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DIETETIC PRACTICE PROJECTS

Dietary Sugars Predict Chronic Disease Risk Factors in College Students

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This study examines the impact of dietary sugar components on risk factors for chronic diseases in college students (n = 261). Mean consumption of the percentage of kilocalories from total and added sugars was 24% and 17%, respectively. Participants consumed 1.1 servings sugar sweetened beverages (SSB) and 28-gram total fructose daily. All sugar components predicted lower high-density lipoprotein cholesterol, and total sugars and SSB predicted higher low-density lipoprotein cholesterol. Fructose intake predicted higher fasting hunger and SSB predicted higher blood glucose. Since consumption of dietary sugars predicts chronic disease risk and consumption of fructose predicts appetite, clinical interventions should include reduction of dietary sugars.
Key words: *cardiovascular diseases*, *chronic disease*, *diabetes mellitus type 2*, *risk factors*, *universities*, *young adults*

DIETARY sugars are under increased scrutiny by health professionals because they are positively associated with body weight,[1,2] type 2 diabetes mellitus (T2D)[3-5] and cardiovascular disease (CVD) risk factors.[6-8] Despite the recent recommendation by the American Heart Association to limit intake of added sugars to no more than 5% of the total kilocalories,[9] 40% of the young adults in college consume more than double this amount.[10]

College students are an important population to investigate because the transition from adolescence to adulthood comes with an important shift in responsibility for food choices. Most college students leave the controlled food environment their parents provided at home and begin making their own choices in a buffet-style dining hall with unlimited food availability. This sudden food independence, along with the heavy caloric load of binge drinking (44% of the college students),[11] creates an environment where college students gain weight about 6 times faster than the general population.[12]

In college, these young adults, defined here as ages 18 to 24, are transitioning from adolescence to adulthood. Weight gain during this time can result in dyslipidemia,[13] including low high-density lipoprotein cholesterol (HDL-C), elevated low-density lipoprotein cholesterol (LDL-C), elevated

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triacylglycerol (TG) and elevated glucose. Dyslipidemia tends to track into adulthood[14] and can predict future risk of CVD and diabetes.[15] Intake of added sugars is positively associated with LDL-C and TG and negatively associated with HDL-C in adolescents,[16] but little is known about the impact of college life on these risk factors. To our knowledge, the relationship between intake of dietary sugars and risk factors for chronic disease remains unexamined in college students.

While the association between dietary sugars and risk for disease is strong, the mechanism for this association and the specific sugar components through which it acts remains unknown. A possible link includes an effect of dietary sugars on appetite and weight regulation.[9,17-20] Obese young adults (19-30 years) consume significantly more fructose than normal-weight young adults, 11.9% of total kilocalories compared with 10.5% of kilocalories, respectively.[21] Fructose may cause appetite dysregulation through insufficient stimulation of satiety hormones such as leptin and inadequate suppression of the hunger hormone, ghrelin.[19,22] Consumption of fructose-sweetened beverages is shown to increase visceral adiposity and other risk factors for T2D and CVD in overweight/obese adults, which is believed to be caused partly by increased hepatic de novo lipogenesis.[23]

Research in animals indicates that increased sugar consumption may impact hunger and satiety signals, thus influencing energy intake and weight gain.[24] The hunger and satiety hormones (leptin and ghrelin) studied in rats also serve similar functions in humans and may be impacted similarly by intake of dietary sugars.

Sugar sweetened beverages (SSB) are the largest source of fructose in Americans' diets, accounting for 30% of the total fructose intake.[21] The high consumption of SSB by children is well described and increases with age.[25] The percentage of kilocalories from SSB rises from 7% to 9% to 13% in children aged 2 to 5, 6 to 11, and 12 to 19 years, respectively.[25] Intake of SSB in college students was examined in only 1 study at the University of Arkansas and SSB accounted for 553 kcals/day.[26] While this study did not examine total dietary intake,[26] given the high amount of kilocalories consumed per day, the college population may have even higher intakes than the 13% of kilocalories seen in 12- to 19-year-olds. More information is needed to fully examine the impact of SSB in the context of the overall diet. The relationship of SSB to chronic disease risk also needs to be studied in young adults, since SSB consumption predicts T2D in 40- to 60-year-old men and women[5] and is positively associated with CVD risk in adult women aged 34 to 59 years.[27]

