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Consumption of Added Sugars and Cardiometabolic Risk Indicators Among US Adolescents

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Abstract

Background—Increased carbohydrate and sugar consumption has been associated with dyslipidemia among adults. However, the effect of high consumption of added sugars (caloric sweeteners) on measures of cardiometabolic risk among US adolescents is unknown.

Methods and Results—This was a cross-sectional study of 2,252 US adolescents (13–18 y) in the National Health and Nutrition Examination Survey (NHANES) 1999–2004. Dietary data from one 24-hour recall were merged with added sugar content data from the USDA MyPyramid Equivalents Databases. Multivariate-adjusted means of cardiometabolic indicators were estimated by added sugar consumption level (<10%, 10– <15%, 15– <20%, 20– <25%, 25– <30%, and ≥30% total energy) and weighted to be representative of US adolescents. Mean consumption of added sugars was 21.4% of daily energy intake. Adjusted mean high-density lipoprotein cholesterol (HDL) levels were lower, 1.38 mmol/L (95% CI: 1.32, 1.43) among the lowest consumers to 1.28 mmol/L (95% confidence interval [CI]: 1.23, 1.33) among the highest (p-trend=0.007). Geometric mean triglyceride levels ranged from 0.79 mmol/L (95% CI: 0.72, 0.86) to 0.89 mmol/L (95% CI: 0.83, 0.96) (p-trend=0.03) with greater consumption of added sugars.

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Among those overweight/obese (≥85th percentile body-mass-index [BMI]), HOMA-IRs were positively associated with added sugars (p-linear trend<0.001), averaging 78% higher among the highest vs. the lowest consumers (p<0.001). No significant trends were seen with low-density lipoproteins, body-mass-index, or blood pressure.

Conclusion—In US adolescents, consumption of added sugars is positively associated with measures of cardiometabolic risk. Long-term studies are needed to determine if reduction in added sugars will improve these parameters and, thereby decrease future cardiovascular events.

Keywords

Sugars; cardiovascular disease risk factors; lipids; triglycerides; diabetes mellitus

Cardiovascular disease (CVD) is the leading cause of morbidity and mortality among U.S. adults.[1] While atherosclerosis and CVD occur later in life, their risk factors, including lipid disorders[2], diabetes[3], and obesity are increasingly being identified among adolescents and even children.[4] Though CVD among children is rare,[4] an increase in cardiometabolic risk factors at younger ages and their apparent tendency to track into adulthood[5–7] highlights the need for early and effective prevention efforts.

Lifestyle changes, including dietary change, have long been a central focus of efforts to reduce CVD risk. Since the 1950's Americans have been advised to reduce their consumption of fats and cholesterol, and replace them with complex carbohydrates.[8] It appears that, in part, Americans have followed this advice. But while food disappearance data suggests that fat consumption has decreased, it is refined rather than complex carbohydrates that have increased.[9] While 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.[10–12] A recent longitudinal study among women demonstrated that the incidence of CVD increased with higher consumption of sugar-sweetened beverages,[13] the largest contributor of added sugars in the U.S. diet.[14] Studies comparing the impact of different sugars have demonstrated that the monosaccharide fructose but not glucose, raises triglyceride levels and lowers HDL levels, suggesting that the metabolic impact may differ substantially by sugar type.[12, 15]

Added sugars are refined, calorie-containing sweeteners added to foods and beverages during processing or preparation. 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 U.S. diet.[16] In 1994–1996, Americans over the age of 2 y obtained nearly 16% of their total energy from added sugars; adolescents, the highest consumers, obtained more than 20% of their energy from sugars added to foods and beverages.[17] Today in the U.S., the most commonly consumed added sugars are refined beet or cane sugar (sucrose) and high fructose corn syrup (HFCS),[18] both of which contribute fructose and glucose, in approximately equal amounts, to the diet. Added sugars are estimated to contribute 74%–80% of the dietary fructose consumed.[19, 20]

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 cardiometabolic health among U.S. adolescents.

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-section surveys of the U.S. civilian, non-institutionalized population designed to obtain nationally representative estimates on diet and health indicators. A description of the complex sampling methodology is described elsewhere.[21] The population for the current study consists of a sample of adolescents ages 12 to18 y living in the US between 1999 and 2004 (n=2,485) who were randomly selected to provide a fasting blood sample for NHANES 1999–2000, NHANES 2001–02, or NHANES 2003–04. Excluded from the sample (in order of exclusion) were: those with unreliable (n=48) or implausible (<600 or >4,500 kcal/day) (n= 111) dietary data, those pregnant (n= 33), those with extreme triglyceride levels (>300 mg/dL) (n=23), and those with previously diagnosed diabetes (n=9). After exclusions, the total sample for this study included 2,251 adolescents.

