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Predicting Body Fat and Blood Lipids with Sugars Intake

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PREDICTING BODY FAT AND BLOOD LIPIDS WITH SUGARS INTAKE

BY

ERIC NELSON

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE

REQUIREMENTS FOR THE DEGREE OF

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OF
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2016

ABSTRACT

Objective: The primary objective of this study was to determine if caloric intake of fructose sugars (free fructose plus sucrose) predicts body fat percentage in young adults. The secondary objective was to determine if caloric intake of fructose sugars predicts total cholesterol (TC) and low density lipoprotein cholesterol (LDL-C).

Participants and Methods: Men (n=55, body fat=16.3±14.0%) and women (n=281, body fat=26.9±7.5%), 18 to 24 years of age, were recruited for an ongoing, cross-sectional study, The Nutrition Assessment Study. Anthropometric, biochemical and dietary data were collected. Linear modeling was used to assess predictions of body fat percentage and blood lipids with sugars intake, and multiple regressions were used to control for possible covariates.

Results: In a linear model, a 1% increase in caloric intake of fructose sugars predicted a 0.56% higher body fat in men ($\beta=0.311$, $R^2=0.097$, $p=0.037$). This prediction remained significant when adjusting for BMI and alcohol intake ($\beta=0.260$, $R^2=0.505$, $p=0.036$). In women, no predictions were seen with caloric intake of fructose sugars and body fat. Fructose sugars did not predict TC or LDL-C in this sample.

Conclusion: In this population of healthy young adults, higher consumption of fructose sugars is associated with higher body fat in men but not in women.

Longitudinal research is needed to determine if these predictions are observed over time.

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PREFACE

This Thesis was written to comply with the University of Rhode Island graduate school Manuscript Thesis Format. This thesis contains one manuscript. *Predicting body fat and blood lipids with sugars intake*. This manuscript has been written in a form suitable for publication in The Journal of the American College of Nutrition.

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CHAPTER 1

Predicting body fat and blood lipids with sugars intake

Prepared for submission to The Journal of the American College of Nutrition

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Predicting body fat and blood lipids with sugars intake

Objective: The primary objective of this study was to determine if caloric intake of fructose sugars (free fructose plus sucrose) predicts body fat percentage in young adults. The secondary objective was to determine if caloric intake of fructose sugars predicts total cholesterol (TC) and low density lipoprotein cholesterol (LDL-C).

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Conclusion: In this population of healthy young adults, higher consumption of fructose sugars is associated with higher body fat in men but not in women.

Longitudinal research is needed to determine if these predictions are observed over time.

INTRODUCTION

A preventable chronic disease, obesity, affects 600 million adults aged 18 years and older worldwide¹. In the United States (US), obesity generates health care costs ranging from \$147 billion to nearly \$210 billion per year². The US has one of the highest overweight and obesity rates, with over 60% of adults defined as overweight or obese (BMI>25)¹.

Young adults (18 to 24 years old) have experienced increases in obesity³, with weight gain in early adulthood linked to increased obesity⁴ and cardiovascular disease (CVD) risk later in life⁵. One dietary factor, consumption of fructose sugars (sucrose plus free fructose), may lead to adverse metabolic outcomes, such as dyslipidemia⁶⁻⁸, cardiovascular diseases⁹⁻¹¹ and obesity¹²⁻¹⁴ through stimulation of de novo lipogenesis^{15,16}. Adolescents and young adults are among the highest consumers of sugars¹⁷, making them a critical group on which to focus. Research shows that the prevalence of dyslipidemia early in life is a strong predictor of the obesity later in life¹⁸⁻²⁰ and that adverse lipid profiles in young adults accelerates the development of atherosclerosis^{17,21}.

Cross-sectional research concludes no significant associations between fructose consumption and body mass index (BMI)²². However, BMI does not take into account body fat percentage, which experimental research suggest may be increased with fructose sugars intake^{12,23}. Despite this, experimental studies utilize consumption levels exceeding the 95th percentile (14.5% daily energy) for fructose consumption²⁴. Thus, a gap exists in the literature as to whether fructose sugars have deleterious associations with body fat percentage when consumed in free-living individuals.

Therefore, this study aimed to determine if consumption of sugars, with emphasis on fructose sugars, predict body fat percentage and fasting blood lipids in young men and women. Primarily, it was hypothesized that caloric intake of fructose sugars would predict body fat percentage. Secondly, it was hypothesized that Fructose sugars intake would predict total cholesterol (TC) and low density lipoprotein cholesterol (LDL-C). Further, predictions with consumption of non-fructose sugars (free glucose plus lactose) and total sugars were explored. Lastly, predictions of the metabolic risk factors high-density lipoprotein cholesterol (HDL-C) and triacylglycerol (TAG) were explored with consumption of sugars.

MATERIALS AND METHODOLOGY

Subjects and Research Design:

This cross-sectional study in college students used data from the Nutrition Assessment Study (NAS). The NAS is an ongoing observational study of health risk factors in college students enrolled in an introductory nutrition course and a senior level nutrition course. The NAS was approved by the University of Rhode Island Institutional Review Board (IRB) (IRB HU1112-069). Demographic survey, anthropometric measures and biochemical indices were extracted from the NAS database.

Consenting students from the fall 2013 through the spring 2015 semester were given the opportunity to complete the validated Comprehensive Nutrition Assessment Questionnaire (CNAQ)²⁵ as a dietary assessment. Study staff informed students of their eligibility to participate in the study, described the study design and collected a signed consent form of those agreeing to participate.

Demographic and Anthropometric Measures

Participants completed a brief demographic survey, the Nutrition Assessment Survey, that collected information on age, gender, and ethnicity. Upon completion of the Nutrition Assessment Survey, anthropometric measures were conducted by trained researchers using standardized protocols. Measurements were performed in duplicate with additional measures collected if the variance of the two measures was outside the pre-established standards (specified below). Averages of all anthropometric measures with acceptable variances were recorded. Height was measured to the nearest 0.1 cm using a Seca 220 stadiometer (Seca Corporation, Hamburg, Germany). Weight was

measured to the nearest 0.1 kg using a calibrated digital Seca 760 Scale (Seca Corporation, Hamburg, Germany). Body mass index (BMI) was calculated as kilograms' body weight divided by meters squared (kg/m^2). To measure body fat percentage, researchers utilized air displacement plethysmography via BOD POD® (COSMED, Concord, California)^{26,27}, with predicted thoracic volume.

Biochemical Measures

After an overnight fast, a full lipid profile including TC, LDL-C, HDL-C and TAG were collected. The lipid profile was measured using the validated Alere Cholestech LDX® System^{28,29} (Alere Inc., Waltham MA). To calculate LDL-C the Friedewald equation was used. Researchers drew 40uL aliquots of blood, via finger stick, from participants using capillary tubes. Measured outcomes were provided immediately from this system and participants were provided with an explanation of and a copy of their results.

Dietary Measures

The CNAQ is a semi-quantitative 297-item online food frequency questionnaire validated in 2010 for use in adults to evaluate intake of 52 nutrients²⁵. The CNAQ was designed to analyze macronutrients, micronutrients, and indigestible carbohydrates. Responses to the CNAQ were processed using the food composition database, created and maintained by Monash University in Melbourne, Australia²⁵. This questionnaire generates immediate feedback including estimated intake of energy (kJ), total sugars (g), fructose (g), sucrose (g), glucose (g), and lactose (g)²⁵. Fructose sugars in this paper will refer the amount of fructose plus the amount of sucrose

consumed. Non-fructose sugars will refer to the amount of glucose plus the amount of lactose consumed.

The CNAQ could be saved, stopped and continued over multiple intervals if necessary. Participants were prompted to evaluate their average intakes over a one-year period (responses include, but are not limited to “daily”, “weekly”, “monthly” or “never or rarely”). The CNAQ provided brief instructions on how to document food items that are consumed only in specific seasons. Prompts encourage participants to identify quantities of foods consumed, while an unanswered question prevented the participants from submitting the CNAQ. In order to navigate differences in food terminology between the US and Australian citizens, study staff developed a translation sheet. An example: what Australians refer to as “rocket”, the US refer to as “arugula”.

Statistical Analysis

All data analyses were conducted using SPSS version 22.0. For demographic variables, dependent variables, and independent variables, descriptive statistics were used to analyze means, standard deviations, and medians. Frequencies were conducted for categorical variables. All variables were normal according to Shapiro-Wilk after eliminations of outliers greater than three standard deviations from the mean for intake of fructose sugars, non-fructose sugars and total sugars, as well as for body fat percentage and lipid values of TC, LDL-C, HDL-C and TAG.

A total of 17 women and 5 men were eliminated for one or more of the categories for sugars intake. In total, 7 men and 20 women did not complete the full lipid panel, but were included for comparisons with body fat. Two men were missing

body fat analysis, and were eliminated as outliers for LDL-C and TAG. One man was eliminated as an outlier for LDL-C and TAG, and one man was eliminated as an outlier for only TAG. Five men did not complete body fat analysis, but completed the full lipid profile. Lastly, one male participant was eliminated as an outlier with a body fat percentage >65%. Among women, two were eliminated as outliers for both TC and LDL-C, and one woman was eliminated as an outlier for TC. Three women were eliminated as outliers for both HDL-C and LDL-C. One woman was eliminated as an outlier for HDL-C and did not have readable LDL-C and TAG by the Cholestech. In addition, 12 women did not have readable levels of TAG and LDL-C. One woman was an outlier for only TAG, and three women for only LDL-C. Lastly, 19 women did not complete body fat percentage and 4 were eliminated as outliers.

To determine associations between independent and dependent variables, Pearson correlations were applied. To determine if there were relationships between potential covariates, such as alcohol, saturated fat intake and BMI with outcome variables, Spearman's Rho was applied to non-normal covariates. To address our hypotheses, linear modeling was used to determine if caloric intake of fructose sugars, non-fructose sugars and/or total sugars predict body fat percent, TC, LDL-C, HDL-C and TAG. To avoid overfitting in regression models, 5-10 participants are required per predictor when assumptions of normality are met, and 10-20 participants are required per predictor when assumptions of normality are not met.³⁰.

RESULTS

Subject Characteristics and Dietary Intakes

In this cross-sectional analysis data from 414 participants were collected, but data from 336 participants, aged 18-24 years old, were included from the database of the NAS 2013 – 2015 database; 40 students did not complete the CNAQ, 27 participants were over 24 years old, 6 participants reporting daily intakes <400kcal or >7000kcal³¹, 4 participants completing data collection twice and 1 person was pregnant. This sample was mostly female (83.6%) and Caucasian (88.1%). The means and medians of demographic and major dietary intakes are presented in **Table 1**. On Average, women consumed 2% more calories from fructose sugars than men ($14.0 \pm 3.8\%$ vs. $12.0 \pm 4.3\%$, $p < 0.05$), respectively.

Prediction of Body Fat Percentage with Caloric Intake of Sugars

The correlations among fructose intake, non-fructose intake and body fat percentage are presented in **Table 2**. Gram intake of fructose sugars and total sugars negatively correlated with body fat percentage in women. However, they did not correlate with body fat percentage when analyzed as percentage of caloric intake, in women. Caloric intake of fructose sugars, non-fructose sugars and total sugars positively correlated with body fat percentage in men.

Caloric intake of fructose sugars, non-fructose sugars and total sugars did not predict body fat percentage in any linear models in women. In men, a 1% increase in caloric intake of fructose sugars predicted a 0.56% higher body fat percentage in men ($\beta = 0.311$, $R^2 = 0.097$, $p = 0.037$), **Figure 1**. Increasing caloric intake of non-fructose sugars the same amount predicted a 0.83% higher body fat ($\beta = 0.370$, $R^2 = 0.103$,

p=0.030), in men. Similarly, a 1% increase in caloric intake of total sugars predicted a 0.40% higher body fat ($\beta=0.319$, $R^2=0.102$, $p=0.033$). Among men, caloric intake of fructose sugars ($\beta=0.260$, $R^2=0.505$, $p=0.036$), non-fructose sugars ($\beta=0.349$, $R^2=0.501$, $p=0.005$), and total sugars ($\beta=0.276$, $R^2=0.516$, $p=0.023$) maintained significant prediction of body fat when adjusted for BMI and alcohol intake.

