied. Sample weights for the 6 years of data that reflect the probability of selection, nonresponse, and poststratification adjustments were calculated as follows: 2/3·wtsaf4yr (fasting sample weight for NHANES 1999 to 2002) and 1/3·wtsaf2yr (fasting sample weight for NHANES 2003 to 2004)[19] and used to ensure that results were representative of the US population. To ensure sufficiently large sample sizes in each group, respondents were grouped into 6 groups of approximately equal size by the percentage of their total energy intake from added sugars: 0% to <10%, 10% to <15%, 15% to <20%, 20% to <25%, 25% to <30%, and ≥30%. All of the *P* values were 2-sided. A *P* value <0.05 was considered statistically significant for main effects.

Percentages, means, and standard error (SE) of key variables were calculated to describe the sample at each level of added sugars intake. Linear regression models were used to assess the relationship between intake of added sugars and our outcome measures while controlling for the effect of potentially confounding variables. As the distribution of triglycerides was skewed, the values in the linear regression models were log transformed, and geometric means are presented. Estimate statements in the regression models were used to determine the adjusted mean of each of the measures of CVD risk for each level of added sugar intake.[36] Contrasts were used to specify linear tests among the levels of added sugars consumption and to compare each group of respondents to the referent group (<10% of total energy from added sugars) for each of the outcomes of interest.[36] χ^2 tests were used to test differences in categorical variables and Wald f-tests were used for continuous variables.

To identify the macronutrients to be included in our regression models, we first performed bivariate analyses to assess the association between the intake of total fat and the intake of protein with each of our outcomes. The energy-adjusted residuals for protein but not fat were found to be associated with measures of dysglycemia (fasting insulin, fasting glucose, and HOMA-IR), blood pressure, and adiposity (BMI and waist circumference). Therefore, we included protein but not fat intake as a covariate in these models. Because we also controlled for total energy intake and intake of carbohydrates (other than added sugars), results obtained using these models can be interpreted as the effect of replacing fat in the diet (the macronutrient left out of the models) with added sugars.[35] In contrast, the energy adjusted residuals for the intake of PUFAs, MUFAs, and SFAs, but not proteins, were each found, in bivariate analyses, to be associated with blood lipid measures (HDL, LDL, TC, and triglycerides). Therefore, we included the intake of each of these dietary fats, but not protein, in the models with lipid measures as the outcome. The results of these models can be interpreted at the effect of replacing protein in the diet with added sugars.

Because of problems with multicollinearity in models that included both BMI and waist circumference, waist circumference was dropped from the regression models. Given that postprandial lipoprotein[37] and insulin responses[38] have been shown to differ by body weight, race, and sex, we tested for the presence of effect modification between level of added sugars intake and each of these variables by including a multiplicative term for each in the models. Body weight was dichotomized as not overweight (<85th percentile BMI; n=1340) and overweight (≥85th percentile BMI; n=817).[39] A *P* value of <0.10 was considered significant.

Sensitivity analysis was performed to examine the association between intake of added sugars and HDL and HOMA-IR levels using the absolute intake of added sugar (in grams) as the exposure rather than the proportion of total energy from added sugars. To do this we grouped all respondents into 6 groups of equal size according to the grams of added sugars consumed. In addition, to determine if our results were consistent when data from two 24-hour recalls were used, we repeated our analysis using a smaller (≈30%) subsample of respondents from whom a second 24-hour dietary recall had been collected. In these analyses, the mean intake of added sugars (percentage total energy) and of other dietary covariates was used for each respondent together with the same nondietary covariates described for the models above.

Results

A description of the study sample by level of added sugars is provided in Table 1. No significant differences were seen between level of added sugars consumed and demographic factors, including age, sex, race/ethnicity, poverty, or educational level. Similarly, no association was seen between the amount of added sugars consumed and physical activity or total energy intake.

Daily consumption of added sugars averaged 118.9 g (28.3 tsp or 476 calories) daily. This represents 21.4% (95% confidence interval [CI] 20.5% to 22.2%) of total daily caloric intake (total energy; not shown). There was no significant difference in consumption across racial/ethnic groups. The increased trend in percentage total energy from carbohydrates with higher intake of added sugars was significant (*P* linear trend <0.0001), as was the increased trend in the absolute intake of carbohydrates (*P* linear trend <0.0001 (Table 1). Intake of added sugars was negatively correlated with both the percentage total energy and the absolute intake (g) of total fats, SFAs, PUFAs, MUFAs, and protein (*P* linear trend <0.0001 for all). Fiber, cholesterol, and sodium intakes were also negatively correlated with intake of added sugars (*P* linear trend <0.0001, 0.0003, and <0.0001, respectively).