Added Sugars and Other Dietary Intake

In NHANES 1999–2000 and NHANES 2001–2002 one 24-hour dietary recall was used to assess dietary intake from all participants. In NHANES 2003–04 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 done using the mean added sugars intake for each of the respondents in NHANES 2003–04. Nutrient content of the foods consumed was determined by NHANES using the Food and Nutrient Database for Dietary Studies, which utilizes food composition data from the United States Department of Agriculture (USDA) 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 1999–2000, 2001–02, and 2003–04.[23] The MyPyramid database provides standard serving size information for the major food categories found on the USDA Food Guide Pyramid (grains, meat, dairy, fruits, vegetables, and beans) as well as for added sugars and excess fat. 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 grams) (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 grams/ teaspoon.[22] 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/day) to obtain the percent of total energy from added sugars.

Cardiometabolic Risk Indicators

Biological indicators known to be associated with CVD[2, 26, 27] were measured in NHANES using standardized laboratory procedures that have been described elsewhere.[28] Measured lipids include fasting serum or plasma: total cholesterol [TC], high-density lipoproteins [HDL], and triglycerides [TG]. Measures 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 based on the Centers for Disease Control and Prevention (CDC) 2000 growth charts.[29]

Low-density lipoprotein levels were calculated using the Friedewald formula: LDL-cholesterol = total cholesterol – HDL-cholesterol– triglycerides/5.[30] 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.[31] HOMA-IR was calculated using the formula developed by Mathews et. al: fasting insulin (pmol/L)*fasting glucose (mmol/L)/22.5.[32]

Covariates

Variables previously shown to be associated with carbohydrate intake and with any of the cardiometabolic risk indicators specified above were included as covariates. These covariates include: waist circumference and BMI z-score as well as self-reported demographic data (participant's age [y], 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. Income level was dichotomized based on poverty-income ratio (ratio of annual family income to federal poverty line). Below poverty indicates income at or below 130% of poverty. MET- (metabolic equivalent) minutes of leisure time physical activity over the previous month were 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), poly-unsaturated fatty acids (PUFAs), and mono-unsaturated fatty acids (MUFAs); proteins; 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.[33]

Data analysis

Statistical Analysis Software (SAS), version 9.2 (SAS Institute, Cary, IN) 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 were calculated as follows: 2/3* wtsaf4yr (fasting sample weight for NHANES 1999–2002) and 1/3* wtsaf2yr (fasting sample weight for NHANES 2003–04)[21] and used to ensure that results were representative of the U.S. population. Respondents were grouped into 6 approximately equally-sized groups by the % of their total energy intake from added sugars: 0 <10%, 10– <15%, 15– <20%, 20– <25%, 25– <30%,[34] and ≥30% of total energy intake from added sugars. All of the p-values were 2-sided. A p-value<0.05 was considered statistically significant.

Percentages, means, and standard errors (SE) of key demographic and dietary 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 the various outcome measures while controlling for the effect of potentially confounding variables. As the distribution of TG was skewed, TG 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 cardiometabolic risk for each level of added sugar intake.[35] 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 (those consuming <10% of their energy from added sugars) for each of the outcomes of interest.[35] Chi-square tests were used to test differences in categorical variables and Wald f-tests were used for continuous variables.

Evidence of a multicollinearity problem was observed (condition index =33 and variance decomposition proportion>0.5) in models that included waist circumference together with all other covariates, therefore waist circumference was dropped from the regression models. As the postprandial lipoprotein and insulin responses[36] have been shown to differ by level of adiposity, race, and sex, we tested for the presence of effect modification between added sugars intake (% total energy) and each of these variables by including a multiplicative term for each in the regression models. The effect of added sugars intake was shown to be modified by BMI in models with fasting insulin and HOMA-IR as the outcome (p=0.02 and 0.04, respectively). Therefore, further analyses of these measures were stratified by weight category, underweight (<5th BMI %ile), normal weight (5th–85th BMI%ile), and overweight/obese (BMI≥85th%ile).[37] No other outcome measures were modified by BMI nor was there any effect modification by race/ethnicity or sex for any of the outcome variables.

Sensitivity analysis was done to examine the association between intake of added sugars and HDL, triglyceride, 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 2 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 (% total energy) for each respondent was used together with the same covariates as 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, or poverty level. Similarly, no association was seen between the amount of added sugars consumed and physical activity.

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%, 22.2%) of total daily caloric intake (total energy). There was no significant difference between the mean added sugars consumption among non-Hispanic whites, 21.6 % of total energy, and consumption among other race/ethnic groups (not shown). The increased trend in % total energy from carbohydrates, and absolute intake of carbohydrates (g) with higher levels of added sugars intake was significant (p-linear trend<0.001 for both,) (Table 1). Intake of added sugars was negatively correlated with both the % total energy and the absolute intake (g) of: total fats, SFAs, PUFAs, MUFAs, and protein (p-linear trend<0.001 for all). Fiber and cholesterol intake (g) were also negatively correlated with intake of added sugars (p-linear trend<0.001).