Prediction of Blood Lipids with Caloric Intake of Sugars

In this sample, no correlations were detected for TC and sugars did not predict TC in any linear models. Consumption of fructose sugars, non-fructose sugars and total sugars did not predict LDL-C in women. Among men, non-fructose sugars in grams correlated positively and moderately with LDL-C, **Table 2**. In a linear model, a 20gram increase in non-fructose sugars predicted a 6.76mg/dL higher LDL-C level in men ($\beta=0.317$, $R^2=0.100$, $p=0.041$). When adjusted for body fat percent and alcohol intake, non-fructose sugars no longer predicted LDL-C ($\beta=0.313$, $R^2=0.148$, $p=0.080$).

The associations between HDL-C, TAG and sugars are presented in **Table 2**. There were significant inverse associations between fructose and total sugars with HDL-C, in men. In linear models, a 1% increase in caloric intake of fructose sugars predicted a 1.10mg/dL lower HDL-C level in men ($\beta=-0.407$, $R^2=0.165$, $p=0.005$), **Figure 2**. A 1% increase in caloric intake of total sugars predicted a 0.71mg/dL lower HDL-C level in men ($\beta=-0.400$, $R^2=0.160$, $p=0.006$). When adjusted for intake of saturated fat, BMI, and TAG, a 1% increase in caloric intake of fructose sugars predicted a 0.77 mg/dL lower HDL-C in men ($\beta=-0.326$, $R^2=0.442$, $p=0.034$). Similarly, a 1% increase in total sugars predicted a 0.53 mg/dL lower HDL-C in men

when adjusted for saturated fat, BMI and TAG ($\beta=-0.315$, $R^2=0.432$, $p=0.046$).

Caloric intake of non-fructose sugars did not predict HDL-C in men.

Among men, a 20gram increase in non-fructose sugars predicted a 9.74mg/dL higher TAG level ($\beta=-0.398$, $R^2=0.158$, $p=0.010$). When adjusted for body fat percentage and alcohol intake a 20gram increase in non-fructose sugars predicted a 7.38mg/dL higher TAG ($\beta=0.332$, $R^2=0.270$, $p=0.046$). There were no significant associations with respect to HDL-C and TAG in women.

DISCUSSION

A recent cross-sectional study using NHANES 1999-2006 data observed relationships with fructose and non-fructose sugars intake with respect to BMI. They concluded no significant associations with BMI in 25,506 participants²². Our study filled a research gap by exploring predictions with fructose sugars and body fat percentage, a more accurate way to assess weight status. We found predictions of body fat percentage with fructose and total sugars intake in men. However, we did not see these predictions in women. A possible explanation could be increases in visceral fat, which may be specific to fructose²³. Visceral fat is stored to a greater extent in men when compared to women³². However, this was not assessed in the present study, and is an area for future research.

In line with previous cross-sectional research^{17,22}, no significant predictions of TC and LDL-C with consumption of fructose sugars were observed, when consumed in free-living young adults. Despite this, in men non-fructose sugars predicted LDL-C. Previous research has shown a relationship between added sugars and LDL-C in women, but not men³⁴. Glucose has a high glycemic index, which when consumed elicits an insulin response to help deliver glucose to the muscles and surrounding tissues³⁵. Insulin is believed to have an indirect stimulation of HMG-CoA reductase in favor of cholesterol biosynthesis³⁵. However, this was no longer significant when adjusting for significant covariates, such as body fat and alcohol intake.

Increased consumption of sugars may also lead to elevations in TAG through stimulation of de novo lipogenesis⁶, leading to increased production of VLDL^{6,36,37}. Previous research cross-sectional research has concluded associations with added

sugars and TAG. In the present study, non-fructose sugars, but not fructose sugars predicted TAG. This could be due to the overestimation of nutrients by the CNAQ, which is a limitation of this study, as the CNAQ overestimated all nutrients on average about 140%²⁵. A recent paper by Morell et al.³⁸ found that in the same age group (18 to 24-year-olds), caloric intake for men was 2694.6 kcals and for women 1862.8 kcals using 3-day recalls. This demonstrates a potential overestimation of calories, specifically in women as in our sample men reported 2690.4 kcals and women reported 2451.5 kcals. These results demonstrate that there may be greater misreporting by women in our sample. As with most means of self-reported dietary assessment, misreporting is common³³ and if it differed by gender in our sample, then it is an additional possible explanation for our differing results in females versus males. Despite this, the CNAQ was validated for measuring fructose intake²⁵. Further, both men and women, in this study, consumed on average about 32.5 teaspoons of total sugars per day, which is consistent with data from NHANES III¹⁷. Among women, about 62% of total sugars were fructose sugars (fructose plus sucrose) and among men, about 60% were fructose sugars. Our research builds upon previous research reporting that fructose sugars contributed to about 60% of total sugars intake and non-fructose sugars contributed to about 40% of total sugars intake³⁹.

Secondly, this study is limited because physical activity data were not collected. Physical activity has been shown to have beneficial effects on body fat as well as on blood lipids³⁸. Despite our inability to adjust for physical activity, we were able to adjust for saturated fat, cholesterol intake and TAG which have been shown to

related to HDL-C. We were also able to adjust for intake of alcohol, body fat percentage and BMI.

CONCLUSIONS

In conclusion, this study helps to fill a gap in the fructose research by exploring predictions with body fat percentage rather than BMI in free-living young adults. Daily energy intake from fructose sugars predicted body fat percentage and HDL-C in men, not in women. When consumed in free-living young adults, consumption of fructose sugars did not appear to be predictors of TC, LDL-C, or TAG. Future research needs to focus on collecting data on the different storage depots for fat to see if there are differences in the distribution and localization of body fat when consuming different dietary sugars. Future research also should focus on creating a tool to accurately measure fructose consumption in the US population.

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Table 1: Demographic and dietary data of the study population, Nutrition Assessment Study 2013 – 2015.

	Women			Men		
	n = 281			n = 55		
	Mean	Median	SD	Mean	Median	SD
Age, years	19.4	19.0	1.4	19.7	19.0	1.7
Body Fat % ‡	26.9	26.7	7.5	16.3	14.0	8.0
TC (mg/dL)	164.6	162.5	26.3	153.5	150.5	24.4
LDL-C (mg/dL)	83.0	81.0	24.2	86.8	82.0	24.4
HDL-C (mg/dL)	61.2	60.0	15.0	50.5	50.0	11.4
TAG (mg/dL)	111.5	99.0	57.4	83.7	73.0	31.4
<i>Dietary Intakes</i>						
Energy, kcal/day	2451.5	2141.9	1179.9	2690.4	2334.0	1301.2
Total Fat g/day	89.8	77.5	52.8	98.7	91.1	51.5
(%kcal/day)	(32.9)	(33.4)	(8.3)	(33.3)	(33.5)	(7.7)
Protein, g/day	108.8	94.1	56.1	136.7	115.6	71.7
(%kcal/day)	(18.0)	(17.6)	(3.8)	(20.3)	(20.4)	(3.7)
Carbohydrate, g/day	271.8	226.0	145.9	283.3	228.9	158.1
(%kcal/day)	(44.2)	(44.3)	(8.8)	(41.6)	(41.4)	(8.3)
Alcohol, g/day	5.7	1.7	18.4	6.0	2.3	8.4
(%kcal/day)	(1.8)	(0.7)	(6.2)	(1.8)	(1.1)	(2.4)
Total Sugars, g/day	131.3	116.1	64.0	129.2	125.1	67.3
(%kcal/day)	(22.7)	(22.0)	(5.9)	(20.1)	(19.4)	(6.4)
Fructose Sugars, g/day	80.4	72.4	39.3	76.7	70.6	40.3
(%kcal/day)	(14.0)	(13.8)	(3.8)	(12.0)	(12.1)	(4.3)
Non-fructose Sugars, g/day	48.6	44.0	24.2	48.1	45.6	23.6
(%kcal/day)	(8.6)	(8.2)	(2.7)	(8.0)	(7.9)	(3.0)

Means, medians and standard deviations are reported using descriptive statistics.

SD= standard deviation

TC= total cholesterol ; LDL-C= low density lipoprotein cholesterol ; HDL-C= high density lipoprotein cholesterol ; TAG= triacylglycerol

‡ females=258 men=47

Table 2: Pearson correlations between sugars and health outcomes.

		Body Fat %	TC ‡	LDL-C ‡	HDL-C ‡	TAG ‡
Women	Fructose sugars (g)	-0.132*	0.003	-0.038	-0.025	0.149*
	%kcal	-0.039	-0.103	-0.047	-0.099	0.050
	Non-fructose sugars (g)	-0.119	0.031	-0.022	0.039	0.111
	%kcal	-0.071	-0.058	-0.003	-0.078	0.015
	Total sugars (g)	-0.145*	-0.003	-0.036	-0.020	0.109
	%kcal	-0.071	-0.081	-0.012	-0.073	-0.007
Men	Fructose sugars (g)	0.188	-0.004	0.105	-0.358*	0.178
	%kcal	0.311*	-0.129	-0.008	-0.407**	0.090
	Non-fructose sugars (g)	0.233	0.261	0.317*	-0.410**	0.398**
	%kcal	0.321*	0.136	0.078	-0.132	0.010
	Total sugars (g)	0.147	0.033	0.130	-0.322*	0.224
	%kcal	0.319*	-0.069	0.035	-0.400**	0.126

Fructose sugars=free fructose plus sucrose ; Non-fructose sugars=free glucose plus lactose ; Total sugars= fructose sugars plus non-fructose sugars ; TC= total cholesterol ; LDL-C= low density lipoprotein cholesterol ; HDL-C= high density lipoprotein cholesterol ; TAG= triacylglycerol

‡ measured in mg/dL

*p<.05 ; **p<.01

Figure 1: Prediction of body fat percent with sugars consumption in healthy male college students. (n=47)

Figure 1a: A one percent increase in caloric intake of fructose sugars predicts a 0.56% higher body fat in men ($p=0.037$)

Figure 1b: A one percent increase in caloric intake of non-fructose sugars predicts a 0.83% higher body fat in men ($p=0.030$)

Figure 1c: A one percent increase in caloric intake of total sugars predicts a 0.40% higher body fat in men ($p=0.037$)

Fructose sugars= free fructose plus sucrose ; Non-fructose sugars=free glucose plus lactose ; Total sugars= fructose sugars plus non-fructose sugars

Body fat percentage measured via air displacement plethysmography

Figure 2: Predictions of HDL-C with sugars consumption in healthy male college students. (n=45)

Figure 2a: A one percent increase in caloric intake of fructose sugars predicts a 1.10mg/dL lower HDL-C in men ($p=0.005$).

Figure 2b: A one percent increase in caloric intake of non-fructose sugars did not predict HDL-C in men ($p=0.382$).

Figure 2c: A one percent increase in caloric intake of total sugars predicts a 0.71mg/dL lower HDL-C in men ($p=0.006$).

Fructose sugars= free fructose plus sucrose ; Non-fructose sugars=free glucose plus lactose ; Total sugars= fructose sugars plus non-fructose sugars
HDL-C = high density lipoprotein cholesterol ; measured via cholestech LDX

APPENDIX A

REVIEW OF THE LITERATURE

Overview:

This literature review will discuss the consumption of sugars, specifically fructose sugars (fructose and sucrose), as well as non-fructose sugars (glucose plus lactose) and sugar sweetened beverages and their potential relationships with markers of weight status and blood lipids. First we will discuss definitions, sources and tools for measuring sugars. Then we will discuss the relationships between sugars and body composition, specifically analyzing the relationships with body mass index (BMI) and body fat percentage. Lastly, the relationships between consumption of sugars and blood lipids will be analyzed, with emphasis on total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C) and triglycerides (TAG).

Defining Sugars:

The American Heart Association (AHA) and Academy of Nutrition and Dietetics (AND) defined sugars in various contexts¹. First, the AHA and AND defined sugars as monosaccharides and disaccharides including glucose, galactose and fructose¹. Similarly, they defined sugars as both naturally occurring (intrinsic) in fruits, vegetables and dairy foods, or as added (extrinsic) to foods during processing, or in preparation for consumption². In contrast the term sugar refers to sucrose, which is derived from sugar cane or beets².