In fully adjusted linear regression models we found that neither body weight, race/ethnicity, nor sex modified the association between added sugar intake and lipid measures. Lipid levels were correlated with intake of added sugars (Table 2). HDL levels were lower among those who consumed more added sugars (*P* linear trend =0.001). Among the highest consumers (≥30% total energy), HDLs were 1.28 mmol/L (95% CI 1.23 to 1.33; 49.5 mg/dL) compared with 1.40 mmol/L (95% CI 1.36 to 1.44; 54.0 mg/dL) among the lowest consumers (<10% total energy), a difference of 9% (*P*=0.001; Figure 1). In contrast, LDL and geometric mean triglyceride levels were higher among those consuming higher levels of added sugars (*P* linear trend =0.01 and 0.05, respectively; Table 2). Among the highest compared to the lowest consumers, adjusted LDL levels were 2.44 mmol/L (95% CI 2.34 to 2.53; 94.3 mg/dL) and 2.24 mmol/L (95% CI 2.12 to 2.37; 86.7 mg/dL), and geometric mean triglyceride levels were 0.89 mmol/L (95% CI 0.83 to 0.96; 79.0 mg/dL) and 0.81 mmol/L (95% CI 0.74 to 0.88; 71.7 mg/dL), respectively. This represents a difference between lowest and highest consumers of 9% in LDL levels (*P*=0.08) and 10% in triglyceride levels (*P*=0.07). There was no significant trend in TC with higher intake of added sugars (*P* linear trend=0.16).

Because the effect of added sugars intake was shown to be modified by body weight (but not by race/ethnicity or sex) in models with HOMA-IR, insulin, glucose, systolic blood pressure, and waist circumference as the outcomes (*P* interaction =0.09 for glucose and ≤0.003 for all other outcomes), the analyses of these measures were stratified by weight status. We found that the intake of added sugars and HOMA-IR measures were positively correlated among overweight adolescents (*P* linear trend =0.004) but not among those who were normal weight (*P* linear trend =0.41; Figure 2). Adjusted mean HOMA-IR among overweight adolescents with the highest consumption was 4.61 (95% CI 4.08 to 5.13)

Table 1. Description of US Adolescents (12 Years to 18 Years of Age) by Intake of Added Sugars, NHANES 1999 to 2004