In fully adjusted linear regression models we found that lipid levels among all adolescents tend to correlate with intake of added sugars (Figure 1). HDL levels were lower among those who consumed more added sugars (p-linear trend=0.007) (Table 2). Among the highest consumers (≥30% total energy) HDLs were 1.28 mmol/L (95% CI: 1.23, 1.33) (49.4 mg/dL) compared to 1.38 mmol/L (95% CI: 1.32, 1.43) (53.3 mg/dL) among the lowest consumer (<10% total energy), a difference of 7% (p=0.01). In contrast, geometric mean triglyceride levels and triglyceride to HDL ratios were higher among those consuming higher levels of added sugars (p-linear trend=0.03 and 0.02, respectively) (Table 2). Adjusted geometric mean triglyceride levels were 0.89 mmol/L (95% CI: 0.83, 0.96) (78.8 mg/dL0) and 0.79 mmol/L (95% CI 0.32, 0.86) (69.9 mg/dL) and mean triglyceride/HDL ratios (mg/dL) were 1.99 (95% CI: 1.65, 2.30) and 1.56 (95% CI: 1.75, 4.98) among the highest compared to the lowest consumers, respectively. This represents a difference of 13% in triglyceride levels (p=0.07) and 22% in the triglyceride/HDL ratio (p=0.04). There was no significant trend in LDL or in total cholesterol levels with intake of added sugars (p=0.10 and 0.29, respectively).

We found that the intake of added sugars and HOMA-IR measures were positively correlated among overweight adolescents (p-linear trend <0.001) but not among those who were normal weight (Figure 2). Adjusted mean HOMA-IR among the highest consumers was 5.04 (95% CI: 4.35, 5.73) compared to 2.03 (95% CI: 2.26, 3.40) among the lowest consumers, a difference of 78% (Table 2). A similar difference was observed with fasting insulin levels (Table 2).

Systolic blood pressure among all adolescents and BMI z-score among those overweight/obese tended to increase slightly with greater added sugar consumption but these trends did not reach significance (p=0.07 and 0.08, respectively) (Table 2). There was no significant trend in diastolic blood pressure among the adolescents studied. Waist circumference tended

to decrease with greater consumption of added sugars among the small sample of underweight adolescent in the study ($p<0.001$) but there was no significant trend among the normal or overweight adolescents (Table 2).

We repeated the analyses of the associations between intake of added sugars and HDL, triglyceride, and HOMA-IR levels with respondents grouped according to their intake of added sugars in grams. HDL results among the six evenly-sized groups who consumed: 0–<42.9, 42.9– <74.2, 74.2– <95.2, 95.2– <126, 126–≥162, and ≥162 grams of added sugars were very similar to those obtained in the main analyses using 6 groups categorized by % total energy from added sugars as the exposure. HDL levels were: 1.40 (95% CI: 1.34, 1.46), 1.34 (95% CI: 1.29, 1.38), 1.29 (95% CI: 1.24, 134), 1.31 (95% CI: 1.26, 1.36), 1.30 (95% CI: 1.24, 1.35), and 1.24 (95% CI: 1.19, 1.29) respectively (p-linear trend=0.001). Geometric means triglycerides were also higher among those whose absolute added sugars intake was higher, ranging 0.79 mmol/L (95% CI: 0.72, 0.88) among the lowest to 0.90 (95% CI: 0.83, 0.96) among the highest consumers though the trend was not significant (p-linear trend=0.24). HOMA-IRs were also greater with higher absolute intake of added sugar; means ranged from 2.63 mmol/L (95%CI: 1.84, 3.42) among the lowest to 5.24 mmol/L (95% CI: 4.21, 6.26) among the highest consumers (p-linear trend=0.002).

When the analysis was repeated again using the mean intake obtained from the smaller subsample of respondents who provided 2 24-hour dietary recalls (those participating in NHANES 2003–04) (n=669) point estimates and trends for HDL and HOMA-IR were similar to those obtained in the primary analyses but the positive trend in triglyceride levels with higher intakes of added sugars was not observed (p=0.50). Among the lowest vs. highest consumers: HDLs were 1.35 mmol/L (95% CI: 1.26, 1.44) and 1.43 (95% CI: 1.35, 1.51), respectively; HOMA-IRs among the overweight/obese were 3.19 (95% CI: 2.43, 3.95) and 4.97 (95% CI: 3.19, 6.74), respectively. Linear trends with higher consumption were p=0.01 for HDL; p=0.19 for LDL and p=0.06 for HOMA-IR.