Fructose is the most common naturally occurring monosaccharide found in fruits and vegetables^{1,2}. In nature fructose is linked as the disaccharide sucrose

(glucose plus fructose), but is also used as a caloric sweetener². Other disaccharides include lactose (glucose plus galactose), which is found in milk products and maltose (glucose plus glucose), which is found in malt and molasses^{1,2}.

Lastly, the AHA and AND define total sugars and high-fructose corn syrup (HFCS)^{1,2}. Total sugars are all sugars (naturally occurring and added) in foods and beverages¹. An alternative to the conventional table sugar (sucrose), HFCS is produced from corn syrup that undergoes enzymatic processing to increase the fructose content and is then mixed with glucose^{1,2}.

Sucrose contains equal parts glucose and fructose bound as a disaccharide bound by an O-glycosidic bond³. In contrast to sucrose, HFCS is composed of free glucose and fructose moieties³. The most common forms of HFCS contain fructose at 42% and 55%³. The most common form of HFCS-55⁴, where the number represents the percentage of fructose in the mixture.

The two most prevalent added sugars in America, HFCS and sucrose, made up 86% of total added sugars used in 2004⁵. Sucrose and HFCS are similar in sweetness, with HFCS-55 being about 95-99% as sweet as sucrose⁶. Pure fructose on the other hand, is 117% as sweet as sucrose⁶, making it a desirable additive in many food and beverage products. In addition to increasing sweetness, sugars have the following functions in food: (1) Inhibit microbial growth by binding with water; (2) add texture, flavor and color to baked goods; (3) Support the growth of yeast for leavening or fermentation; (4) contribute to the volume of ice cream and baked goods; (5) Enhance the crystallization of confectionary products; and (6) they balance the acidity in salad dressings, sauces and condiments².

Several studies have investigated the content of fructose in popular beverages^{7,8}. Ventura et al.⁷ used high performance liquid chromatography to acquire information on free fructose, free glucose as well as sucrose intake in 23 popular sugar-sweetened beverages. Total sugar content of popular beverages ranged from 85% - 128% of what was listed on the label⁷. Average fructose content in beverages made with HFCS was 59% (47 – 65%)⁷. The three most popular beverages (Coke, Pepsi and Sprite) contained between 64-65% fructose⁷. These beverages that are consumed in large quantities in America, contain fructose in greater amounts than the most common forms of HFCS.

Using gas chromatography Walker et al.⁸ concluded that mean fructose concentration of beverages with HFCS and without HFCS were 59.4 ± 8.9 g/L and 30.8 ± 19.5 g/L, respectively. The authors also concluded that the five most popular beverages (Coke, Pepsi, Sprite, Mountain Dew and Dr. Pepper) had fructose:glucose ratios exceeding 60:40⁸. Lastly, they concluded that despite sucrose being listed on Pepsi's ingredients list, no sucrose was detected⁸. With results similar to those of previous research, products may actually be misrepresenting the proportion of and type (bound or free) of fructose consumed. It would appear that the actual percentage of fructose varies in popular beverage items despite being labelled as sucrose or HFCS^{7,8}. This may have an effect on tools used for measuring fructose, when the actual amount of fructose consumed is not easily quantifiable.

Tools for measuring nutrient intake levels

There are multiple ways to determine a person's nutrient intake. These include 24-hour dietary recalls, food frequency questionnaires, and food records. Large data

sets such as those seen in the National Health and Nutrition Examination Survey (NHANES) utilize 24-hour recalls as a way to collect information on nutrient intakes⁹. Some studies have gone further and have analyzed differences between tools for measuring nutrient intakes.

The Observing Protein and Energy Nutrition (OPEN) study involving 484 participants assessed intake using a food frequency questionnaire, two 24 hour recalls, urinary sucrose and fructose as a predictive biomarker of total sugars and doubly labelled water to adjust for grams per 1000kcal^{10,11}. Compared to the predictive biomarkers, self-reported intake of total sugars by food frequency questionnaire was 13.5% lower in men and women and 24-hour recall were biased high in men and nearly identical with women¹⁰. **Table 1** and **Table 2** summarizes the differences between the food frequency questionnaire used (DHQ), 24 hour recalls and total energy expenditure. Compared with the total energy expenditure, men underreported energy intake by 12-14% on 24hour recalls and 31-34% on food frequency questionnaires¹¹. In contrast, women underreported 16-20% on 24 hour recalls and 34-38% on food frequency questionnaires¹¹. This suggests that women might be greater under reporters of energy intake when compared to men.

Despite previous research suggesting underreporting by food frequency questionnaires, a food frequency questionnaire was recently developed to quantify intake of fermentable oligosaccharides, disaccharides, monosaccharides and polyols¹². Barrett et al.¹² conducted a validation paper comparing a food frequency questionnaire to four sets of seven day recalls taken three months apart¹².

Table 1: Nutrient Intakes based on biomarkers and self-reported dietary assessment instruments (women), the OPEN Study¹¹.

Nutrient	No.	Geometric mean	95% CI*	25th percentile	Median	75th percentile
Energy (kcal)						
TEE*	206	2,277	2,226, 2,329	2,031	2,283	2,526
24HR* 1	223	1,919	1,833, 2,009	1,565	1,937	2,438
24HR 2	222	1,814	1,732, 1,899	1,497	1,808	2,275
DHQ* 1	222	1,514	1,438, 1,594	1,173	1,516	1,991
DHQ 2	221	1,405	1,333, 1,481	1,088	1,384	1,838
Protein (g)						
PBM* 1†	174	77.5	74.4, 80.8	63.9	77.1	93.5
PBM 2	150	77.3	73.9, 80.8	63.0	74.7	91.8
24HR 1	223	69.2	65.3, 73.2	54.2	72.2	90.3
24HR 2	222	65.6	61.8, 69.6	50.1	67.7	89.6
DHQ 1	222	56.6	53.5, 59.8	43.9	56.4	76.4
DHQ 2	221	52.7	49.9, 55.7	39.8	51.8	70.1
Protein density (%)						
Biomarker 1‡	160	13.7	13.1, 14.3	11.4	13.9	16.3
Biomarker 2	140	13.6	13.0, 14.2	11.2	13.8	16.1
24HR 1	223	14.4	13.9, 15.0	12.3	14.9	17.4
24HR 2	222	14.5	13.9, 15.1	12.0	14.3	17.4
DHQ 1	222	15.0	14.5, 15.4	13.1	15.1	17.2
DHQ 2	221	15.0	14.6, 15.4	13.1	15.0	17.2

* OPEN, Observing Protein and Energy Nutrition; CI, confidence interval; TEE, total energy expenditure; 24HR, 24-hour dietary recall; DHQ, Diet History Questionnaire; PBM, protein biomarker.

† Protein biomarker = urinary nitrogen/0.81 (converts urinary nitrogen to dietary nitrogen) × 6.25 (converts dietary nitrogen to dietary protein).

‡ Biomarker for protein density = PBM × 4 kcal (kcal per g of protein)/TEE × 100%.

Table 2: Nutrient Intakes based on biomarkers and self-reported dietary assessment instruments (men), the OPEN Study¹¹.

Nutrient	No.	Geometric mean	95% CI*	25th percentile	Median	75th percentile
Energy (kcal)						
TEE*	245	2,849	2,788, 2,912	2,553	2,813	3,146
24HR* 1	261	2,512	2,416, 2,610	2,085	2,577	3,108
24HR 2	260	2,436	2,338, 2,537	1,989	2,466	3,032
DHQ* 1	260	1,959	1,863, 2,061	1,537	1,955	2,550
DHQ 2	259	1,818	1,727, 1,914	1,409	1,870	2,347
Protein (g)						
PBM* 1†	192	104.2	100.3, 108.2	88.7	102.8	124.3
PBM 2	202	103.8	99.9, 107.9	88.1	106.0	125.8
24HR 1	261	91.7	87.6, 96.1	71.9	94.1	118.9
24HR 2	260	92.9	88.2, 97.9	71.5	95.0	124.9
DHQ 1	260	73.0	69.1, 77.1	56.5	73.9	98.0
DHQ 2	259	69.0	65.3, 73.0	51.4	74.7	93.1
Protein density (%)						
Biomarker 1‡	180	14.6	14.1, 15.2	12.7	14.9	17.1
Biomarker 2	189	14.6	14.1, 15.1	12.8	14.8	17.1
24HR 1	261	14.6	14.1, 15.1	11.9	14.5	17.8
24HR 2	260	15.3	14.7, 15.8	12.6	15.5	18.3
DHQ 1	260	14.9	14.5, 15.3	13.4	15.4	17.0
DHQ 2	259	15.2	14.8, 15.6	13.6	15.5	17.1

* OPEN, Observing Protein and Energy Nutrition; CI, confidence interval; TEE, total energy expenditure; 24HR, 24-hour dietary recall; DHQ, Diet History Questionnaire; PBM, protein biomarker.

† Protein biomarker = urinary nitrogen/0.81 (converts urinary nitrogen to dietary nitrogen) × 6.25 (converts dietary nitrogen to dietary protein).

‡ Biomarker for protein density = PBM × 4 kcal (kcal per g of protein)/TEE × 100%.

The Comprehensive Nutrition Assessment Questionnaire (CNAQ), a food frequency questionnaire, overestimated intake of almost every nutrient, with the exception of fat, saturated fat and alcohol intake¹². On average, the CNAQ overestimated nutrients 140%¹². Despite this, the CNAQ was validated for measuring total sugars, fructose and lactose intake¹². Energy, starch and carbohydrate intake was moderately validated using this food frequency questionnaire¹².

Fructose consumption levels

Data suggest that added sugars intake has decreased recently^{13,14}. However, the amount of fructose consumed actually increased from 1977 to 2004 in all genders and age groups⁵. Despite this, it is hard to accurately measure fructose consumption, yet data suggests that adolescents and young adults are the highest consumers of fructose¹⁵. In 1993, Park et al.¹⁵ investigated fructose intake in women (n=922) and men (n=738) aged 19-22 years old. The mean and 90th percentile intake for fructose consumption in females is 35g and 62g, and for men is 47g and 80g respectively¹⁵. More recently, Marriott et al⁵ observed national estimates of dietary fructose intake from 1977 to 2004 and concluded that the mean, 90th percentile and 95th percentile for fructose consumption in 2004 was 61g, 100g and 116g per day in 19-22year old women and 75g, 117g and 134g per day for 19-22 year old men⁵. Furthermore, in 2004 fructose accounted for roughly 42% of the sweeteners used in this country, up from 16% in 1978⁵. Over this same time period, sucrose has decreased from 75% to 44% of the sweeteners⁵. Three years later, in 2007, sucrose was 45% of sweeteners used, HFCS was 41% and all others sweeteners made up about 14% of total sweeteners used¹⁶.

Data from NHANES 1999-2006 suggest that fructose containing sugars intake was 58.85g (10.33% energy) in ≤ 18 year olds and 48.07grams (8.53%) in ≥ 19 year olds¹⁷. Using the same data, total sugars intake was 158.64g (27.83% energy) and 129.65g (23.16%) for ≤ 18 year olds and in ≥ 19 year olds, respectively¹⁷.

One of the main dietary sources of fructose, HFCS, is consumed largely in the US⁴. In fact, a recent review by Goran et al.⁴, explored the prevalence of global HFCS using availability data from 43 countries. **Figure 1**⁴ displays the countries that were defined as countries with HFCS ($>0.5\text{kg/capita/year}$). Of the 43 countries, 22 were defined as users of HFCS, with US having HFCS much more highly available⁴.

Figure 1: Countries defined as users of high-fructose corn syrup ($>0.5\text{kg/capita/year}$) using global availability data from 43 countries⁴.

Total and added sugar consumption levels

According to the United States Department of Agriculture, in 1970 roughly 2,109kcal/day were consumed on average per person¹³. By 2010, that number increased to 2,568kcal/day per person¹³. This is the equivalent to an increase of about 22%, or an additional 459kcal daily¹³. Of this increase, about 4% (20kcal) comes from an increase in added sugars with the rest coming from an increase in flour, cereal products and added fats¹³. While this is only a small increase in added sugars over 40 years, some data suggest that sugars consumption actually decreased in recent years¹⁴. In 1999, annual sugars intake was on average 89.3 lbs/person/year, in America¹⁴. However, in 2013 annual sugars intake was at 75.4 lbs/person/year¹⁴. This is equivalent to a 16% decrease in annual sugars intake¹⁴.