	% Total Energy From Added Sugars					
	0% to <10% (n=300)	10% to <15% (n=364)	15% to <20% (n=425)	20% to <25% (n=369)	25% to <30% (n=303)	≥30% (n=396)
Age, y	14.8 (0.2)	14.8 (0.1)	15.1 (0.2)	14.9 (0.2)	15.2 (0.1)	14.9 (0.2)
Sex, male, %	53.1 (3.4)	50.6 (3.6)	48.7 (3.4)	46.5 (3.9)	52.8 (3.5)	52.2 (2.7)
Race/ethnicity						
Non-Hispanic white, %	57.5 (4.1)	64.9 (3.7)	61.0 (2.9)	67.3 (3.6)	62.9 (3.9)	63.0 (2.9)
Non-Hispanic black, %	14.8 (2.7)	13.4 (2.0)	15.0 (1.8)	14.5 (2.1)	15.0 (2.3)	13.9 (1.8)
Hispanic, %	18.5 (0.9)	17.6 (2.5)	18.3 (2.7)	13.0 (2.1)	17.3 (2.8)	14.1 (2.2)
Other, %	10.5 (2.7)	4.1 (1.5)	4.1 (1.5)	5.2 (1.6)	4.8 (1.5)	9.0 (2.7)
Poverty–income ratio	2.46 (0.15)	2.82 (0.15)	2.72 (0.09)	2.87 (0.11)	2.54 (0.13)	2.41 (0.14)
Education of parent/guardian (≤high school diploma)	49.0 (5.2)	44.8 (5.2)	44.7 (5.2)	45.1 (5.2)	48.2 (5.2)	55.3 (5.2)
Physical activity, MET min	12268 (1520)	14154 (1156)	11514 (867)	13715 (1552)	13165 (1552)	10375 (723)
Energy intake, kcal/day	2070 (75)	2303 (58)	2344 (58)	2347 (49)	2299 (66)	2081 (62)
Carbohydrate intake						
Total, % total energy*	46.6 (0.01)	50.3 (0.01)	52.8 (0.01)	55.0 (0.01)	57.4 (0.01)	64.8 (0.01)
Total, g*	239 (8.2)	286 (6.6)	306 (8.6)	322 (7.2)	327 (9.3)	334 (10.7)
Added sugars, g*	31.0 (1.5)	73.2 (2.7)	103 (2.7)	132 (2.7)	158 (4.4)	200 (8.2)
Fiber intake, g*	14.9 (0.6)	14.6 (0.4)	15.0 (0.6)	13.6 (0.5)	11.7 (0.5)	9.8 (0.4)
Protein intake						
% Energy*	17.1 (0.4)	15.3 (0.3)	14.0 (0.2)	13.3 (0.3)	12.5 (0.3)	10.4 (0.2)
Total, g*	87.6 (3.6)	88.7 (2.4)	81.9 (2.3)	77.9 (2.4)	72.2 (2.9)	53.8 (1.8)
Fat intake						
Total, % total energy*	36.5 (0.7)	34.9 (0.6)	33.8 (0.5)	32.6 (0.6)	31.3 (0.4)	26.2 (0.4)
Total, g*	85.5 (4.3)	89.9 (3.1)	89.3 (2.5)	85.1 (2.3)	81.1 (2.8)	61.8 (2.2)
MUFAs, % energy*	13.5 (0.3)	13.2 (0.3)	12.8 (0.2)	12.4 (0.2)	11.9 (0.2)	10.0 (0.2)
PUFAs, % energy*	7.1 (0.3)	7.0 (0.3)	6.7 (0.2)	6.4 (0.2)	6.1 (0.2)	5.1 (0.2)
SFAs, % energy*	13.0 (0.3)	12.0 (0.2)	11.6 (0.2)	11.3 (0.3)	10.9 (0.2)	9.1 (0.2)
Cholesterol intake, g†	264 (18)	289 (15)	250 (8)	251 (16)	251 (23)	171 (10)
Sodium intake, mg*	3638 (155)	3805 (111)	3616 (112)	3499 (114)	3237 (120)	2569 (87)
Waist circumference, cm	78.0 (0.9)	80.7 (1.2)	77.0 (0.7)	80.3 (1.2)	79.8 (1.2)	80.6 (1.1)
BMI, z-score	0.52 (0.07)	0.63 (0.09)	0.32 (0.06)	0.56 (0.08)	0.49 (0.09)	0.61 (0.08)
HOMA-IR†	2.54 (0.11)	2.69 (0.14)	2.46 (0.08)	3.01 (0.18)	2.89 (0.14)	2.92 (0.13)
Triglyceride, mmol/L‡	0.95 (0.04)	0.98 (0.04)	0.93 (0.01)	1.01 (0.01)	1.03 (0.01)	1.02 (0.01)
HDL, mmol/L*	1.38 (0.02)	1.31 (0.02)	1.31 (0.03)	1.29 (0.03)	1.23 (0.03)	1.27 (0.03)
LDL, mmol/L	2.33 (0.05)	2.30 (0.04)	2.37 (0.06)	2.50 (0.06)	2.38 (0.06)	2.39 (0.06)
TC, mmol/L	4.14 (0.06)	4.06 (0.05)	4.11 (0.04)	4.25 (0.09)	4.08 (0.06)	4.13 (0.04)
Systolic BP, mm Hg‡	107 (0.7)	109 (1.1)	108 (0.7)	108 (1.0)	110 (0.9)	108 (0.7)
Diastolic BP, mm Hg‡	60.7 (0.9)	61.7 (0.7)	62.9 (0.7)	61.1 (0.8)	65.5 (0.7)	62.7 (0.7)

% Energy indicates percentage total energy intake; physical activity, sum of MET (metabolic equivalent)·frequency·duration for all leisure time activities previous month; and poverty–income ratio, ratio of annual family income to federal poverty level.