DISCUSSION

In 1986, the Sugars Task Force of the Food and Drug Administration (FDA) 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.[38] Since then, the results of several new epidemiologic studies and short and long-term experimental studies have provided more evidence linking the intake of carbohydrates[39, 40] and sugars,[12, 13, 40, 41](particularly fructose),[12, 42, 43] 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 1977–78. The results of our study indicate that by 1999–2004 consumption among this group had risen to 119 g, an increase of 42%–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 triglycerides) among all adolescents 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 previously 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.[44] 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.[36]

Clearly, added sugars play a significant role in the U.S. diet. They increase the desirability of foods by increasing their sweetness. They also contribute substantially to energy intake without contributing other important nutrients to the diet.[45] Existing guidelines for limiting the consumption of added sugars vary widely. The Institute of Medicine (IOM) suggests a limit of 25% of total energy from added sugars in order to ensure adequate intake of important nutrients,[46] the World Health Organization (WHO) advises limiting added sugars to <10% total energy to prevent dental caries,[34] and recently released recommendations from the American Heart Association (AHA) advise that daily intake of added sugars should be limited to <100 calories daily for women and 150 calorie for men[44] (approximately 5% of total energy) as a strategy for preventing heart disease. The 2005 U.S. Dietary Guidelines for Americans encourage consumers to “choose and prepare foods and beverages with little added sugars or caloric sweeteners”[45] but do not specify an upper limit. While 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 risk.

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 lipid measures among U.S. adolescents. Second, we were able to control for several important confounding variables, including BMI and physical activity. Also, as 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 with-in 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.[47] In addition, when we evaluated those with 2 available 24 hour recalls,

our key findings remained consistent. While underreporting of certain foods high in sugars, such as sodas and sweets, may occur more frequently among those who underreport total energy,[48] such as those overweight or obese[49] who are also at increased risk of diabetes and dyslipidemia, systematic misclassification of this type would be expected to bias our findings toward the null.

In conclusion, higher consumption of added sugars among U.S. adolescents is associated with several important cardiometabolic 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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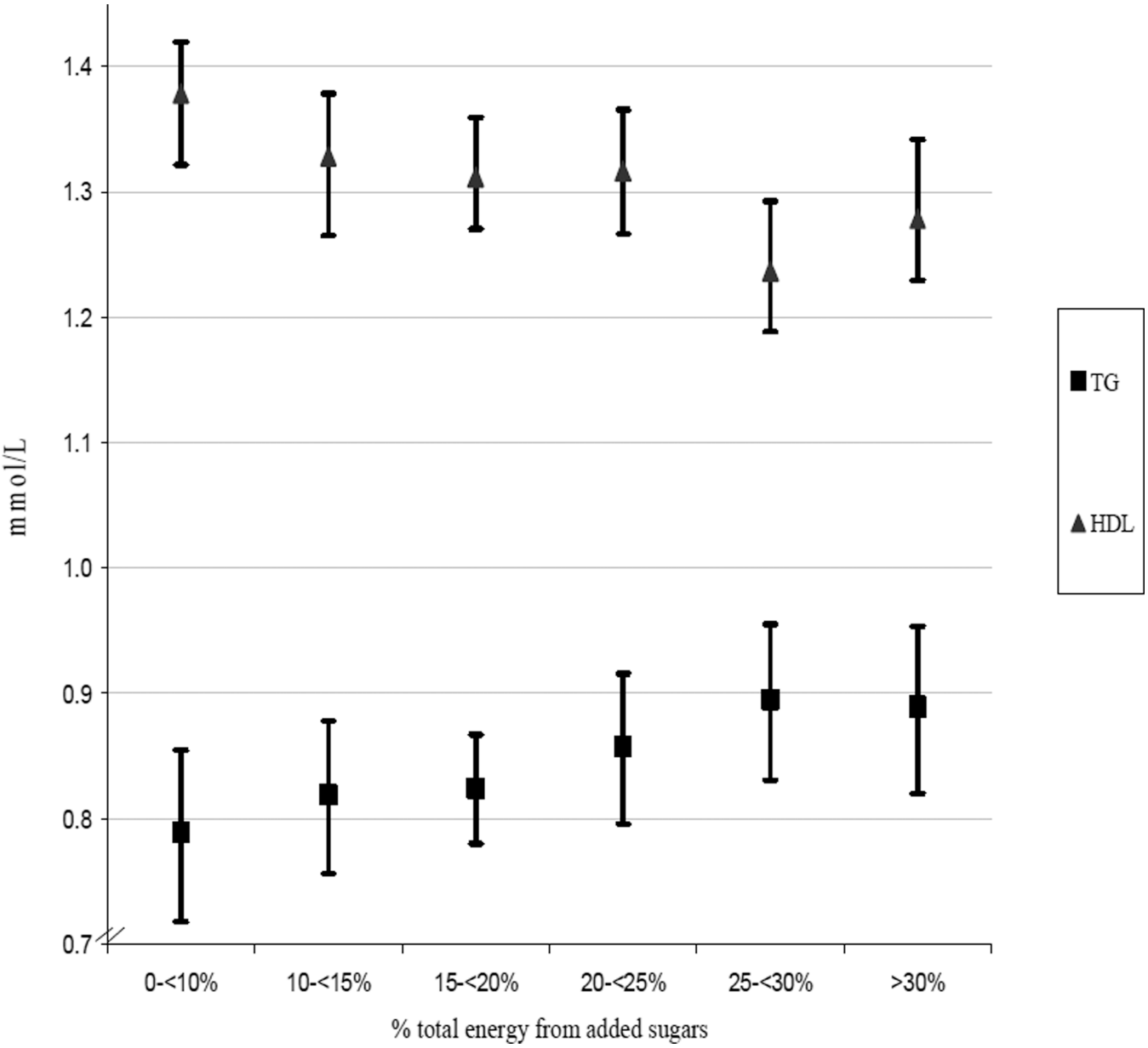

Figure 1.