Using data from 1971 and 1994 (NHANES I and III), Chun et al.¹⁸ estimated total and added sugars intake in participants <18 years old, participants >19 years old and among all participants¹⁸. Total and added sugars intake was higher among participants <18 years old¹⁸. On average participants <18 years old consumed 138g total sugar with 88g added according to NHANES I and 139g total sugars with 92 grams added according to NHANES III¹⁸. This translates to an increase in one gram of total sugars, but a 3g decrease in natural sugars and a 4g increase in added sugars, corresponding with the rise in refined carbohydrates¹⁸. Among participants 19 and older, total sugars intake was 110g with 71g added sugars in NHANES I and was 126g and 84g in NHANES III, respectively¹⁸. This age group experienced a 14-gram increase in total sugars with an increase seen in both natural and added sugars. According to NHANES III data, free fructose accounted for 21% of sugars intake

among both age categories and sucrose accounted for 39% and 43% of total sugars intake in <18 year olds and >19 year olds, respectively¹⁸. All fructose sugars (free fructose plus sucrose) contributed about 60% of total sugars intake. All other sugars, non-fructose sugars (glucose, galactose, lactose and maltose), contributed about 40% of total sugars in <18 year olds and 35% in >19 year olds¹⁸.

Data from NHANES 1999-2006 estimates that intake of added sugars accounts for 15.8% of caloric intake in participants ≥ 18 years old¹⁹. More recent data from NHANES 2005-2010 suggests that in adolescents, aged 12-19 years old, intake of added sugars was 16% of caloric intake⁹. This suggests that added sugars intake has remained relatively constant over the last decade. Of these adolescents, 88% consumed $\geq 10\%$ energy from added sugars and 5.5% had a usual intake above 25% total caloric intake⁹. This suggests that of the adolescents surveyed, only a small percent of them are meeting current guidelines for consumption of sugars set by the AHA.

Table 3: Usual intake of Added sugars (in teaspoons), 2001-2004¹

Age, y	n	Mean	SE
1-3	1515	12.2	0.33
4-8	1701	21.0	0.54
Males 9-13	1061	29.2	0.92
Males 14-18	1424	34.3	1.03
Males ≥ 19	4650	25.4	0.48
Females 9-13	1112	23.2	0.82
Females 14-18	1362	25.2	0.71
Females ≥ 19	5063	18.3	0.37
All persons ≥ 1	17 888	22.2	0.29

n Indicates number of persons in sample; SE, standard error of the mean (degrees of freedom=30).

*Includes white, brown, and raw sugar; syrup; honey; and molasses, eaten separately or used as ingredients in processed or prepared foods such as breads, cakes, soft drinks, jams, and ice cream.

One teaspoon of added sugars has the same amount of total sugars as 1 teaspoon (4 g) of table sugar (sucrose).

Adapted from National Cancer Institute.⁹

Guidelines for Sugars Consumption

In an AHA scientific statement, Johnson et al.¹ reviewed the guidelines for added sugars intake. Using the National Cancer Institute's report on estimates of added sugars from NHANES 2001 – 2004 and adapted the data to reflect teaspoons of added sugars, **Table 3**¹. The AHA has determined that no more than half of your discretionary calories come from added sugars, equating to about 100 calories, or 6 teaspoons, for most women and 150 calories, or 9 teaspoons, for men^{1,20}. Despite these recommendations from the AHA, data from 17,888 participants suggest that Americans were consuming 22.2 teaspoons of added sugars daily, equivalent to about 88.8g or 355kcal/day¹. This consumption amount is over two fold higher than the recommendations for men, and over three fold higher than the recommendation for women.

Despite the AHA guidelines, current guidelines set by the Institute of Medicine for added sugars consumption are set at <25% of total caloric intake²¹. However, the recommendations set by the Institute of Medicine were developed in 2002, and since then many researchers have demonstrated evidence of the harmful effects of added sugars, specifically SSB²⁰.

More recent recommendations come from the World Health Organizations, stating that no more than 10% of your caloric intake come from free sugars, with further recommendations to limit to 5% if possible²⁰. For a 2000 calorie diet, this would mean reducing added sugars intake to 12.5 teaspoons at 10% of caloric intake and 6.25 teaspoons at 5% of caloric intake, similar to the recommendations of the AHA²⁰.

Prevalence of overweight and obesity

Using reliable data with large sample sizes, researchers are able to estimate the prevalence of obesity in America. Data from NHANES 2007-2012 reveal prevalence of underweight, normal weight, overweight, and the three obese classes by gender, race and age in participants 25 years and older²². According to the weighted sample sizes, 39.96% (weighted n= 36,325,297) of men and 29.74% (weighted n=28,894,030) of women were overweight and 35.04% (weighted n=35,792,733) of men and 36.84% (weighted n=35,792,733) of women were defined as obese according to their BMI classification²². Men were just as likely to be obese at 25-54 years (34.9%) and ≥ 55 years (35.3%), while women were more likely to be obese in their later years (38.7%) when compared to 24-54 year olds (35.7%)²². According to race/ethnicity by gender totals, Non-Hispanic black women (56.8%), Mexican American women (43.3%) and Non-Hispanic black men (39.2%) had the three highest rates of obesity, respectively²².

According to a 2005 World Health Organization Report, approximately 1.6 billion adults are overweight and at least 400 million are obese worldwide²³. These number were projected to hit 2.3 billion overweight adults and 700 million, respectively, by 2015²³. However, despite projections, a joint report in 2015 from the World Health Organization and World Obesity Federation found that approximately, 2 billion adults worldwide are overweight or obese with projections reaching 2.8 billion people by 2025²⁴. On average 98 million adults were severely obese ($\text{BMI} > 35\text{kg/m}^2$) in 2014²⁴. Currently obesity rates in America generate healthcare costs ranging from \$147 billion to nearly \$210 billion per year²⁵. These costs stem largely from metabolic consequences of excess adiposity²⁵.

Metabolic Risk Factors

Metabolic Syndrome is a cluster of interrelated risk factors of metabolic origin that directly promote the development of cardiovascular diseases and other metabolic diseases²⁶. These risk factors include elevated waist circumference, elevated TAG, reduced HDL-C, elevated blood pressure and elevated fasting glucose, which are defined in **Table 4**²⁶.

The transition from adolescence to young adulthood is shown to be a time of increased risk of developing obesity²⁷, yet many young people do not see themselves as being at risk for chronic diseases²⁸⁻³⁰. Large survey data demonstrate that young adults, aged 18 to 24 years of age, have risk factors associated with chronic diseases^{31,32}.

Recent research conducted from large universities show the prevalence of risk factors in young adults^{28,33}. First, a large sample of participant data was collected as a part of the Young Adult Health Risk Screening Initiative²⁸. The investigators found that about 77% of men and about 54% of women had at least one criterion for metabolic syndrome, and about 10% of men

Table 4: Criteria for Clinical Diagnosis of Metabolic Syndrome²⁶

Measure (any 3 of 5 constitute diagnosis of metabolic syndrome)	Categorical Cutpoints
Elevated waist circumference*†	≥102 cm (≥40 inches) in men ≥88 cm (≥35 inches) in women
Elevated triglycerides	≥150 mg/dL (1.7 mmol/L) or On drug treatment for elevated triglycerides‡
Reduced HDL-C	<40 mg/dL (1.03 mmol/L) in men <50 mg/dL (1.3 mmol/L) in women or On drug treatment for reduced HDL-C‡
Elevated blood pressure	≥130 mm Hg systolic blood pressure or ≥85 mm Hg diastolic blood pressure or On antihypertensive drug treatment in a patient with a history of hypertension
Elevated fasting glucose	≥100 mg/dL or On drug treatment for elevated glucose

*To measure waist circumference, locate top of right iliac crest. Place a measuring tape in a horizontal plane around abdomen at level of iliac crest. Before reading tape measure, ensure that tape is snug but does not compress the skin and is parallel to floor. Measurement is made at the end of a normal expiration.

†Some US adults of non-Asian origin (eg, white, black, Hispanic) with marginally increased waist circumference (eg, 94–101 cm [37–39 inches] in men and 80–87 cm [31–34 inches] in women) may have strong genetic contribution to insulin resistance and should benefit from changes in lifestyle habits, similar to men with categorical increases in waist circumference. Lower waist circumference cutpoint (eg, ≥90 cm [35 inches] in men and ≥80 cm [31 inches] in women) appears to be appropriate for Asian Americans.

‡Fibrates and nicotinic acid are the most commonly used drugs for elevated TG and reduced HDL-C. Patients taking one of these drugs are presumed to have high TG and low HDL.

and about 3% of women had metabolic syndrome²⁸. They further analyzed the number of risk factors by BMI categories (18.5-24.9, 25-29.9, and $\geq 30\text{kg/m}^2$) and stated that both men and women with a BMI $\geq 30\text{kg/m}^2$ had significantly more metabolic criteria than those in the other BMI categories²⁸. Although the focus of this paper was to look at risk factors, they did include that women had a greater intake of total sugars in relation to calories as compared to men (21.9% vs 20.0%, $p < 0.001$)²⁸. Despite this, they concluded that overweight/obese college aged men present with a greater prevalence of risk when compared to college aged women²⁸.

A similar study observed metabolic risk factor criterion across large (>10,000 students) diverse universities to examine the relationship with weight status and adiposity³³. Overall, more than half of the sample had at least one metabolic syndrome criterion, with men twice as likely to have metabolic syndrome when compared to women (12% vs 6%, respectively)³³. Metabolic syndrome was five times more prevalent among overweight and obese participants when compared to normal weight (16% vs 3%, $p < 0.001$ respectively)³³. Lastly, overfat ($\geq 20\%$ body fat for men and $\geq 33\%$ for females)^{33,34} participants had significantly more metabolic syndrome criteria than participants with normal levels of body fat (1.7 vs 0.7; $p < 0.001$)³³.

Metabolism of sugars

The Academy of Nutrition and Dietetics stated that HFCS and sucrose are similar in composition². Similarly, the metabolic effects of HFCS and sucrose do not differ making it essential to observe fructose sugars (sucrose and free fructose) to assess metabolic impacts¹⁶. However, there are differences in the metabolism of the two monosaccharides that make up sucrose and HFCS, glucose and fructose³⁵.

Glucose metabolism occurs in all tissues of the body with about 30-40% of metabolism occurring in the liver.³⁶ Glucose is a high glycemic index non-fructose containing sugar³⁵. The glycemic index is a physiological classification of the available carbohydrate content in foods, which was first proposed in 1981³⁷. It reflects the capacity of a carbohydrate containing food to raise blood glucose³⁸. The glycemic index is determined by comparing the postprandial glycemic response of a food with the post prandial glycemic response to the same amount of available carbohydrate from a standard food, usually bread or glucose^{37,38}. The actual glycemic index value is the area under the blood glucose curve for the test food, expressed as a percentage of the standard control³⁷. The glycemic index therefore depends on the food rather than the characteristics of the individual^{37,39}. Generally speaking a low glycemic index food would be one that scores less than 70, and a high glycemic index food would score over 100⁴⁰. Factors that can effect glycemic index include the nature of the starch, amount of fiber, fat and protein in addition to cooking method and time³⁹.

Glucose, the standard control for glycemic index, elicits a high insulin response upon consumption³⁵. Insulin has an indirect role in stimulation of HMG-CoA reductase, the rate limiting enzyme in cholesterol biosynthesis³⁵. This indirect relationship stimulates an overproduction of mevalonate in favor of conversion to cholesterol, with increased consumption³⁵. Thus, some experts have cautioned against chronic consumption of a high glycemic index diet⁴¹. Indeed, a number of trials have demonstrated that a low glycemic index and low glycemic load diet may be protective against obesity related chronic diseases⁴¹.

Fructose has a very different metabolism when compared with the metabolism of glucose. When consumed alone fructose is poorly absorbed, but absorption is enhanced in the gut when consumed with glucose²³. Thus, there is a rapid and almost complete absorption of fructose and glucose when consumed as sucrose and HFCS²³. While HFCS is composed of free fructose and glucose moieties, the O-glycosidic bond in sucrose needs to be hydrolyzed by the α -glucosidase sucrose in the sucrase-isomaltase complex of the enterocytes in the small intestines to produce glucose and fructose². Once sugars are in monosaccharide units they can be absorbed into the enterocyte². There are differences in the absorption of fructose versus glucose.