All results are adjusted to account for the complex sampling method used by NHANES and weighted to be representative of the US population. Results are presented as means (SEs) unless specified as % (SEs). Analysis of contrasts in linear and logistic regression was used to test trends using χ^2 for categorical variables and Wald F tests for continuous variables.

*P linear trend <0.0001; †P linear trend <0.001; ‡P linear trend <0.05.

compared with 3.49 (95% CI 3.02 to 3.95) among the lowest consumers, a difference of 32% (Table 2). A similar difference was observed with fasting insulin levels. No significant association was observed between consumption of added sugars and fasting glucose. Similarly, there were no significant trends in systolic or diastolic blood pressure, waist circumference, or BMI (among either the overweight or not overweight) with increased intake of added sugars (Table 2).

We repeated the analyses of the associations between intake of added sugars and mean HDL and HOMA-IR levels with respondents divided into 6 equally-sized groups according to their absolute daily intake of added sugars (0 to <49.5,

Table 2. Intake of Added Sugars and Indicators of Cardiovascular Disease Risk, NHANES 1999 to 2004

	% Total Energy From Added Sugars						
	0% to <10% (referent) (n=300)	10% to <15% (n=364)	15% to <20% (n=425)	20% to <25% (n=369)	25% to <30% (n=303)	≥30% (n=396)	*P* Linear Trend
Model 1							
Lipid measures, mmol/L							
HDL cholesterol	1.40 (1.36 to 1.44)	1.35 (1.30 to 1.40)	1.31† (1.27 to 1.35)	1.32* (1.27 to 1.36)	1.24§ (1.19 to 1.29)	1.28† (1.23 to 1.33)	0.001
LDL cholesterol	2.24 (2.12 to 2.37)	2.27 (2.16 to 2.37)	2.37* (2.31 to 2.44)	2.51* (2.35 to 2.66)	2.42 (2.29 to 2.55)	2.44 (2.34 to 2.53)	0.01
TC	4.05 (3.92 to 4.19)	4.04 (3.94 to 4.15)	4.11 (4.02 to 4.19)	4.27 (4.11 to 4.43)	4.12 (3.99 to 4.25)	4.16 (4.05 to 4.27)	0.16
Triglycerides	0.81 (0.74 to 0.88)	0.83 (0.78 to 0.89)	0.84 (0.82 to 0.87)	0.87 (0.82 to 0.93)	0.90 (0.84 to 0.97)	0.89 (0.83 to 0.96)	0.05
Model 2							
HOMA-IR							
Not overweight	2.70 (2.06 to 3.33)	2.73 (2.11 to 3.36)	2.71 (2.09 to 3.34)	2.77 (2.12 to 3.41)	2.91 (2.23 to 3.58)	2.74 (2.11 to 3.37)	0.41
Overweight	3.49 (3.02 to 3.95)	3.65 (3.15 to 4.16)	4.17* (3.86 to 4.47)	4.74† (4.07 to 5.41)	4.34* (3.81 to 4.86)	4.61† (4.08 to 5.13)	0.004
Insulin (fasting), pmol/L							
Not overweight	78.5 (59.9 to 97.0)	80.1 (62.2 to 98.0)	78.5 (62.2 to 97.1)	80.9 (62.2 to 99.5)	84.6 (79.6 to 89.6)	80.7 (62.7 to 98.7)	0.33
Overweight	108 (96.0 to 121)	112 (97.9 to 126)	127* (122 to 136)	140* (122 to 159)	130* (115 to 145)	139† (124 to 155)	0.006
Glucose (fasting), pmol/L							
Not overweight	5.36 (5.18 to 5.55)	5.33 (5.14 to 5.52)	5.42 (5.17 to 5.63)	5.37 (5.17 to 5.57)	5.44 (5.24 to 5.65)	5.35 (5.12 to 5.57)	0.54
Overweight	5.03 (4.91 to 5.15)	5.04 (4.95 to 5.14)	5.09 (5.04 to 5.15)	5.15 (5.04 to 5.26)	5.14 (5.06 to 5.22)	5.08 (4.99 to 5.18)	0.16
Systolic blood pressure, mm Hg							
Not overweight	89.6 (83.4 to 95.9)	90.9 (84.8 to 97.0)	90.8 (84.6 to 97.0)	90.6 (83.4 to 97.8)	93.1† (86.9 to 99.2)	91.3 (85.0 to 97.5)	0.07
Overweight	110 (108 to 113)	112 (110 to 114)	112 (110 to 115)	113 (110 to 115)	114* (112 to 117)	114 (111 to 116)	0.11
Waist circumference, cm							
Not overweight	47.2 (44.7 to 49.8)	48.5 (46.3 to 51)	48.5 (46.1 to 50.8)	48.2 (46.1 to 50.4)	47.9 (45.6 to 50.3)	48.7‡ (46.5 to 50.9)	0.31
Overweight	93.6 (92.3 to 94.8)	94.2 (92.8 to 95.6)	92.6 (91.5 to 93.8)	94.5 (93.2 to 95.9)	93.7 (92.4 to 95.0)	92.3 (90.7 to 93.8)	0.52
BMI, z-score							
Not overweight	0.32 (0.00 to 0.90)	0.41 (0.00 to 1.00)	0.30 (0.00 to 0.85)	0.28 (0.00 to 0.87)	0.21‡ (0.00 to 0.76)	0.44 (0.00 to 0.96)	0.92
Overweight	1.65 (1.54 to 1.76)	1.80 (1.67 to 1.92)	1.65 (1.57 to 1.74)	1.72 (1.60 to 1.85)	1.73 (1.61 to 1.84)	1.88‡ (1.77 to 2.00)	0.07