Multivariable-adjusted mean triglyceride and high-density lipoprotein cholesterol (HDL) levels by intake of added sugars among US Adolescents. Participants grouped by percentage of total energy intake from added sugars; <10% comprises the referent group. P for linear trend=0.03 for triglyceride levels and <0.001 for HDL levels. Error bars indicate 95% confidence intervals. NHANES refers to National Health and Nutrition Examination Survey. To convert triglyceride values to mg/dL, multiple by 89. To convert HDL values to mg/dL, multiply by 39.

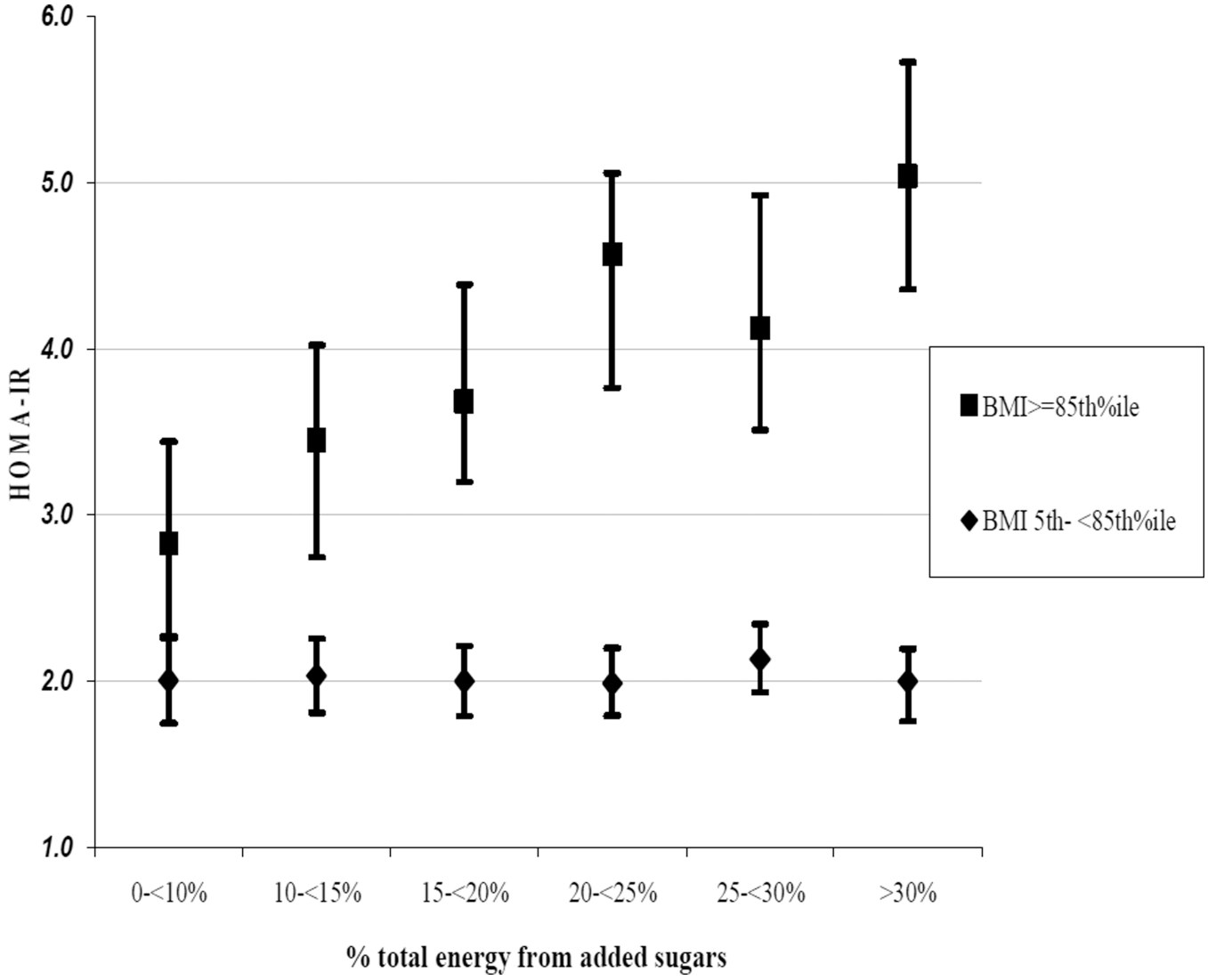

Figure 2.