The aim of this project was to examine the impact of dietary sugar components on risk factors for obesity, CVD, and T2D in young adult college students. Our primary hypothesis was that percentage of kilocalories from added sugars, SSB, and grams of fructose would be positively associated with TG and LDL-C and negatively associated with HDL-C, while total dietary sugars would not share these associations. Our secondary hypothesis was that greater fructose intake would be associated with greater fasting hunger and lower satiety.

METHODS

Human participants

The institutional review board at the University of Rhode Island (URI) approved this study, and written informed consent was obtained from all volunteers. Participant recruitment occurred via advertisement in the school newspaper, flyers, recruitment tables, and class presentations. Exclusionary criteria included pregnancy, lactation, diabetes (type 1 or type 2), cancer, coronary heart disease, liver disease, a bleeding disorder, or use of lipid-lowering medications. Of the 294 participants who signed consents, 33 were unwilling or unable to complete the study, leaving a final sample size of 261. Reasons for dropout included participating in a weight loss study

(n = 2), lack of time or interest (n = 12), illness or medication use (n = 6), discomfort with blood draws (n = 4), lack of response to study contacts (n = 6), dropped a course offering extra credit for a research project (n = 1), and scheduling difficulties (n = 2).

Study design

The Nutrition Assessment and Chronic Disease Risk Factor Identification Study was a cross-sectional study of first year students performed at URI during 4 school semesters from 2008 to 2009. After signing the consent form, participants completed their first 24-hour recall (24HR) in person in the laboratory. They returned to the laboratory for additional assessment measures on 2 nonconsecutive weekdays, and were contacted by phone for the last two 24HR during the course of the semester. Research evaluating the efficacy of phone dietary recall collection shows that it is a valid, feasible, and reliable method for collecting dietary data,[28] and the technique has been utilized effectively in nationally representative surveys.[29]

When participants completed all measurements and visits described below, they were compensated for their time with $30 and given feedback that included their lipid values, anthropometrics, mean total kilocalorie intake, and percentage of kilocalories from carbohydrate, fat, and protein.

Dietary analysis

Participants completed 3 nonconsecutive 24HR using the multiple pass system in conjunction with the Nutrition Data System for Research (University of Minnesota, Minneapolis, Minnesota). During the first in-person recall, participants used food models (eNasco, Inc, Fort Atkinson, WI), measuring cups and spoons and a food amounts booklet to help estimate portion sizes. Participants were provided with a food amounts booklet to assist with portion size estimation during the 2 subsequent 24HR. The mean of the 3 days was calculated to determine usual intake.

This study examined 4 interrelated but distinct components of dietary sugars, which together will be referred to as dietary sugar components. Three of these components are percentage of kilocalories from total sugar, percentage of kilocalories from added sugar, and fructose in grams, all taken directly from Nutrition Data System for Research. Total sugars include all glucose, fructose, galactose, sucrose, lactose, and maltose, while added sugars include the following ingredients: white sugar (sucrose), brown sugar, powdered sugar, honey, molasses, pancake syrup, corn syrups, high fructose corn syrups, invert sugar, invert syrup, malt extract, malt syrup, fructose, glucose (dextrose), galactose, and lactose. The fourth component, servings of SSB, is a composite component made up of sugar-sweetened iced tea, soft drinks, and sweetened fruit drinks, and does not include 100% fruit juice. Total and added sugars are examined in percentage of kilocalories to control for caloric need variation, but information about SSB was only available in servings in the nutrient database, so they are defined as such. Fructose was examined in grams because many studies examining the physiological effects of fructose study the effect of certain gram amounts of fructose on the body.[30-33] The accuracy of reporting was assessed using the procedure outlined by McCrory et al.[34] Using the predicted total energy expenditure to reported energy intake ratio, inaccurate reporters were considered as those above or below 2 standard deviations from the mean. Using these guidelines, those reporting less than 44% or greater than 156% of estimated energy needs were considered inaccurate reporters.[34] Of the 261 students in the study, 92.4% were considered accurate reporters, with 7.6% (n = 20) underreporting and 0.03% (n = 1) overreporting.