BMI is adjusted for age and sex.

Model 1: means adjusted for sex, race, age, education, BMI (excluding model with BMI as outcome), physical activity, total energy intake, nutrient residuals for intake of fats (MUFAs, PUFAs, SFAs), sodium, cholesterol, and fiber.

Model 2: means adjusted for all covariates included in Model 1 except that all fats (PUFAs, MUFAs, SFAs) have been replaced with the energy-adjusted nutrient residuals for protein. Not overweight indicates BMI <85th percentile; Overweight, overweight or obese (BMI ≥85th percentile).

*Mean values differ significantly from the referent: $P<0.05$.

†Mean values differ significantly from the referent: $P<0.01$.

‡Mean values differ significantly from the referent: $P<0.001$.

§Mean values differ significantly from the referent: $P<0.0001$.

49.5 to <79.6, 79.6 to <106.7, 106.7 to <137.1, 137.1 to <180.4, and ≥180.4 g of added sugars) rather than by consumption relative to their total energy intake. The results were very similar to those obtained in the primary analysis. HDLs were 1.19 mmol/L (95% CI 1.10 to 1.28) among the highest consumers and 1.40 mmol/L (95% CI 1.31 to 1.49) among the lowest (*P* linear trend =0.004). HOMA-IRs among those who were overweight were 4.85 (95% CI 3.99 to 5.72) among the highest consumers and 3.39 (95% CI 2.84 to 3.94) among the lowest (*P* linear trend=0.02).

When the analysis was repeated using the mean intake obtained from the smaller subsample of respondents who provided two 24-hour dietary recalls (those participating in NHANES 2003 to 2004; n=646), point estimates and trends for HDL and HOMA-IR were again similar to those obtained in the primary analyses. Among the highest versus lowest added sugar consumers (percentage total energy), HDLs were 1.34 mmol/L (95% CI 1.24 to 1.44) and 1.43 mmol/L (95% CI 1.34 to 1.53), respectively (*P* linear trend =0.009), and HOMA-IRs among the overweight/obese were 4.97 (95% CI 3.19 to 6.74) and 3.19 (95% CI 2.43 to 3.95), respectively (*P* linear trend =0.05).

Discussion

In 1986, the Sugars Task Force of the US Food and Drug Administration published a review of the research then available and concluded that there was no conclusive evidence of an association between sugar consumption and CVD or its risk factors.[40] Since then, the results of several new epidemiological studies and short- and long-term experimen-