Intake of added sugars and adjusted mean homeostasis model assessment for insulin resistance (HOMA-IR) by weight status. Participants grouped by percentage of total energy intake from added sugars; <10% comprises the referent group. Among overweight/obese adolescents, p for linear trend=0.03; among normal weight adolescents, p for linear trend=0.85. Error bars indicate 95% confidence intervals. NHANES refers to National Health and Nutrition Examination Survey.

Table 1

Demographic and Dietary Characteristics of US Adolescents (12 to 18 y) by Intake of Added Sugars, NHANES 1999–2004*

Characteristics	0 – <10% (n=318)	10 –<15% (n=377)	15 – <20% (n=440)	20 –< 25% (n=380)	25 – <30% (n=320)	≥30% (n=417)	p-linear trend
Demographic	mean or %SE	mean or % SE	mean or %SE	mean or %SE	mean or %SE	mean or %SE	
Age, mean years	14.8 ± 0.1	14.7 ± 0.1	15.1 ± 0.2	14.9 ± 0.1	15.2 ± 0.2	14.9 ± 0.2	0.11
Male, %	46.9% ± 3.3	50.0% ± 3.6	48.8% ± 3.3	53.5% ± 3.7	48.0% ± 3.3	47.4% ± 2.7	0.97
Race/ethnicity, %							
Non-Hispanice white, %	56.8% ± 4.1	64.7% ± 3.6	59.8% ± 2.9	67.3% ± 3.6	61.8% ± 2.4	0.623 ± 2.9	0.36
Non-Hispanice black, %	14.6% ± 2.5	13.2% ± 1.9	14.6% ± 1.8	14.5% ± 2.2	14.2% ± 1.2	0.141 ± 1.8	0.83
Hispanic, %	18.5% ± 5.4	18.1% ± 2.5	18.8% ± 2.6	13.1% ± 2.1	15.9% ± 1.8	0.145 ± 2.1	0.10
Other, %	10.1% ± 2.7	3.9% ± 1.4	6.8% ± 1.9	5.1% ± 1.6	6.0% ± 2.0	0.091 ± 2.4	0.82
Physical activity (leisure time), mean MET minutes/month	12508 ± 1448	13869 ± 972	11374 ± 1014	13765 ± 1522	13094 ± 1617	10409 ± 700	0.33
Income (below poverty)[e], %	28.0% ± 3.6	23.2% ± 2.6	24.0% ± 2.6	19.4% ± 2.8	25.9% ± 2.9	30.7% ± 3.4	0.19
Dietary							
Total calories, mean kcal/day	2057 ± 71	2302 ± 58	2342 ± 58	2357 ± 49	2289 ± 66	2068 ± 59	0.95
Carbohydrates							
total carbohydrates, % energy	46.9% ± 1	50.3% ± 0.6	52.8% ± 0.5	55.1% ± 0.7	57.5% ± 0.6	64.9% ± 0.6	<0.001
total carbohydrates, mean g	239 ± 7.8	286 ± 6.5	306 ± 8.2	324 ± 7.6	326 ± 9.2	333 ± 10	<0.001
-added sugar, mean g	308 ± 1.5	73.4 ± 2	103 ± 2.5	133 ± 3	157 ± 4.5	199 ± 7.9	<0.001
Fiber, mean g	14.9 ± 0.6	14.7 ± 0.4	15.1 ± 0.6	13.6 ± 0.5	11.7 ± 0.4	9.7 ± 0.3	<0.001
Protein							
total protein, % energy	17.0% ± 0.4	15.3% ± 0.3	14.0% ± 0.2	13.3% ± 0.3	12.4% ± 0.3	10.4% ± 0.2	<0.001
total protein, mean g	86.5 ± 3.5	88.3 ± 2.5	81.7 ± 2.3	78.3 ± 2.5	71.8 ± 3.2	53.5 ± 1.8	<0.001
Fats							
total fats, % total energy	36.3% ± 0.7	34.9% ± 0.6	33.8% ± 0.5	32.5% ± 0.6	31.2% ± 0.4	26.2% ± 0.4	<0.001
total fats, mean g	84.7 ± 4.1	90.1 ± 3.1	89.1 ± 2.5	85.4 ± 2.4	80.5 ± 2.9	61.3 ± 2.2	<0.001
-MUFAs, % energy	13.4% ± 0.3	13.2% ± 0.3	12.8% ± 0.2	12.4% ± 0.2	11.9% ± 0.2	9.9% ± 0.2	<0.001
-MUFAs, mean g	31.6 ± 1.5	34.1 ± 1.3	33.9 ± 1.0	32.4 ± 1.0	30.6 ± 1.1	23.3 ± 0.8	<0.001
-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	<0.001
-PUFAs, mean g	16.4 ± 1.1	17.7 ± 0.8	17.8 ± 0.8	16.6 ± 0.6	15.4 ± 0.7	12 ± 0.7	<0.001