Anthropometric and clinical measurements

Study personnel measured participant height with a Seca 220 stadiometer (Seca Corporation, Hamburg, Germany), measured weight using a calibrated Seca digital 769 scale (Seca Corporation, Hamburg, Germany), and

measured waist circumference at the top of the iliac crest upon normal exhalation using a Gulick fiberglass (Patterson Medical, Mount Joy, PA), non-stretchable tape measure with an attached tensometer. If the 2 measurements taken were not within 0.2 kg in weight, 0.2 cm in height or 2 mm in waist circumference, measurements were repeated until consistent measurements were obtained. Body mass index (BMI) was calculated (weight in kilograms/height in meters^2), with normal weight defined as a BMI of 18.5 to 24.9 kg/m^2, overweight 25 to 29.9 kg/m^2 and obese 30 kg/m^2 or more. Trained kinesiology and nutrition graduate students took blood pressure measurements after the participant rested for 5 minutes using a Select stethoscope (Littman, St Paul, MN) and sphygmomanometer (Welch-Allyn, Skaneateles Falls, NY). The mean of 2 measurements taken 1 minute apart was used for analysis.

The following surveys were self-administered: a health history survey, the Weight Related Eating Questionnaire,[35] the International Physical Activity Questionnaire (short form),[36] and a visual analog scale (VAS) to rate appetite components (described below).[37]

Appetite

Participants rated appetite on a 100 mm visual analog scale after a 12-hour fast, by responding to 4 questions: (1) How hungry are you right now? (2) How satisfied (satiated) are you right now? (3) How much could you eat right now? (4) How thirsty are you right now? The scales were anchored with statements such as "not at all" and "extremely." Visual analog scales are valid and reliable instruments to assess subjective appetite.[37]

Blood sampling and lipid/lipoprotein analyses

Two 12-hour fasting blood draws were performed on nonconsecutive days to account for normal variance in blood lipids. Blood samples (~50 mL) were collected into BD vacutainers containing ethylenediaminetetraacetic acid (BD, Franklin Lakes, NJ) in the morning. Immediately upon collection, whole blood was centrifuged (Centrifuge 5810R; Eppendorf North America, Westbury, NY) at 1500 rpm for 20 minutes at 4°C to separate plasma and serum. A preservation cocktail of 0.1 mL of phenyl methyl sulfonil fluoride (Roche, Indianapolis, IN) per 100 mL of plasma, 0.1 mL of sodium azide (Fisher, Fairlawn, NJ) per 100 mL of plasma, and 0.5 mL of aprotinin per 100 mL of plasma (Fisher, Fairlawn, NJ) were added. Samples were aliquoted into separate 500 μL labeled microcentrifuge tubes and stored at –80°C until analysis.

Enzymatic colorimetric assays were performed on plasma to measure total cholesterol (TC), TG (Roche/Hitachi Chol and Trig/GB kits; Roche/Hitachi, Indianapolis, IN) and glucose (Autokit glucose; Wako Diagnostics, Richmond, VA). Samples were analyzed using a Biotek Reader (ELx808 Absorbance Microplate Reader; Biotek, Winooski, VT). High-density lipoprotein cholesterol was measured (Roche Diagnostics - USA standards and kits) after precipitation of the apo-B containing lipoproteins using a dextran sulfate and magnesium chloride solution (Across Organics, Morris Plains, NJ). The mean of the 2 days was computed for TC, HDL-C, and TG and used for analysis. Low-density lipoprotein-cholesterol was calculated using the Friedewald equation.[38] A sample from 1 day was used to obtain glucose concentrations.