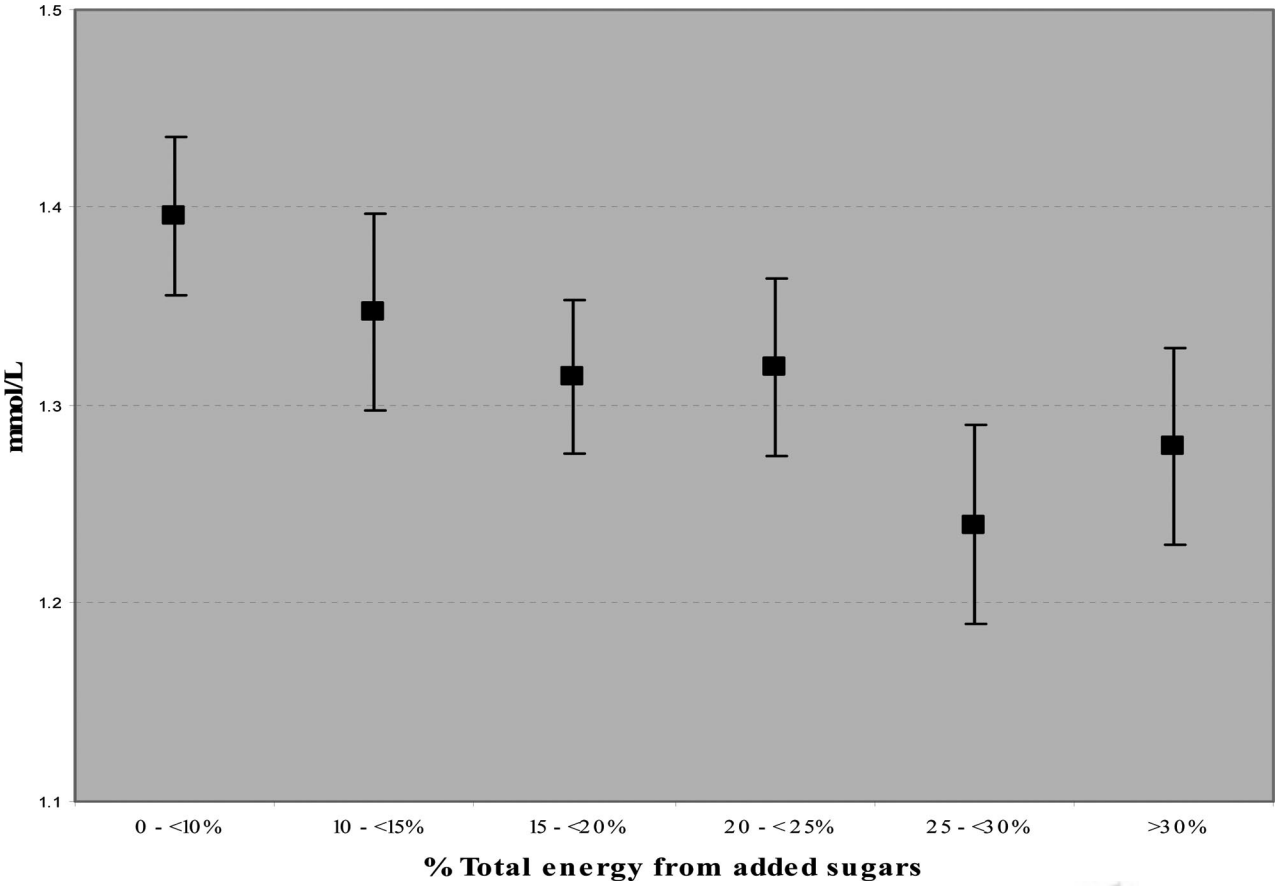

Figure 1. Multivariable-adjusted mean HDL levels by intake of added sugars among US Adolescents. Participants grouped by percentage of total energy intake from added sugars. *P* for linear trend =0.001 for HDL levels. Error bars indicate 95% CIs. To convert HDL values to mg/dL, multiply by 39.

tal studies have provided more evidence linking the intake of carbohydrates[41,42] and sugars[10,11,42,43] (particularly fructose[10,44,45]) and increased risk of CVD. And importantly, consumption of added sugars has risen substantially since the research reviewed in the Sugar Task Force report was done. The Task Force report estimated that consumption of added sugars among adolescents was 62 to 84 g in 1977 to 1978. The results of our study indicate that by 1999 to 2004 consumption among this group had risen to 119 g, an increase of 42% to 92%.