Characteristics	0 – <10% (n=318)	10 –<15% (n=377)	15 – <20% (n=440)	20 –< 25% (n=380)	25 – <30% (n=320)	≥30% (n=417)	p-linear trend
-SFAs, % energy	12.8% ± 0.3	11.9% ± 0.2	11.6% ± 0.2	11.3% ± 0.3	10.9% ± 0.2	9.1% ± 0.2	<0.001
-SFAs, mean g	30.0 ± 1.6	31.2 ± 1.2	30.6 ± 0.9	29.8 ± 1.0	28.3 ± 1.2	21.4 ± 0.7	<0.001
Cholesterol intake, mean g	260 ± 17	286 ± 15	249 ± 8	252 ± 16	248 ± 23	168 ± 9.4	<0.001

Abbreviations: y=year; NHANES=National Health and Nutrition Examination Survey; kcal=kilocalories; %=percent; SE=standard error; % energy= % total enery intake; MUFAs=mono-unsaturated fatty acids; PUFAs=poly-unsaturated fatty acids; SFAs=saturated fatty acids

*All results are adjusted to account for the complex sampling method used by NHANES and weighted to be representative of the U.S. population

†Analysis of contrasts used to test trends using X^2 for categorical variables and Wald F tests for continuous variables

Table 2

Cardiometabolic Indicators Among Adolescents by Level of Added Sugars Intake, NHANES 1999–2004*

	% total energy from added sugars												
	0 – <10% (referent) (n=318)		10 – <15% (n=377)		15 – <20% (n=440)		20 – < 25% (n=380)		25 – <30% (n=320)		≥30% (n=417)		p-trend
Indicator	**mean**	**(95% CI)**	**mean**	**(95% CI)**	**mean**	**(95% CI)**	**mean**	**(95% CI)**	**mean**	**(95% CI)**	**mean**	**(95% CI)**	
Lipid measures (mmol/L)													
HDL cholesterol	1.38	(1.32, 1.43)	1.33	(1.27, 1.39)	1.31 [f]	(1.27, 1.35)	1.32 [§]	(1.27, 1.37)	1.24 [†]	(1.19, 1.28)	1.28 [‡]	(1.23, 1.33)	0.007
LDL cholesterol	2.26	(2.12, 2.39)	2.28	(2.16, 2.39)	2.38 [f]	(2.30, 1.34)	2.49 [§]	(2.35, 2.63)	2.40	(2.28, 2.52)	2.40	(2.27, 2.52)	0.10
Triglycerides[//]	0.79	−(0.32, 0.86)	0.82	−(0.27, 0.88)	0.82	(0.78, 0.87)	0.86	(0.80, 0.92)	0.90	(0.84, 0.96)	0.89	(0.83, 0.96)	0.03
Total cholesterol	4.04	(4.03, 4.20)	4.03	(3.91, 4.14)	4.11	(4.02, 4.20)	4.25 [§]	(4.09, 4.40)	4.09	(3.97, 4.22)	4.12	(3.99, 4.26)	0.29
TG/HDL (mg/dL)	1.56	(1.75, 4.98)	1.75	(1.51, 1.99)	1.73	(1.50, 1.96)	1.89 [§]	(1.61, 2.17)	2.13 [§]	(1.74, 2.53)	1.99 [§]	(1.68, 2.30)	0.02
Blood pressure (mmHg)													
systolic	109	(107, 111)	111	(108, 113)	111	(109, 112)	111	(109, 113)	113 [‡]	(111, 114)	112	(110, 114)	0.07
diastolic	62.7	(60.3, 65.1)	63.7	(62.3, 65.1)	61.6	(60.0, 63.2)	62.4	(60.8, 64.0)	66.2 [§]	(64.7, 67.8)	63.1	(61.1, 65.1)	0.54
Glucose (fasting) (mmol/L)	4.95	(4.87 5.04)	4.94	(4.85 5.04)	5.02	(4.94 5.11)	5.02	(4.91 5.12)	5.08	(4.99 5.16)	5.00	(4.91 5.10)	0.05
Insulin (fasting) (pmol/L)													
underweight[#]	57.1	(31.5, 82.8)	45.7	(18.4, 73.2)	64.1	(57.6, 70.5)	68.8	(61.3, 76.3)	50.2	(38.7, 61.7)	21.5	−(1.8, 44.8)	0.58
normal weight[#]	59.7	(52.7, 66.7)	59.3	(52.0, 66.6)	60.3	(55.6, 65.0)	62.3	(57.6, 67.1)	59.5	(58.8, 60.2)	61.7	(53.3, 70.2)	0.84
overweight/obese[#]	79.0	(62.6, 95.4)	90.5	(69.9, 111)	101 [e]	(90.3, 113)	130 [†]	(110, 151)	111 [‡]	(94.5, 128)	139 [†]	(120, 157)	<0.001
HOMA-IR													
underweight	2.13	(1.88, 2.38)	1.62	(1.58, 1.66)	1.77	(1.52, 2.03)	2.36	(2.29, 2.42)	1.81	(1.74, 1.88)	0.80	(0.71, 0.90)	<0.001
normal weight	2.01	(1.74, 2.27)	2.03	(1.81, 2.26)	2.00	(1.79, 2.21)	1.99	(1.79, 2.18)	2.13	(1.93, 2.33)	2.00	(1.76, 2.24)	0.85
overweight/obese	2.83	(2.26, 3.40)	3.45	(2.75, 4.15)	3.68 [e]	(3.20, 4.17)	4.57 [†]	(3.76, 5.37)	4.12 [‡]	(3.51, 4.74)	5.04 [†]	(4.35, 5.73)	<0.001
BMI (z-score)													
underweight	−1.67	−(2.66, −0.68)	−1.71	−(2.14, −1.28)	−1.96	−(2.46, −1.45)	−1.94	−(2.40, −1.47)	−2.46	−(2.81, −2.12)	−2.47	−(2.7, −2.24)	0.12
normal weight	0.04	−(0.16, 0.25)	0.11	−(0.08, 0.31)	−0.02	−(0.21, 0.18)	0.04	−(0.14, 0.23)	−0.19 [§]	−(0.38, −0.01)	−0.03	−(0.25, 0.18)	0.11
overweight	1.63	(1.49, 1.77)	1.77	(1.63, 1.91)	1.64	(1.53, 1.75)	1.72	(1.6, 1.84)	1.72	(1.60, 1.85)	1.89 [§]	(1.7, 2.03)	0.08
Waist circumference (cm)													