Statistical analysis

All analyses were conducted using SPSS (version 19.0, IBM Corp, Summers, NY). Continuous data are presented as mean $\pm$ standard deviation and categorical data as percentages. To ensure normality, skewness and kurtosis were examined, and when not normally distributed, outliers more than 2 standard deviations from the mean were removed: waist circumference (n = 2), BMI (n = 5), LDL-C (n = 3), TG (n = 3), and SSB (n = 6).

Once normality was achieved, partial Pearson correlations were performed to examine the associations between sugar components and CVD risk factors and appetite.

Since significant differences were found between genders and BMI in sugar components, and both are known to be associated with CVD and T2D risk,[39] they were used as covariates. Percentage of kilocalories from carbohydrate was also used as a covariate so that intake would not influence the relationship seen between sugar intake and chronic disease risk factors. To further examine these relationships, multiple linear regression analyses were used to test a model for predicting CVD risk factors and appetite rating scores from intake of dietary sugars. Body mass index, gender, and percentage of kilocalories from carbohydrate were again included as covariates.

FINDINGS

Sample description

The University of Rhode Island is a medium-sized university in the Northeastern United States. Continuous descriptive data were presented in mean ± SD and categorical data in percentages (Table 1). Of the participants who completed the study, the majority were 18-year-olds (73.9%), did not use tobacco (88.1%), and lived on campus (87%). The participants were mostly normal weight (73.2%), with 3.8% classified as underweight, 17.2% overweight, and 5.7% as obese. The student body, mainly Caucasian (74.0%), is reflected in our sample; 82.0% were Caucasian, 4.6% Asian, 4.6% African American, and 8.8% other races or racially mixed.

Participants consumed a mean of about 24% of their daily kilocalories from total sugars, and 17% of daily kilocalories from added sugars (Table 1). They drank a mean of 1 serving of SSB and consumed 28 grams of fructose per day. Their mean daily intake was close to 2100 kilocalories, with 53% coming from carbohydrates, 15% from protein, and 31% from fat.

Sugars and CVD Risk factors and appetite

All sugar components were negatively correlated with HDL-C ($P < .05$) and all but fructose were positively correlated with TC:HDL-C ratio (Table 2). Percentage of kilocalories from total sugars and SSB were positively associated with LDL-C, and only SSB were positively associated with blood glucose. Fructose was positively associated with fasting appetite and added sugars negatively correlated with fasting thirst. Using Cohen's *d*, all correlation coefficients (*r*) were small to moderate.[40]

Each of the predictor variables had a significant ($P < .05$) partial effect on HDL-C in the model, and all but fructose had a partial effect on TC:HDL-C ratio. Both percentage of kilocalories from total sugars and SSB had partial effects on LDL-C. Only SSB had a partial effect on blood glucose. Only fructose had a partial effect on appetite, hunger, and diastolic blood pressure. According to Cohen's interpretation, all effects were small to medium[40] (Table 3).

DISCUSSION

This study was the first to examine the ability of dietary sugar components to predict chronic disease risk and hunger and appetite in college students. The primary finding was that intake of all examined sugar components were associated with and can predict HDL-C, an independent predictor of CVD.[15,41] In addition, higher fructose intake was the only component that predicted higher fasted hunger and appetite.

Students at URI consumed a lower percentage of their kilocalories from added sugar compared with the general US adolescent population (aged 12-18 years), as estimated by Welsh et al,[16] at 16.8% and 21.4%, respectively.[42] The amount of SSB URI first year students consumed (1.1 servings = ~95 kcals) was also lower than that consumed by the only other college sample currently available, students at the University of Arkansas (553 kcals).[26] The higher percentage of African Americans at the University of Arkansas than at URI (35% compared with 4.6%) and the higher intake of SSB seen in African Americans[26] may explain part of the

Table 1. Characteristics of Nutrition Assessment Study Participants[a]