Transporters are required for all monosaccharides to enter and exit the enterocyte of the small intestines^{2,36}, **Figure 2**. When there is a low concentration of

Figure 2: When sugar concentrations in the lumen are low, sugars enter the enterocyte via SGLT1 and GLUT5, exiting via GLUT 2 to enter bloodstream³⁶. The consumption of a sugar-rich meal saturates SGLT1 and GLUT5 can result in recruitment of GLUT2 to transport sugars across apical membrane³⁶. This can triple the sugar uptake by enterocytes³⁶.

monosaccharides within the lumen, glucose and galactose are transported into the enterocyte by the sodium/glucose cotransporter 1 (SGLT1)^{2,36}. The SGLT1 is located on the apical membrane of the enterocyte, and has a high affinity for glucose and galactose^{2,36}. The lumen of the intestines has a higher concentration of sodium when compared to the enterocyte, which allows for an inward gradient into the enterocyte³⁶. This drives glucose and galactose absorption against their own concentration gradients with help from two sodium ions³⁶.

Once inside the enterocyte the glucose and galactose part from the sodium ion³⁶. Fructose, on the other hand, is not transported into the enterocyte via SGLT1, but is instead transported by the facilitated fructose transporter 5 (GLUT5)³⁶. This transporter is also located on the apical membrane, but this transporter has a low affinity but high capacity for transporting fructose³⁶. However, when sugars are consumed in large quantities, SGLT1 and GLUT5 become saturated resulting in recruitment of glucose transporter (GLUT2), which can triple the amount of sugar taken up by the enterocyte³⁶.

Despite the differences of these monosaccharides in their entry in to the epithelial absorptive cells, most monosaccharides cross the basolateral membrane of the enterocyte via the facilitated GLUT2³⁶. It should be noted that GLUT5 transporters are also located on the basolateral membrane, however GLUT5 transporters compliment GLUT2-mediated exit of fructose from the enterocyte³⁶. The transporters located on the basolateral membrane help deliver the newly absorbed monosaccharides into the capillaries and portal blood³⁶.

Once absorbed, fructose metabolism occurs preferentially and primarily in the liver and does not elicit the same insulin response as glucose, identifying fructose as a lower glycemic index sugar^{23,42}. The liver has a high level of glucokinase, the enzyme responsible for phosphorylation of glucose in the liver.³⁶ Once glucose becomes phosphorylated, glucose 6-phosphate can continue through glycolysis.³⁶ However, this enzyme does not phosphorylate fructose.³⁶ Thus, the liver utilizes fructokinase, an enzyme that catalyzes the reaction of fructose to fructose 1-phosphate, instead of the glycolytic intermediate glucose 6-phosphate.³⁶ Fructose 1-phosphate can then enter fructolysis, bypassing the regulated phosphofructokinase enzymatic reaction in glycolysis.³⁶ This inadvertently provides fructose with a less regulated metabolism^{36,42}. Thus, high fructose consumption can result in unchecked carbon flow towards acetyl CoA³⁶. If this exceeds the demands of the Krebs cycle, then the carbon will be directed to fatty acid biosynthesis via acetyl CoA carboxylase³⁶.

Chronic over consumption of fructose sugars increases de novo lipogenesis, resulting in elevated serum triglyceride (TAG) concentrations in adults of 80-200%⁴³⁻⁴⁶. Excessive fructose consumption may lead to adverse metabolic effects, such as increased visceral adiposity or dyslipidemia⁴⁷.

Fructose in relation to body composition

When compared to glucose, consumption of fructose does not attenuate circulating levels of ghrelin, an appetite stimulating hormone^{48,49}. Thus, fructose intake may lead to increased food intake⁵⁰. In fact, several studies examining fructose consumption have reported increased caloric intake with higher energy consumption of fructose, which may be responsible for changes in body composition⁵¹⁻⁵³. Many of these studies have focused on fructose in beverages, although a cross-sectional study

in college students found elevated fasting hunger with higher total dietary fructose intake⁵⁴.

To observe the effects of fructose consumption on body composition, Lowndes et al.⁵¹ conducted a randomized, prospective, parallel group, blinded study in which participants consumed 3 different levels (8%, 18% and 30% kcals/day) of HFCS or sucrose. There were no significant differences when looking at sucrose intake vs HFCS intake, therefore the participants were pooled for analyses⁵¹. In the entire cohort, weight, BMI, body fat percentage, fat mass and waist circumference were increased.^{51,55} Participants who consumed fructose at 30% of caloric intake displayed greater increases in body weight and BMI when compared to participants consuming fructose at 8% and 18% of caloric intake⁵¹. One limitation to this study is that participants consuming 30% of their caloric intake from fructose experienced greater increases in energy intake when compared to those consuming 8% or 18% of their caloric intake⁵¹. Whether this is due to the fructose-induced hunger is a topic for future research.

To compare the effects of a caloric and non-caloric sweetener on body weight, 30 males and females were recruited for a 3-week intervention in which participants consumed either HFCS, aspartame or no soda.⁵² When compared to aspartame and the no soda group, the HFCS group significantly increased weight in both males (+0.97±0.25 kg) and females (+0.52±0.23 kg).⁵² However it should be noted that there was only about a 37% compensation in calories of the extra 530 kcals provided by the HFCS beverage⁵². This means that on average participants were consuming an extra 335 calories/day.⁵²

Some results even suggest that higher fructose may have less impact on body fat percentage than glucose. In a double blind, randomized cross-over trial, with four 3-week interventions, compared to a high glucose diet (80g/day), a high fructose diet (80g/day) resulted in lower body fat % ($16.8 \pm 2.8\%$ vs. $15.8 \pm 2.2\%$, $p < .05$)⁵⁶. In a very high fructose feeding study where 20 participants received either 150g fructose (600kcal) or 150g glucose (600kcal) in a hyper energetic diet for 4-weeks, no changes in liver fat or visceral fat were observed⁵⁶. Similarly, no changes in body fat percentage were observed in either diet⁵⁶. These studies, however, were limited by the length of their interventions.

Using the NHANES data from 1999-2006, Sun et al.¹⁷ concluded that there was no significant relationship between percent energy from fructose containing and non-fructose containing sugars with relation to BMI and WC¹⁷. While these measures are used for estimates of obesity worldwide²⁴ and as a criterion for metabolic syndrome²⁶, these are not the best measures of determining one's obesity status and are limited in their ability to determine body fatness.

To test the effects of a low fructose diet ($<20\text{g/day}$) and a moderate fructose diet ($50\text{-}70\text{g/day}$) with natural fruit supplements on weight loss, Madero et al.⁵⁷ conducted a randomized control trial in 131 overweight and obese participants. Percent of total energy for carbohydrates remained the same.⁵⁷ Each intervention group showed significant weight loss when compared to baseline, with the moderate fructose group experiencing greater decreases in weight.⁵⁷ Body fat percent was significantly decreased in both the low fructose diet (-2.09 ± 6.32 , $p < .05$) and in the moderate fructose diet (-0.89 ± 6.33 , $p < 0.01$).⁵⁷ Similarly, BMI was decreased in both

the low fructose (-1.18 ± 0.82 , $p < 0.001$) and moderate fructose (-1.57 ± 1.08 , $p < 0.001$)⁵⁷. This suggests that the moderate fructose group, although losing significantly less body fat, lost more lean body mass when compared with the low fructose group.

Similarly, Lowndes et al.⁵⁸ conducted a randomized, prospective, double blind weight loss trial with four hypocaloric diets containing either HFCS or sucrose at 10% or 20% of their caloric intake. The average energy deficit was 309 calories.⁵⁸ A total of 162 participants completed the 12-week intervention, which supplied all sweeteners in a low-fat (1%) milk.⁵⁸ All four hypocaloric diets reduced body mass, BMI, body fat percentage, WC and fat mass in these overweight and obese adults.⁵⁸ Similar to the findings of Madero et al.⁵⁷ larger losses in body fat percentage were observed in those consuming HFCS and sucrose at 10% of caloric intake when compared to 20% of caloric intake⁵⁸. Although this difference was not significant, it suggests that there may be a benefit to decreasing the amount of fructose one takes in if they are trying to lose more weight, specifically through loss of body fat.

Total and added sugars in relation to body composition

Research from Rikkers et al.⁵⁹ attempted to estimate Australian refined sucrose consumption over decades and concluded that it was not possible to produce reliable data. In response to this Barclay and Brand-Miller released a report in which they demonstrate the “Australian paradox”, where sugars consumption declined over the same period that obesity rates increased⁶⁰. This is true in Americans as well, where from 1977 to 2012 obesity rates have increased and sugars intake increased through 1998, but has since dropped to similar consumption levels seen in 1991^{13,14}. **Figure**

3^{13,14,61,62}, shows the rise in obesity over the last 50 years, the increase of 450 kcals total over 40 years, and the decrease in pounds of sugar consumed over 15 years.

Several reviews have explored the relationship of total and/or added sugars and body composition⁶³⁻⁶⁶. In 2003, Saris et al.⁶⁴ concluded that there is little evidence that sugars have direct negative effects on body weight control. However, the combination of frequent consumption of sugar sweetened beverages (SSB) with an inactive lifestyle, reduces the metabolic need for fat as fuel, potentially leading to considerable increases in weight⁶⁴.

In contrast, 10-years later Te Morenga et al.⁶³ conducted a systematic review

Figure 3: Trends in calorie intake, sugars intake and obesity rates in America^{13,14,61,62}.

and meta-analysis of randomized control trials and cohort studies and concluded among free living people, intake of free sugars or SSB was a determinant of body weight. They reviewed 30 randomized control trials and concluded that by reducing

intake of free sugars in ad libitum diets, there was an average of 0.8kg reduction in weight⁶³. When increasing free sugar intake there was an association with a comparable 0.75kg increase in weight⁶³. Increases in SSB at a one year follow up in prospective studies, concluded a higher odds ratio of being overweight or obese with higher consumption of SSB when compared to lower consumption⁶³.

In 2009 van Baak et al.⁶⁷ concluded that there are inverse associations between content of sugars and body adiposity and weight using randomized control trials that replaced fat in the diet to increase carbohydrate intake^{65,67}. In another review, Ruxton et al.⁶⁶ examined whether sugar consumption is detrimental to health. They concluded similar findings that sugars intake in place of fat intake increases body weight^{65,66}.

Cross-sectional studies suggest there may not be a relationship between BMI and sugars intake. To investigate if the uptrend of obesity prevalence in the USA was associated with dietary sugar intake, Song et al.⁶⁸ used NHANES I and III data to compare intakes. They concluded that the primary contributor to BMI in all age groups was energy intake⁶⁸. Total sugars intake was a non-predictor for BMI in all age groups⁶⁸. A similar cross-sectional study examining data from NHANES 1999-2006 categorized sugars intake of participants ≥ 18 years old into five categories: $<5\%$, 5% - $<10\%$, 10% - $<17.5\%$, 17.5% - $<25\%$, $\geq 25\%$ of total calories⁶⁹. There were no significant differences in BMI or WC among the groups⁶⁹. Similarly, a cross-sectional study used NHANES data from 1999-2004 to categorize sugars intake of 12-18 year olds¹⁹ using the same five categories above⁶⁹. There were no significant differences in BMI z-score among the categories¹⁹.

Sugar Sweetened Beverages and Body Composition:

Sugar-sweetened beverages include soft drinks, fruit drinks, energy and vitamin waters and are composed of naturally derived caloric sweeteners such as sucrose, HFCS and fruit juice concentrates²³. Collectively, these are the largest contributors to added sugars intake in the US^{1,23}. From 1970 to 2006, per capita consumption of SSBs increased twofold from 64.4kcal/day to 141.7kcal/day, with adolescents and young adults consuming over 200kcal/day⁷⁰.

To assess disparities in calorie intake between SSB consumers and non-consumers and determine associations with obesity and overweight-obesity, a New York City population study was conducted in 488 adults⁵³. Consumers of SSBs consumed on average 193kcal/day from SSB, equating to roughly 10% of total caloric intake⁵³. When compared to non-consumers, adults who consumed SSBs consumed on average 572 kcal more, possibly due to greater SSB consumption⁵³. However not all of these calories can be attributed to SSB consumption, so it is believed that higher intake of fructose may disrupt regulating hormones, as previously discussed^{48,49,54}. Lastly, this study concluded that each 10oz serving of SSBs increased obesity likelihood and increased overweight-obesity likelihood⁵³. A cross-sectional study using NHANES data suggests that there is also an increased likelihood of being overweight or obese, defined by BMI, with consumption of SSBs⁷¹.