	% total energy from added sugars												
	0 – <10% (referent) (n=318)		10 – <15% (n=377)		15 – <20% (n=440)		20 – < 25% (n=380)		25 – <30% (n=320)		≥30% (n=417)		p-trend
Indicator	**mean**	**(95% CI)**	**mean**	**(95% CI)**	**mean**	**(95% CI)**	**mean**	**(95% CI)**	**mean**	**(95% CI)**	**mean**	**(95% CI)**	
underweight	65.3	(64.5 66.1)	67.2	(66.4 68.1)	66.5	(65.6 67.3)	67.5	(67.2 67.8)	62.4	(61.0 63.8)	64.7	(63.8 65.6)	0.01
normal weight	75.1	(73.4 76.8)	76.4	(75.4 77.5)	75.4	(74.3 76.5)	75.1	(74.1 76.1)	73.7	(72.4 75.0)	74.9	(73.5 76.3)	0.19
overweight	95.3	(91.6 98.9)	99.6	(95.7 104)	95.3	(92.8 97.9)	99.0	(95.5 103)	98.4	(95.2 102)	101 §	(97.2 105)	0.11

Abbreviations: NHANES=National Health and Nutrition Examination Survey; %=percent; HDL=high-density lipoprotein cholesterol; LDL=low-density lipoprotein cholesterol; TG=triglycerides; BP=blood pressure; HOMA-IR=homeostatis model assessment of insulin resistance;BMI=body-mass-index

*All means are adjusted for age; sex; race; bmi percentile (excluding model with BMI as outcome); physical activity; total energy intake; nutrient residuals for intake of: fats (mono-unsaturated fatty acids, poly- PUFAs, and saturated), protein, fiber, cholesterol, and other carbohydrates (excluding fiber and added sugar). Results are weighted to represent the US population and adjusted to account for complex sampling methods used. To covert mmol/L to mg/dL, multiply by 39 for HDL and LDL; multiply by 89 for triglycerides; multiply by 38.67 for total cholesterol; and multiply by 18 for glucose.

†,‡,§Mean values significantly different from the referent (10% total energy from added sugars),

†p<0.001;

‡p<0.01;

§p<0.05.

//Geometric mean triglycerides are presented.

#Under weight=<5th age-and sex-adjusted BMI %ile (n=44). Sub-sample size by level of added sugar intake: 0–<10% n=3; 10–<15% n=5; 15–<20% n=10; 20–<25% n=13; 25–<30% n=5; ≥30% n=8; normal weight=5th–<85th age- and sex- adjusted BMI %ile (n=1348). Sub-sample size by level of added sugar intake: 0–<10% n=179; 10–<15% n=225; 15–<20% n=283; 20–<25% n=231; 25–<30% n=179; >30% n=251; overweight/obese–>=95th age-and sex-adjusted BMI %ile (n=844). Sub-sample size by level of added sugar intake: 0–<10% n=131; 10–<15% n=147; 15–<20% n=142; 20–<25% n=135; 25–<30% n=133; >30% n=156