		Dietary Intake	
Male	32.6%	Total kcals	2095.2 ± 726.4
Female	67.4%	Carbohydrate, % kcals	52.7 ± 7.3
Age, y	18.3 ± 0.5		
Height, cm	168.1 ± 8.8	Protein, % kcals	15.2 ± 3.6
Weight, kg	66.0 ± 13.2	Fat, % kcals	31.3 ± 5.9
Body mass index, kg/m^2	23.0 ± 3.2	Total sugars, g	123.7 ± 47.0
Waist circumference, cm	77.8 ± 8.0	Total sugars, % kcals	24.2 ± 6.9
TC, mmol/L	3.8 ± 0.7	Added sugars, g	86.0 ± 39.1
LDL-C, mmol/L	1.9 ± 0.6	Added sugars, % kcals	16.8 ± 6.5
HDL-C, mmol/L	1.5 ± 0.4	Sucrose, g	48.4 ± 22.9
TC:HDL-C ratio	2.7 ± 0.7	Fructose, g	27.6 ± 14.6
Triacylglycerol, mmol/L	1.0 ± 0.5	Fruit, servings	0.78 ± 0.96
Glucose, mmol/L	4.9 ± 0.5	Dietary fiber, g	16.0 ± 6.6
SBP, mm Hg	105.0 ± 10.6	SSB, servings	1.1 ± 1.1
DBP, mm Hg	66.8 ± 7.4	Soft drinks, servings	0.5 ± 0.68
VAS 1: hunger	45.9 ± 24.0	Alcohol intake, g	40.4 ± 12.91
VAS 2: satiety	45.7 ± 19.0	**Physical Activity**	
VAS 3: appetite	49.9 ± 19.9	Met/min/wk	2209.6 ± 1304.7
VAS 4: thirst	56.4 ± 22.7		

Abbreviations: DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; Met/min/wk, metabolic equivalents, in minutes per week; SBP, systolic blood pressure; SSB, sugar sweetened beverages; TC, total cholesterol; VAS, visual analog scale.
[a]Values are described as mean ± SD, or percentages; N = 261.

difference in intakes at the 2 universities. Although the intake of SSB and added sugar reported by URI first year students might be lower, it is of concern. A similar intake of SSB (≥1 SSB/day) in the Nurse's Health Study II was strongly associated with risk of T2D (Risk ratio = 1.98, 95% confidence interval = 1.60-2.44).[3] Prevention of T2D in young adults is important; the estimated direct and indirect costs of T2D in the United States in 2002 were $132 billion.[43] The prevalence of T2D has increased since 2002,[44] making the costs even higher today.

Similar to findings from cross-sectional and prospective cohort studies examining SSB and added sugars, dietary sugar components consistently predicted concentrations of HDL-C.[7,16,45,46] High-density lipoprotein cholesterol is an inversely correlated, independent risk factor for CVD.[15,41] Evidence from randomized controlled trials shows that a 1%-increase in HDL-C is associated with a 3%-decrease in CVD risk,[41] and current research implies that HDL-C has direct effects on atherogenesis.[15]

Fructose intake (27.6 g) was reported to be much lower than intakes seen in national surveys (54.7 g).[21] Regardless of the low reported levels of intake, fructose intake still emerged as a significant predictor of hunger and appetite after fasting. Chronic fructose consumption is postulated to increase energy intake by activating neural reward pathways (opioids and dopamine in the pleasure center of the brain)[19] and by promoting feelings of hunger by failing to suppress ghrelin, a hunger-inducing hormone.[19,22] These reward pathways and hormonal responses to chronic consumption of fructose may be why only fructose, of the 4 examined components, predicted hunger and appetite. A positive relationship between intake of fructose and TG is often found,[31,45,47] but did not occur in these participants. This may be due to the majority of the sample being female, as a prior study

Table 2. Correlations of Dietary Sugar Components and Risk Factors for Chronic Disease and Appetite[a]