To compare consumption of SSB with consumption of an isocaloric milk and with consumption of a non-caloric SSB on changes in total fat mass, Maersk et al.⁷² conducted a 6-month randomized intervention with four groups (control was given water). On average, SSB consumption had significantly higher liver fat (132%-143%

change) and visceral fat (24-31% change), when compared to the non-caloric SSB and isocaloric milk⁷². However, total fat mass was not different across the four groups⁷².

To explain the possible associations between SSB and increase overweight and obesity, Bachman et al.⁷³ reviewed four possible mechanisms: (1) excess calories, (2) glycemic index and glycemic load, (3) lack of effect of liquid calories on satiety, and (4) displacement of milk. The evidence on whether liquid and solid foods differ in the effects on caloric compensation is conflicting, and research needs to more carefully consider the many factors that influence satiety⁷³. However, evidence is inconsistent about whether this displacement has implications on obesity⁷³. This review concluded that the evidence regarding SSB consumption and obesity remains inconclusive⁷³.

A positive relation between added sugars consumption and total energy intake is observed in many cross-sectional studies^{52,74-79}. Despite this, some cross-sectional studies have shown inverse associations between added sugars consumption and body weight or BMI⁸⁰⁻⁸². Next, a review of 31 short term studies (<1day) found that only 15 studies show an association between low glycemic index meals and greater satiety and reduced hunger; while 16 studies reported reduced satiety or no differences with low glycemic index foods⁸³. During the same time that SSB consumption increased, milk consumption in children decreased^{74,84}. Similarly, other reviews have also deemed SSB consumption and obesity as inconclusive based on current evidence^{85,86}.

Despite the previous review⁷³, research conducted in children shows a relationship between the consumption of SSB and BMI⁷⁵. A prospective observational study has shown that for each additional serving of SSB, there is an average increase in BMI of 0.24kg/m² in children⁷⁵. There was also an increased frequency of obesity

observed in this sample of 548 ethnically diverse school children⁷⁵. Another review observed the relation between SSB and body weight in children and adults⁸⁷. Cohort studies and randomized control trials were included, 20 in children 12 in adults⁸⁷. In cohort studies, 1 serving increment of SSB was associated with a 0.06 and 0.05-unit increase in BMI in children and a .22kg and .12kg increase in weight in adults over one year in random and fixed effects models, respectively⁸⁷. Meanwhile, randomized control trials in children show reductions in BMI gain when SSBs are reduced and increases in body weight in adults when SSBs were added⁸⁷.

A Systemic review of SSB and weight gain concluded that large cross-section studies, in conjunction with well-powered prospective cohort studies with long periods of follow-up, show a positive association between greater intakes of SSB and weight gain and obesity in both children and adults⁸⁸. They finish by adding, short-term feeding trials in adults also support an induction of positive energy balance with weight gain by intake of SSB⁸⁸.

Despite all previous research on SSB, added sugars and fructose consumption, a review by Dolan et al.⁸⁹ concluded that there is no convincing evidence from long term studies that fructose ingestion up to 100 g/day instead of glucose or sucrose is associated with an increase in body weight. Similarly, they did not find any associations with blood lipids when consuming fructose up to 100g/day⁸⁹.

Fructose and blood lipids

As discussed earlier, higher intake of fructose may be associated with increased hepatic de novo lipogenesis,^{43,45,46,90} however, more evidence is needed.⁹¹ The metabolic fate of fructose is described in **Figure 3**⁹². Several studies have concluded that fructose ingestion increases TC and LDL-C,⁹³⁻⁹⁷ while other studies have not seen these results.^{51,56,57,89,98-100}

Sugars research from the 1980's and 1990's set the

groundwork for the research that is being conducted today. In 1983, Hallfrisch et al.⁹⁷ conducted a cross-over study where 12 hyperinsulinemic men consumed three diets in 5-weeks intervals. The three diets consumed in this trial included: (1) a diet containing 0% energy from fructose and 15% energy from starch, (2) a diet containing 7.5% energy from fructose and 7.5% energy from starch, and (3) a diet containing 15% energy from fructose and 0% energy from starch⁹⁷. When comparing the 0% energy fructose, 7.5% energy fructose and 15% energy fructose, significant increases in TC (191.3, 202.8, 200.9mg/dL) and LDL-C (136.0, 145.5, 142.9mg/dL) were observed

Figure 3: Metabolic fate of oral fructose load⁹⁰

FIG. 3. Metabolic fate of an oral fructose load: after fructose ingestion, fructose metabolism takes place essentially in splanchnic tissues. In the liver, a large proportion of fructose is converted into glucose, which can be either stored as hepatic glycogen or released as plasma glucose. Part of the fructose load is converted into lactate in enterocytes and in the liver, which leads to increased lactacidemia. A small portion of fructose is converted into fatty acids; although quantitatively minor, this pathway may play an important role in the development of fructose-induced hepatic steatosis and dyslipidemia. The estimated contribution of glucose production, glycogen synthesis, lactate production, and total oxidation is indicated based on experimental data described in section II.D. Data for lipogenesis and very-low-density lipoprotein (VLDL)-triglyceride (TG) secretion are not available, but these pathways are quantitatively minor.

respectively. Both the 7.5% fructose diet and the 15% energy fructose diet produced statistically higher TC and LDL-C then when participants consumed 0% energy from fructose⁹⁷.

However one year later, in 1984, Crapo et al.¹⁰⁰ did not have these same findings when observing fructose consumption in healthy individuals. The diet consisted of between 63 to 99grams fructose or roughly 24% of total carbohydrates consumed (with carbohydrates consumed at 55% of total caloric intake)¹⁰⁰. When consumed for 2-weeks, TC decreased from baseline to 14 days (188mg/dL to 173mg/dL, $p < .05$)¹⁰⁰. According to the sample diet, roughly 13% of calories were consumed as fructose, similar to the upper level used in a study one year prior^{97,100}. One main difference was that the participants used in the studies were different in terms of health status, one study observing changes in hyperinsulinemic participants⁹⁷ and the other in healthy individuals¹⁰⁰. The other is that one study, which found significance, was 5-weeks in length⁹⁷, while the other, which did not find significance, was only 2-weeks¹⁰⁰.

In 1985, Crapo et al.⁹⁸ repeated this 2-week trial in diabetic subjects with the diet consisting of between 63 to 99grams of fructose or roughly 24% of total carbohydrates consumed (with carbohydrates consumed at 55% of total caloric intake).⁹⁸ He concluded that there was no significant difference in TC after 14-days of consuming a fructose in diabetic subjects.⁹⁸ However, the length of the study was rather short, limiting the potential for changes to occur.

Bantle et al.⁹⁹ conducted a cross-over study in which participants consumed, in random order, a fructose diet at 17% energy needs and an isoenergetic diet with

glucose at 14% and with fructose at less than 3% energy needs⁹⁹. Despite consuming fructose at higher levels and for a longer duration than previous studies^{98,100}, no long-term changes were observed in TC or LDL-C⁹⁹. However, TAG were higher in men consuming fructose at 17% of energy ($p<.05$)⁹⁹.

Jameel et al.⁹³ conducted a randomized, single blind, cross-over study in 14 men and women. Three different isocaloric beverages (50 grams fructose, 50 grams glucose and 50 grams sucrose dissolved in water) were served on three different occasions.⁹³ Consumption of fructose led to an initial significant increase in TC at 30min and 60min, when compared to glucose and sucrose.⁹³ However, at 120min the increase was no longer significantly different from glucose and sucrose.⁹³ Similarly, LDL-C was significantly increased with consumption of fructose at 30 min and 60 min, but not at 120 minutes.⁹³ Overall, plasma TC area under the curve (AUC) and LDL-C AUC were higher when consuming fructose.⁹³

Next, Aeberli et al.⁹⁴ conducted a randomized, double blind, cross-over study in which nine males consumed a medium fructose diet (40g/day), a high fructose diet (80g/day), a high glucose diet (80g/day) and a high sucrose diet (80g/day) for three weeks each with a four-week wash-over between diets. Compared with the high glucose group, the all fructose containing diets had higher TC and LDL-C at 3-weeks.⁹⁴

To compare the effects of fructose, glucose and HFCS on risk factors for CVD, 48 adults enrolled in a three tiered study.⁹⁵ First, participants completed a 3.5day inpatient baseline testing, while consuming an energy balanced diet.⁹⁵ Next, participants completed 12 outpatient days consuming 25% of their energy

requirements via a glucose, fructose or HFCS sweetened-beverage (n=16 per group) with an ad libitum diet.⁹⁵ Lastly, participants completed a final 3.5day inpatient testing, in which they consumed an energy-balanced diet containing 25% energy from sugar-sweetened beverages.⁹⁵ Fasting LDL-C was significantly increased during fructose consumption ($+0.29 \pm 0.082$ mmol/L) and HFCS ($+0.42 \pm 0.11$ mmol/L) but not in the glucose sweetened beverages group.⁹⁵ Participants consuming fructose and HFCS also had higher postprandial concentrations of LDL-C⁹⁵.

To determine if fructose had adverse effects on metabolic outcomes, 14 healthy participants consumed an isoenergetic diet consisting of either 20% energy from fructose or < 3% of energy from fructose with the remaining carbohydrate source from starch for 28 days each⁹⁶. The high starch low fructose diet resulted in decreases in TC and LDL-C after 28 days⁹⁶. The higher fructose diet, from baseline to post treatment, resulted in increases in fasting TC (4.43 ± 0.20 mmol/L to 4.47 ± 0.16 mmol/L, $p < .01$) and LDL-C (2.62 ± 0.17 mmol/L to 2.73 ± 0.13 mmol/L, $p < .01$)⁹⁶. There was a significant difference between consuming a higher fructose diet and consuming a higher starch lower fructose diet with respect to TC and LDL-C post treatment⁹⁶.

A randomized control trial by Lowndes et al.⁵¹ examined the effects of fructose-containing sugars (HFCS and sucrose) on cardiometabolic risk factors. For 10 weeks, 355 overweight and obese participants were placed on a eucaloric diet, which incorporated sucrose and HFCS at either 8%, 18% or 30% of caloric intake⁵¹. The study observed mixed changes in the participants' lipid profile, specifically observing an increase in TAG when pooled as fructose containing sugars⁵¹. This study administered fructose up to the 90th percentile of consumption, equating to levels five

times the recommended amount for consumption of sugars by the American Heart Association and three times the level of the World Health Organization, furthering research with higher consumption levels than what is consumed on average in Americans⁵¹. Despite this, they did not observe any changes in blood pressure, TC, or LDL-C⁵¹. This could be due to the method used for delivering the added sugars. In this study, added sugars were added through a low-fat milk, which increased consumption of vitamin D⁵¹. Vitamin D may contribute to lower LDL-C⁵¹.

A review by Zhang et al.⁴⁷ concluded that higher consumption of fructose (>100 grams) in place of other carbohydrate sources caused higher levels of TC and LDL-C (13.0 mg/dL and 11.6 mg/dL respectively). These results did not emerge when participants were consuming less than 100 grams of fructose daily⁴⁷. Contradictory to this, Silbernagel et al.⁵⁶ conducted a study using very high fructose (150g) and very high glucose (150g) and did not observe any significant changes in TC or LDL-C. This could be due to increased absorption of sugars seen when consumed in a sugar rich meal³⁶.

Using a within subjects cross-over design, healthy participants consumed three meals a day with 30% of caloric intake from either glucose or fructose on two separate visits¹⁰¹. They concluded that plasma TAG concentrations were elevated after ingestion of fructose-sweetened beverages with meals when compared with glucose-sweetened beverages¹⁰¹.

In a longer 4-week long randomized single blind intervention trial the effects of a very high fructose and a very high glucose hyperenergetic diet on plasma lipids and body fat were explored⁵⁶. Despite no changes in body fat, plasma TAG levels

increased in fructose group by 350mg/L⁵⁶. No significant changes were observed in plasma TAG with consumption of a very high glucose diet⁵⁶. Overall, no changes in HDL-C were observed⁵⁶.