Our results demonstrate that intake of added sugars is positively associated with known cardiovascular risk factors when controlling for other characteristics. We found increased dyslipidemia (lower HDLs and higher LDLs and triglyceride levels) among adolescents, regardless of body size, and increased insulin resistance (higher fasting insulin and HOMA-IR measures) among those overweight or obese with higher intake of added sugars. Several mechanisms have been proposed to explain the dysmetabolic effects of carbohydrates and specifically sugars. These include (1) the insulin response to the metabolism of high–glycemic index foods, such as processed sugars, that cause a rapid postprandial rise and fall in glucose levels; (2) the increased de novo lipogenesis that results when high levels of fructose are metabolized by the liver; and (3) increased hepatic triglyceride synthesis combined with increased secretion and/or decreased clearance of very-low-density lipoproteins.[46] Modification of the effect of added sugars on measures of glucose metabolism by weight status could be explained by the decreased insulin sensitivity known to result from increased adiposity.[38]

Clearly, added sugars play a significant role in the US diet. They increase the desirability of foods by increasing sweetness. They also contribute substantially to energy intake without contributing other important nutrients to the diet.[47] Existing guidelines for limiting the consumption of added sugars vary widely. The Institute of Medicine suggests a limit of 25% of total energy from added sugars in order to ensure adequate intake of important nutrients,[48] the World Health Organization advises limiting added sugars to <10% total energy to prevent dental caries, obesity, and chronic disease[49] and recently released recommendations from the American Heart Association advise that daily intake of added sugars should be limited to <100 calories daily for women and 150 calories for men[46] (≈5% of total energy) as a strategy for preventing heart disease. The 2005 US Dietary Guidelines for Americans encourage consumers to "choose and prepare foods and beverages with little added sugars or caloric sweeteners"[47] but do not specify an upper limit. Although our results support the need for dietary guidelines that encourage lower intake of added sugars, they also highlight the need for a comprehensive examination of the evidence on the effect of added sugars on cardiovascular and other chronic disease risks.