	Total Sugars % kcals	Added Sugars % kcals	Fructose, g	SSB, Servings
Waist circumference, cm				
r	–0.04	–0.02	–0.03	0.03
P	.59	.75	.66	.66
TC, mmol/L				
r	0.10	0.02	–0.05	0.08
P	.10	.78	.45	.23
LDL-C, mmol/L				
r	0.18[b]	0.11	0.02	0.14[b]
P	.006	.09	.79	.025
HDL-C, mmol/L				
r	–0.13[b]	–0.14[b]	–0.13[b]	–0.15[b]
P	.036	.028	.048	.019
TC:HDL-C ratio				
r	0.20[b]	0.14[b]	0.10	0.21[b]
P	.002	.033	.13	.001
Triacylglycerol, mmol/L				
r	0.00	–0.05	–0.03	0.04
P	.96	.44	.69	.57
Glucose, mmol/L				
r	0.03	0.06	0.01	0.15[b]
P	.69	.33	.86	.018
SBP, mm Hg				
r	–0.05	–0.09	–0.04	0.04
P	.40	.18	.56	.52
DBP, mm Hg				
r	–0.05	0.07	–0.08	–0.09
P	.41	.27	.21	.15
VAS 1: hunger				
r	–0.01	–0.02	0.11	0.02
P	.87	.74	.10	.70
VAS 2: satiety				
r	–0.02	0.00	–0.12	0.00
P	.77	.99	.05	1.00
VAS 3: appetite				
r	0.03	0.04	0.20[b]	0.10
P	.60	.50	.002	.11
VAS 4: thirst				
r	–0.07	–0.13[b]	–0.02	–0.07
P	.26	.048	.75	.26

Abbreviations: DBP, diastolic blood pressure; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; TC, total cholesterol; VAS, visual analog scale.

[a]Values indicate partial correlations controlling for BMI, gender, and % kilocalories from carbohydrate; N = 261.

[b]Significant ($P < .05$); correlation coefficients (r): large = 0.5, moderate = 0.3, and small = 0.1.[40]

Table 3. Predictive Value of Dietary Sugar Elements and CVD Risk Factors; Biochemical, Clinical, and Anthropometric[a]

	Total Sugars, % kcals				Fructose, g			
	β	***t***	***P***	**Effect**	*β*	***t***	***P***	**Effect**
BMI, kg/m^2	–0.06	1.05	.30	0.00	–0.01	0.10	.92	0.00
Waist circumference, cm	–0.12	1.87	.06	0.01	–0.05	1.13	.26	0.00
Total cholesterol, mmol/L	0.09	1.50	.14	0.01	–0.05	0.73	.47	0.00
LDL cholesterol, mmol/L	0.13[b]	2.10	.037	0.02	0.01	0.19	.85	0.00
HDL cholesterol, mmol/L	0.18[b]	2.87	<.001	0.03	0.14[b]	2.25	.025	0.02
TC:HDL-C ratio	0.23[b]	3.70	<.001	0.05	0.10	1.60	.11	0.01
Triacylglycerol, mmol/L	0.07	1.08	.28	0.00	–0.01	0.12	.91	0.00
Glucose, mmol/L	–0.03	0.47	.64	0.00	–0.03	0.52	.61	0.00
SBP, mmHg	–0.09	1.51	.13	0.01	–0.06	1.04	.30	0.00
DBP, mmHg	–0.07	1.06	.29	0.00	0.10[b]	3.36	<.001	0.04
VAS 1: hunger	0.04	0.58	.56	0.00	0.15[b]	2.27	.024	0.02
VAS 2: satiety	0.04	0.67	.50	0.00	–0.08	1.27	.20	0.01
VAS 3: appetite	0.02	0.33	.74	0.00	0.21[b]	3.29	.001	0.04
VAS 4: thirst	–0.09	1.35	.18	0.01	–0.03	0.44	.66	0.00
	Added Sugars, % kcals				**SSB, servings**			
	β	***t***	***P***	**Effect**	*β*	***t***	***P***	**Effect**
BMI, kg/m^2	–0.07	0.89	.37	0.00	–0.07	1.19	.23	0.01
Waist circumference, cm	–0.09	1.51	.13	0.01	0.02	0.55	.58	0.00
TC, mmol/L	0.01	0.16	.88	0.00	0.05	0.82	.41	0.00
LDL-C, mmol/L	0.10	1.57	.12	0.01	0.13[b]	2.09	.038	0.02
HDL-C, mmol/L	0.17[b]	2.78	.006	0.03	0.16[b]	2.71	.007	0.03
TC:HDL-C ratio	0.16[b]	2.49	.013	0.02	0.20[b]	3.24	<.001	0.04
Triacylglycerol, mmol/L	–0.02	0.30	.77	0.00	0.02	0.39	.70	0.00
Glucose, mmol/L	0.02	0.34	.74	0.00	0.15[b]	2.44	.016	0.02
SBP, mm Hg	–0.10	1.71	.09	0.01	0.04	0.72	.47	0.00
DBP, mm Hg	–0.10	1.56	.12	0.01	–0.10	1.73	.09	0.01
VAS 1: hunger	0.02	0.37	.71	0.00	0.03	0.44	.66	0.00
VAS 2: satiety	0.03	0.51	.61	0.00	0.02	0.24	.81	0.00
VAS 3: appetite	0.05	0.75	.46	0.00	0.10	1.57	.12	0.01
VAS 4: thirst	–0.12	1.87	.06	0.01	–0.05	0.81	.42	0.00