The previous studies provided doses of fructose exceeding levels consumed in national populations. A recent cross-sectional analysis in 25,506 participants aged 12-18 years and in participants aged 19-80 years old suggests the median, 95th and 99th percentile for fructose consumption to be 8.65%, 17.78% and 22.8% of caloric intake¹⁷. When consumed in this sample, Sun et al.¹⁷ did not find a significant correlation with TAG. However, a 6 week cross-over study comparing fructose at 17% of diet, about the 95th percentile for fructose consumption versus 14% glucose and 3% fructose TAG concentrations were higher in men consuming fructose⁹⁹.

Several studies have investigated the relationships between TAG concentration and HDL-C¹⁰²⁻¹⁰⁴. A review of HDL mechanisms concluded that the majority of low HDL-C occurs with other clinical features, such as insulin resistance and hypertriglyceridemia¹⁰⁴. It is hypothesized that TAG enrichment of HDL particles with enhanced cholesterol ester transfer protein (CETP)-mediated exchange of TAG and cholesterol esters between HDL and TAG rich lipoproteins, combined with the lipolytic actions of hepatic lipase, are the driving forces for low plasma HDL-C¹⁰⁴. Despite this, there have been instances where HDL-C has been lowered without effects of TAG and vice versa^{102,105}.

In a randomized single blind cross-over study, participants were tested on three different occasions where they consumed either 50grams of fructose, glucose or sucrose with blood collection at 0, 30, 60, and 120 minutes no differences in TAG

were observed after consumption of all three sugars⁹³. Despite no differences observed in TAG, postprandial HDL-C significantly increased⁹³, suggesting that HDL-C can change regardless of a lack of change in TAG.

Total and added sugars in relation to cholesterol levels

To conduct a systemic review and meta analyses of dietary free sugars and lipids, Te Morenga et al.¹⁰⁶ analyzed 39 randomized control trials. When comparing high and low intakes of sugars, higher intake raised TC (mean difference 0.16mmol/L) and LDL-C (mean difference 0.12mmol/L)¹⁰⁶. Cross-sectional studies on NHANES data suggest that added sugars may be detrimental to health^{19,69}. NHANES data from 1999-2006 in adults aged 18 and older show a linear trend between added sugars intake and LDL-C in women, but not men⁶⁹. NHANES data from 1999-2004 in children ages 12-18 years old show a positive correlation between LDL-C and added sugars intake¹⁹. Among the lowest and highest consumers of added sugars, LDL-C was 9% higher in participants with higher intake of added sugars after controlling for several covariates¹⁹. Using more recent NHANES data, 2005-2010, no association with TC or LDL-C were observed⁹.

SSB in relation to cholesterol levels

Data from the Coronary Artery Risk Development in Young Adults (CARDIA) study were used to assess the relations of low- and whole-fat milk, fruit juice and SSB consumption with cardio metabolic risk factors¹⁰⁷. Higher SSB consumption was associated with higher risk of elevated LDL-C and TAG, while intake of whole-fat milk was associated with lower risk of elevated TAG¹⁰⁷.

To observe the effects of SSB on LDL particle size, researchers conducted a prospective, randomized control trial in twenty-nine subjects¹⁰⁸. Subjects participated in six three week interventions where they consumed 600mL SSB of 40 and 80grams of glucose, fructose or sucrose¹⁰⁸. LDL particle size was reduced after high fructose and high sucrose and a more atherogenic LDL subclass distribution was seen when consuming any of the fructose-containing SSB, when compared to glucose¹⁰⁸. Similar shifts in LDL particle size and subclass were seen in a 10-week long investigation where participants consumed 25% energy from with fructose sweetened or glucose sweetened beverages, with only the fructose sweetened beverages altering LDL particle size¹⁰⁹.

Summary

In conclusion, higher consumption of fructose sugars may alter both body fat and blood lipids in healthy participants, although more research is needed. Current consumption levels of sugars are higher than recommendations among all age groups. With high consumption levels, and high rates of obesity and dyslipidemia, more research is needed to determine if there is a relationship between consumption of sugars, specifically fructose, and markers of obesity and dyslipidemia.

APPENDIX B

EXTENDED RESULTS

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Table 1a: Random sampling of women and the associated differences by gender, Sample 1.

	Women			Men			P-value
	n = 55			n = 55			
	Mean	Median	SD	Mean	Median	SD	
Body Fat % ‡	25.9	25.8	6.7	16.3	14.0	8.0	<0.001***
TC (mg/dL)	165.4	165.0	23.3	153.5	150.5	24.4	0.016**
LDL-C (mg/dL)	84.2	82.0	22.1	86.8	82.0	24.4	0.592
HDL-C (mg/dL)	60.2	61.0	13.9	50.5	50.0	11.4	<0.001***
TAG (mg/dL)	107.8	103.5	35.7	83.7	73.0	31.4	0.001***

Table 1b: Random sampling of women and the associated differences by gender, Sample 2.

	Women			Men			P-value
	n = 55			n = 55			
	Mean	Median	SD	Mean	Median	SD	
Body Fat % ‡	25.9	25.8	6.7	16.3	14.0	8.0	<0.001***
TC (mg/dL)	165.4	165.0	23.3	153.5	150.5	24.4	0.016**
LDL-C (mg/dL)	84.2	82.0	22.1	86.8	82.0	24.4	0.592
HDL-C (mg/dL)	60.2	61.0	13.9	50.5	50.0	11.4	<0.001***
TAG (mg/dL)	107.8	103.5	35.7	83.7	73.0	31.4	0.001***

Table 1c: Comparison of the two random samples of women

	Women			Women			P-value
	n = 55			n = 55			
	Mean	Median	SD	Mean	Median	SD	
Body Fat %	25.9	25.8	6.7	28.0	27.7	6.3	0.286
TC (mg/dL)	165.4	165.0	23.3	170.3	169.0	30.5	0.970
LDL-C (mg/dL)	84.2	82.0	22.1	84.4	79.0	28.5	0.620
HDL-C (mg/dL)	60.2	61.0	13.9	61.3	62.0	15.1	0.362
TAG (mg/dL)	107.8	103.5	35.7	112.3	99.0	44.1	0.194

SD= standard deviation ; TC= total cholesterol ; LDL-C= low density lipoprotein cholesterol ; HDL-C= high density lipoprotein cholesterol ; TAG= triacylglycerol ; *p<0.05 ; **p<0.01 ; ***p<0.001

Table 2a: Random Sample of Women for comparison of sugars intake by gender, Sample 1.

	Women			Men			P-value
	n = 55			n = 55			
	Mean	Median	SD	Mean	Median	SD	
Total Sugars, g/day	132.9	118.7	66.3	129.2	125.1	67.3	0.772
(%kcal/day)	(21.8)	(21.9)	(3.8)	(20.1)	(19.4)	(6.4)	0.085
Fructose Sugars, g/day	82.1	77.3	41.2	76.7	70.6	40.3	0.489
(%kcal/day)	(13.5)	(13.6)	(2.8)	(12.0)	(12.1)	(4.3)	0.030*
Non-fructose Sugars, g/day	49.0	45.2	24.3	48.1	45.6	23.6	0.844
(%kcal/day)	(8.3)	(8.3)	(1.9)	(8.0)	(7.9)	(3.0)	0.563

Table 2b: Random sample of women for comparison of sugars intake by gender, Sample 2.

	Women			Men			P-value
	n = 55			n = 55			
	Mean	Median	SD	Mean	Median	SD	
Total Sugars, g/day	127.4	105.9	59.6	129.2	125.1	67.3	0.884
(%kcal/day)	(21.3)	(21.8)	(3.6)	(20.1)	(19.4)	(6.4)	0.230
Fructose Sugars, g/day	79.6	66.3	36.9	76.7	70.6	40.3	0.692
(%kcal/day)	(13.4)	(13.4)	(2.5)	(12.0)	(12.1)	(4.3)	0.046*
Non-fructose Sugars, g/day	47.7	41.6	24.3	48.1	45.6	23.6	0.931
(%kcal/day)	(7.9)	(8.0)	(1.8)	(8.0)	(7.9)	(3.0)	0.809

Table 2c: Comparison of random samples of women on intakes of sugars.

	Women 1			Women 2			P-value
	n = 55			n = 55			
	Mean	Median	SD	Mean	Median	SD	
Total Sugars, g/day	132.9	118.7	66.3	127.4	105.9	59.6	0.684
(%kcal/day)	(21.8)	(21.9)	(3.8)	(21.3)	(21.8)	(3.6)	0.437
Fructose Sugars, g/day	82.1	77.3	41.2	79.6	66.3	36.9	0.738
(%kcal/day)	(13.5)	(13.6)	(2.8)	(13.4)	(13.4)	(2.5)	0.748
Non-fructose Sugars, g/day	49.0	45.2	24.3	47.7	41.6	24.3	0.316
(%kcal/day)	(8.3)	(8.3)	(1.9)	(7.9)	(8.0)	(1.8)	0.266

SD= standard deviation ; *p<0.05 ; **p<0.01 ; ***p<0.001

Table 3a: Risk factors in men.

Risk Factor	Number of Students
Waist Circumference	2
High Triglycerides	3
Low HDL-C	6
High Blood Pressure	28
High Fasting Blood Glucose	0

Table 3b: Number of risk factors in men. (n=55)

Number of Risk Factors	Number of Students (%)
0	23 (41.8)
1	27 (49.1)
2	3 (5.5)
3	2 (3.6)
4	0 (0)

Table 4a: Risk factors in women (n=281).

Risk Factor	Number of Students
Waist Circumference	27
High Triglycerides	40
Low HDL-C	57
High Blood Pressure	34
High Fasting Blood Glucose	0

Table 4b: Number of risk factors in women (n=281).

Number of Risk Factors	Number of Students (%)
0	166 (59.1)
1	83 (29.5)
2	22 (7.8)
3	9 (3.2)
4	1 (0.4)

Figure 1: Comparison of nutrition and non-nutrition majors (n=281).

**p<0.01

Nutrition: n = 147

Non-nutrition: n = 135

Table 5: Pearson correlations between sugars intake as a percentage of carbohydrate intake with body fat and blood lipids.

	n	Total Sugars ‡	n	Fructose Sugars ‡	n	Non-fructose Sugars ‡
Body Fat (%)	293	0.081	293	0.129*	292	0.008
Total Cholesterol (mg/dL)	293	0.023	293	0.008	291	0.076
LDL-C (mg/dL)	272	0.051	272	0.014	271	0.112
HDL-C (mg/dL)	292	0.015	292	0.024	290	0.014
Triglycerides (mg/dL)	268	-0.042	268	-0.029	267	-0.042

‡ Percentage of carbohydrate intake.

*p<0.05

Table 6: Pearson correlation of glycemic index with body fat percent and fasting blood lipids.

	Glycemic Index			
	n	Women	n	Men
Body Fat (%)	258	0.052	47	0.182
Total Cholesterol (mg/dL)	258	0.049	48	-0.118
LDL-C (mg/dL)	240	0.020	45	-0.194
HDL-C (mg/dL)	257	0.106	48	0.106
Triglycerides (mg/dL)	236	0.112	44	0.098

No significant results.

Figure 2: Comparison of sugars intake and the presence of having or not having a metabolic syndrome risk factor (n=334).

*p<0.05

Figure 3: Relationships between having or not having a metabolic syndrome risk factor with respect to BMI and body fat percent.

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

BMI measures in kg/m^2

Body fat percent measured using air displacement plethysmography

Table 7: Reported average times per day recorded by the top 10 male and top 10 female consumers of fructose sugars from the CNAQ of categories of foods that may contain sugars.

		Fruit	Dried Fruit	Dairy	Cereal	Bread	Sugars and Spreads	Beverages	Snacks and Commercial meals	Condiments
Men	Min	7.58	1.8	2.39	0.99	1.7	1.37	3.79	1.45	1.37
	Max	10.9	2.5	3.17	1.36	2.31	1.87	5.5	2.32	2.09
Women	Min	11.48	2.03	3.03	1.57	1.62	2.76	1.52	3.74	1.89
	Max	15.63	2.88	3.78	2	2.44	3.42	2.16	4.64	2.45

Table 8: Reported times per day recorded by the top 10 male consumers of fructose sugars from the CNAQ of categories of foods that may contain sugars.