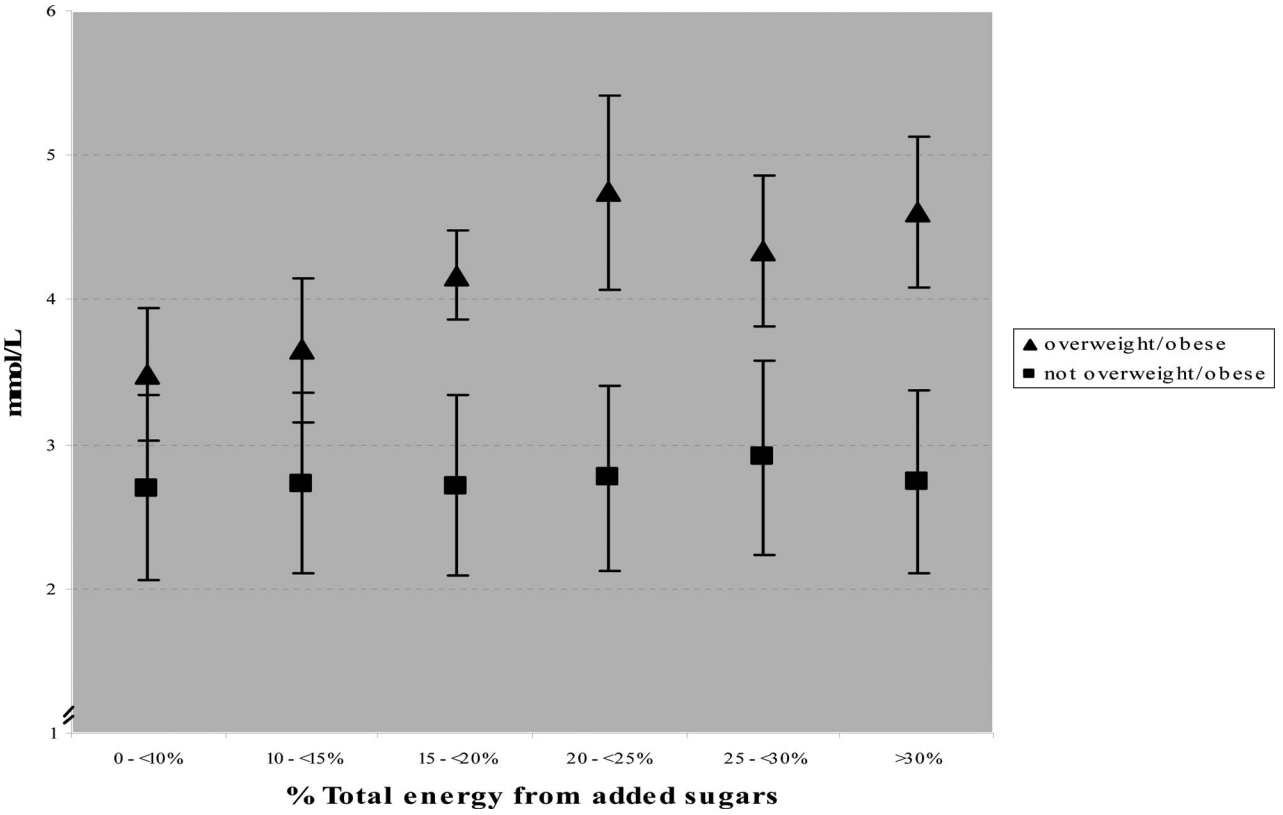

Figure 2. Intake of added sugars and adjusted mean HOMA-IR by weight status. Participants grouped by percentage of total energy intake from added sugars. Among overweight/obese adolescents, *P* for linear trend =0.004; among normal weight adolescents, *P* for linear trend =0.41. Error bars indicate 95% CIs.

Our study has several important strengths. First, we have used nationally representative data, and, to our knowledge, this is the first study to assess the association between added sugars and indicators of CVD risk among US adolescents. Second, we were able to control for several important confounding variables, including BMI, socioeconomic status, and physical activity. Also, because we had complete 24-hour dietary recall data on all participants, we were able to control for total energy intake, the intake of specific fats, and other dietary factors. Availability of a second 24-hour dietary recall in a subsample of respondents enabled us to do a sensitivity analysis using the mean of 2 days intake of added sugars. Finally, the use of trained staff following standardized protocols to measure height and weight and collect laboratory and interview data increases the accuracy and validity of the data collected.

Our study is also subject to some limitations. Cross-sectional studies such as ours are limited by the fact that exposures and outcomes are measured at the same time. As a result, our data can be used only to assess associations. They cannot be used to assess the direction or temporarily of these associations or to determine causality. Also, as only a single 24-hour dietary recall was used to assess diet, the dietary intake data may not represent the usual diet of respondents. Our inability to account for within person day-to-day variability may have resulted in some misclassification of the intake of added sugars, but we expect that this would be random.[50] In addition, when we evaluated those with 2 available 24-hour recalls, key findings remained consistent.

Whereas underreporting of certain foods high in sugars, such as sodas and sweets, may occur more frequently among those who underreport total energy,[51] such as those overweight or obese[52] who are also at increased risk of diabetes mellitus and dyslipidemia, systematic misclassification of this type would be expected to bias our findings toward the null. In addition, as no information on the validity of the process used to estimate added sugar content data in the US Department of Agriculture MPED database is available, there could be some misclassification of our exposure variable. Similarly, because the instruments used to assess important covariates such as physical activity have not been validated in this population, residual confounding could also be present.

In conclusion, higher consumption of added sugars among US adolescents is associated with several important CVD risk factors. Though long-term trials to study the effect of reducing the consumption of added sugars are needed, the results of this study suggest that future risk of CVD may be reduced by minimizing consumption of added sugars among adolescents.

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Disclosures

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CLINICAL PERSPECTIVE

Consumption of added sugars (caloric sweeteners), which contribute calories but no other nutrients to the diet, are the source of more than one fifth of the calories consumed by US adolescents. The results of our study show that higher consumption of these sugars is associated with blood lipid levels that may place adolescents at increased risk of future cardiovascular disease. We also found that the risks associated with added sugar consumption may be higher among overweight or obese adolescents because higher consumption among this group was also associated with increased insulin resistance. Our findings highlight the prominence of added sugars in the diets of adolescents and suggest that reducing this consumption could be a strategy for modifying cardiovascular disease risk factors and helping to prevent cardiovascular disease. The associations demonstrated in our cross-sectional study point to the need for controlled trials to determine if reducing consumption of added sugars can improve cardiovascular disease risk factors in adolescents and prevent future disease.

Consumption of Added Sugars and Indicators of Cardiovascular Disease Risk Among US Adolescents

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