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; SSB, sugar sweetened beverages; TC, total cholesterol; VAS, visual analog scale.

[a]Values show results of a multiple regression analysis which includes gender, BMI, and % kilocalories in the model, N = 261.

[b]Significant ($P < .05$); Effect size = semi partial correlation; small = 0.01, medium = 0.06, large = 0.13.[40]

showed a dulled metabolic effect of fructose consumption in females, ascribed to differences in sex hormones and fat mass at comparable BMI to males.[31]

Chronic consumption of foods such as SSB, with high amounts of fructose, can cause insulin resistance, an underlying cause of T2D,[15,19] and intake of SSB was a significant predictor of blood glucose concentrations, a risk factor for both CVD and T2D.[48] The reason why SSB emerged as the only measured component to predict

blood glucose is unclear, but warrants further investigation.

Strengths of this study include the dietary methodology, which is more robust than most studies that examine intake of dietary sugars[5,8,10,26,46] and gives information on the total diet, not just dietary sugar components. As full dietary data and anthropometric measurements were available, all analyses were able to control for both total carbohydrate consumption, which could mask the effect of the dietary sugars, and BMI, which is known to negatively affect lipoprotein metabolism.[39]

This study was unique because it was the first to examine the intake of these 4 dietary sugar components in college students, and their associations with chronic disease risk and appetite. It expanded upon the work done by West et al[26] and Song et al[10] to characterize SSB and added sugar intake in college students, and extended some of the findings of Welsh et al[18] in adolescents to college students.

The effect of added sugars on HDL-C and LDL-C is currently unclear[9] and this study increases the evidence supporting a damaging effect of added sugars on CVD risk factors. While this study gives evidence to support the hypothesis that SSB are positively associated with chronic disease risk factors, it does not support a positive association between SSB and obesity, a relationship that remains controversial.[3,49,50] Future research should investigate the effect of nutrition interventions aiming to reduce intake of total and added sugars, SSB and fructose on risk factors for chronic disease in college students.

Limitations

This is a cross-sectional study, so no causal relationships can be drawn. A second consideration is the predominantly Caucasian and female sample, so the ability to generalize our results to populations with different ethnic or gender compositions is limited. Since URI students consume comparatively less added sugars and SSB than most young adults,[16,26] those risk factors associated with higher levels of intake may have been missed. The reported intake of dietary sugars may be lower than actual intake, as most participants were underage and may not have reported SSB consumed with alcohol.

IMPLICATIONS FOR DIETETICS PRACTICE

Dietary sugar components are predictive of chronic disease risk factors in young adult college students, especially HDL-C.[51] Since these components predict risk factors for disease, it is important for dietitians to assess the contribution of dietary sugars in their clients' diets. If this contribution is larger than recommended (more than 5% of the total kilocalories),[9] dietitians should translate their knowledge into recommendations that can reduce this impact. Dietitians should encourage consumption of nutrient dense foods lower in dietary sugars and discourage consumption of SSB and other foods high in fructose or added sugar. Specific, realistic recommendations for replacing typically consumed desserts and SSB with healthful, whole food alternatives should be tailored to the individual's habits, preferences, and socioeconomic status.

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