			Dried Fruit	Dairy	Cereal	Bread	Sugars and Spreads	Beverages	Snacks and Commercial meals	Condiments
1	Min	22.6	6.9	0.6	3	2.9	2	0.8	0.5	0.2
	Max	31.3	10.8	0.9	3.4	3.3	2	1.6	1	0.2
2	Min	19	4.1	1.5	0.4	1.2	0.4	1.2	0.9	1.2
	Max	24.7	4.3	2.5	0.4	2.4	0.9	1.7	2.1	2.3
3	Min	6.7	4.5	1.3	0.8	0.2	0.6	0.6	1.2	1.4
	Max	10.1	5.9	2	1.4	0.3	1.2	1.2	2.4	2.7
4	Min	2.6	0.2	10	0	1.7	4.1	6.1	0.9	0.1
	Max	4.4	0.3	11.1	0	1.9	5.1	8.7	1.1	0.2
5	Min	5.7	0	2.3	0.1	0.3	0.1	10.3	0.3	0.5
	Max	9.7	0	2.8	0.1	0.5	0.3	15.5	0.8	0.9
6	Min	3.1	0.5	0	0.3	2.1	2.5	0.5	5	5.4
	Max	5.4	0.9	0	0.6	2.3	3.1	0.6	5.9	7.1
7	Min	3.4	0.2	2	0.7	1.8	0	4.8	2.4	2.6
	Max	4.9	0.3	3.5	1.3	2.5	0	5.6	3.3	3.7
8	Min	8	1.5	0	4.3	4.8	0.2	0.6	0.9	0.6
	Max	12.1	2.2	0	6	6.6	0.6	1.9	2.7	1.7
9	Min	0.4	0.1	1.7	0.3	0.6	0.4	6.7	1.7	0.2
	Max	1	0.2	2.1	0.3	1.5	0.8	10.7	2.9	0.2
10	Min	4.3	0	4.5	0	1.4	3.4	6.3	0.7	1.5
	Max	5.4	0.1	6.8	0.1	1.8	4.7	7.5	1	1.9

Table 9: Reported times per day recorded by the top 10 female consumers of fructose sugars from the CNAQ of categories of foods that may contain sugars.

		Fruit	Dried Fruit	Dairy	Cereal	Bread	Sugars and Spreads	Beverages	Snacks and Commercial meals	Condiments
1	Min	3.5	0.6	2.6	0.4	0.5	3.4	7.2	3.5	2.9
	Max	5.8	1.2	4.2	0.6	0.9	4.7	9.6	4.9	3.7
2	Min	23	0	4.4	0.5	1	0.4	0.1	0.5	0.8
	Max	31	0	5.8	1	1.5	0.4	0.2	0.9	1.3
3	Min	13.4	4.6	0	0.1	0.1	0.1	0.2	0.1	0.2
	Max	20.2	6.8	0	0.2	0.2	0.1	0.2	0.4	0.2
4	Min	4.7	0.5	8.8	6	1.1	8.1	1.5	3.6	7
	Max	5.8	0.6	9	6.1	1.2	9.3	1.7	4.8	9.1
5	Min	9.4	4.9	0	0.3	0	0.4	0.1	0.3	0.3
	Max	12.2	7.3	0	0.7	0.1	0.7	0.2	0.5	0.5
6	Min	12.1	0.9	3.9	1.2	1.6	3.3	0.9	2.6	1
	Max	17.7	1.1	5.7	1.4	3.3	5	2.1	4	1.8
7	Min	8.7	4	0	0	0.3	0.2	0.7	0.1	0.1
	Max	12.5	5.4	0	0.1	0.7	0.2	1.3	0.3	0.3
8	Min	5.5	2.6	5.1	3.3	3.3	1.7	1.7	1.9	2.2
	Max	7.2	3.3	6	5.6	4.7	2.2	2.2	2.6	1.8
9	Min	26	0.3	45	2.7	3.3	8.2	1	21.9	1.7
	Max	30.9	0.4	5.8	3	3.3	9.2	1.4	23.6	2.1
10	Min	8.5	1.9	1	1.2	5	1.8	1.8	2.9	2.7
	Max	13	2.7	1.3	1.3	8.5	2.4	2.7	4.4	3.7

APPENDIX C

CONSENT FORM

THE
UNIVERSITY
OF RHODE ISLAND

COLLEGE OF
THE ENVIRONMENT
AND LIFE SCIENCES

NUTRITION AND FOOD SCIENCES
112 Ranger Hall, Kingston, RI 02881 USA

p: 401.874.2283 f: 401.874.5874 cefs.uri.edu/info

THINK BIG WE DO

Title of Project: Nutrition Assessment Secondary Data Analysis

INFORMED CONSENT TO PARTICIPATE IN RESEARCH

You are invited to take part in a research project described below. Students enrolled in NFS 210 and NFS 443 currently have anthropometric and biochemical assessments and complete dietary assessment as part of their coursework. These assessments are used for classroom assignments. We are asking you to give us permission to use these data for research. In addition, we are asking you to complete a few additional demographic and dietary questions. The purposes of the research is to validate assessment methodologies and to investigate the relationship between anthropometric, biochemical, and dietary variables that are related to chronic disease risk. If you have questions you may contact the Geoffrey Greene, the person mainly responsible for this study at 874-4028 or email him at ggw@uri.edu.

Description of the Project:

The purpose of the study is to use nutrition assessment data for research to help us understand the relationship between diet and disease risk in college students.

My Participation

You must sign this informed consent form for the data collected as part of this class to be used for research, and must complete the additional brief questionnaire.

What will be done:

If you take part in this study, your information entered into a password protected computer. Your data will be identified by code number only. Once all data have been entered and verified, the link between code number and identifying data will be destroyed. All data analysis will be conducted by code number only. Assessments that we will be using are listed below (these are collected as part of your class and the additional brief demographic questionnaire):

Demographics	✓
Dietary Assessment	✓
Height, Weight	✓
Waist and Hip Circumference	✓
Air Displacement Plethysmography (BodPod)	✓
Sonographic Measurement of the Heel (bone density)	✓
Standard Blood Tests (TG, HDL, LDL, Total Cholesterol, Glucose)	✓

The University of Rhode Island is an equal opportunity employer committed to the principles of affirmative action.

Risks or discomfort:

The risks are minimal. The only risks would be loss of confidentiality and that will be minimized as described below.

Benefits of this study:

You will not receive any direct benefit. Allowing us to use your data and filling out the brief questionnaire will help us with research to better understand the relationship between diet and chronic disease in college students.

Confidentiality:

Your part in this study is confidential. None of the information will identify your name. We will keep all consent forms in a locked cabinet in Room 307 Ranger for five years. All information collected for the class will be identified by code numbers and will not include any link to your name. This information will be confidential.

Decision to quit at any time:

You have been given the opportunity to decide whether or not to participate in this study. Your decision to participate will not affect your grade in the class or your relationship with your class instructor. Your instructor will not know who is participating in this study. You have the right to stop participating at any time, but once data have been entered and verified and the link between participant and code has been destroyed, we will not be able to remove your data.

Rights and Complaints:

If you are not satisfied with the way this study is performed, you may discuss your complaints with Geoffrey Greene (401-874-4028) anonymously, if you choose. In addition, if you have questions about your rights as a research participant, you may contact the Office of Research Integrity, 70 Lower College Road, Suite 2, University of Rhode Island, Kingston, RI, telephone: (401) 874-4328.

You have read this Consent Form. Your questions have been answered. Your signature on this form means that you understand the information and you agree to participate in the study. Please note that you must be at least 18 years of age in order to participate.

Print Your Name: _____

_____ Signature of Participant	_____ Date	_____ Signature of Researcher
-----------------------------------	---------------	----------------------------------

Please sign both consent forms, keeping one for yourself

APPENDIX D

NUTRITION ASSESSMENT SURVEY

Name: _____ Date: _____
Please Print

Nutrition Assessment Study Survey

1. What is your age (in years)?

<18, 18, 19, 20, 21, 22, 23, 24, 24+

2. What is your gender?

Male, female, choose not to answer

3. Which one of the following best applies to you?

-White

-Black or African American

-Hispanic/Latino

-Asian

-Native Hawaiian or other Pacific Islander

-American Indian or Alaskan Native

-Mixed

-Other (please specify):

-Choose not to answer

4. What is your year in school?

Freshman, Sophomore, Junior, Senior, Graduate

5. What is your current major?

- Agricultural Sciences

- Biological Sciences

- Business/Communication

- Education

- Exercise Science/Kinesiology

- Fine Arts/Humanities

- Health/Nursing

- Nutrition

- Social Sciences

- Undeclared

- Graduate Student

- Other (please specify):

6. Place of residence during the academic year?

- On campus
- Off campus

7. Green Eating is: Eating locally grown foods, limited amounts of processed/fast foods, eating meatless meals at least one day per week, choosing organic foods as much as possible, and only taking what you plan on eating.

Are you a green eater?

- No, and I do not intend to start within the next 6 months
- No, but I am thinking about becoming a green eater within the next 6 months
- No, but I am planning on becoming a green eater within the next 30 days
- Yes, I am a green eater and have been for less than 6 months
- Yes, I am a green eater and have been doing so for 6 months or more
- I choose not to answer

8. Which of the following best describes the MAJORITY of your meals during the academic year?

- I eat meals prepared at home.
- I purchase frozen or ready-to-eat meals
- I eat at dining halls/restaurants
- I get fast food/take-out

9. Do you have a campus meal plan?

- Yes
-No

10. What is your usual rate of eating?

Very slow	Slow	Medium	Fast	Very fast
1	2	3	4	5

11. Do you experience abdominal discomfort such as cramping, bloating, or excess gas? (this refers to gastrointestinal discomfort, NOT menstrual discomfort)

- Never or very seldom
- Seldom, less than once per month
- Occasionally, a few times per month
- Fairly often, once or twice per week
- Very often, several times per week or daily

12. If you experience abdominal discomfort , how severe is it?

- I do not experience abdominal discomfort
- Very mild –not very noticeable
- Moderate – noticeable but not too bad
- Somewhat uncomfortable – it's kind of bad, but manageable
- Very uncomfortable – I cannot carry out my normal activities

13. Please select the answer that BEST describes your usual behavior.

	Barely ever to never	Rarely (25%)	Sometimes (50%)	Often (75%)	Almost always
- Locally grown foods are grown within 100 miles of your location. Based on this, how often do you eat locally grown foods?	0	0	0	0	0
- When in season, how often do you shop at farmer's markets?	0	0	0	0	0
- How often do you choose foods that are labeled certified organic?	0	0	0	0	0
- How often do you select meats, poultry, and dairy products that are raised without antibiotics or hormones?	0	0	0	0	0
- How often do you select food or beverages that are labeled fair trade certified?	0	0	0	0	0
- How often do you buy meat or poultry products labeled "free range" or "cage free"?	0	0	0	0	0

APPENDIX E

TRANSLATION SHEET

CNAQ Food Frequency Questionnaire Translation Sheet

Department of Nutrition & Food Sciences, University of Rhode Island, USA

Translation of Australian food terms to American food terms:

1. 'whole meal' = 'whole grain'
2. 250 milliliters (ml) = ~1 cup
3. Fibre = Fiber
4. Spirit = Alcoholic beverage (e.g. whiskey, vodka, rum)
5. Cordial/Squash Drink = Non Alcoholic mixer (usually fruit based, containing sugar and water).
6. Muesli Bar = Granola bar made primarily of rolled oats, fruits, nuts and seeds.
7. Takeaway meal = Fast Food
8. *Chocolate Biscuits = Chocolate cookies and other chocolate confectionaries (e.g. kitkats)
9. *Fruit Biscuits = Cookies made with fruit
10. Beetroot = Beet
11. Silver beet = Chard
12. Capsicum = Red Bell Pepper
13. Rocket = Arugula
14. Swede = Turnip or Rutabaga
15. Broad Bean = Faba or Fava bean
16. *Butter bean = Lima Bean
17. Minced meat = ground meat
18. Yoghurt = Yogurt

Places to include American foods not listed:

1. Greek yogurt - include under 'yogurt'
2. Edamame - include under 'soy products'
3. Almond milk include under 'almonds'
4. Oatmeal - include under cooked cereal, eg porridge

Other foods you do not recognize at all:

Answer 'never' because if you do not know what it is, it is likely that you have not eaten it.

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