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Processed Sugars and Coronary Heart Disease

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Introduction

Consumption of processed sugars has increased two fold over the past thirty years (Marriot, Olsho, Hadden, & Connor, 2010). It is considered to be one of the leading risk factors for hyperglycemia, diabetes mellitus type 2 (DM 2), metabolic syndrome, coronary heart disease (CHD), and dyslipidemia (Marriott, Olsho, Hadden, & Connor, 2010). Increased consumption of processed sugars has created problems in all age groups in relation to their health status (Yeung et. al., 2014). Many children are now battling the epidemics of obesity and metabolic syndrome due to excess consumption of sugars (Marriott et. al., 2010). The Centers for Disease Control and Prevention (CDC) reports that more than a third of the adult American population is considered to be obese with a BMI greater than 30 (Go et. al., 2014). Most adults and children consume their sugars by drinking sugar-sweetened beverages (SSB) such as soda, fruit juice, and dairy products (Chen et al., 2010). Many children are now battling the epidemics of obesity and metabolic syndrome due to excess consumption of sugars (Marriott et. al., 2010). Other forms consumed include sweetened confectionary treats such as cakes, candy, breads, and etc. (Welsh et. al., 2010). Confectionary treats make up less than 40% of the amount of sugar consumed by the population (Welsh et. al., 2010). The American Heart Association (AHA) suggests that less than 15% of total energy consumption should be from sugars (Welsh et. al., 2011).

Increasing incidences of metabolic syndrome, dyslipidemia, HTN, and obesity is leading to increased occurrence of CHD. More than one third of the nation is believed to have metabolic syndrome due to increasing hypertension and abdominal obesity (Chen et. al., 2010).

Dyslipidemia is on the rise with a third of the population having increased low density lipoproteins, triglycerides, and total cholesterol. There are also rising numbers of decreased high density lipoproteins that are significant in diagnosing metabolic syndrome. Hypertension (HTN)

is on the rise with one in three adults having high blood pressure with less than half having their blood pressure under control. More than two thirds of the U.S. population is considered overweight and obese with the problem increasing every year. The combination of factors related to dyslipidemia, metabolic syndrome, obesity, and HTN are all precursor signs of CHD.

Ninety two percent of the population in the U.S. consumes at least one serving of sugar on a daily basis (Marriott et. al., 2010). Since the 1950s sugar intake has doubled in all industrialized nations with increasing incidence of CHD. The World Health Organization (WHO), CDC, AHA, and the American Diabetes Association (ADA) have all announced recommendations for lower sugar intake. The U.S. has become the gold standard for poor dietary choices, and often the "Western Diet" is recognized as the Standard American Diet (SAD), which has become synonymous with increased sugar consumption in both the home and fast establishments (Wang, Y. et. al., 2008). These poor dietary decisions have been promoted by media and major corporations and thus encouraging the nutrient dense foods for the majority of the population (Van Horn et. al., 2010).

Across the U.S., the epidemic of obesity, hyperglycemia, and dyslipidemia is promoted by the intake of increasingly energy dense foods. Energy dense foods are foods that have an increase in the number of calories per serving compared to less energy dense foods that are filled with more nutrients such as fiber and water. Those who eat lower energy dense foods often feel more satiety than those who eat high energy dense foods (Kent et al., 2013). The consumption of sugar stimulates the pleasure center in the brain, which in turn produces an effect similar to those who take opioid medications (Goto et. al., 2012). Foods with higher sugar content (more energy dense foods) are therefore, more addictive than those have less amounts of sugars (less energy dense foods).

Interventions for those who consume energy dense foods have been an increasing concern, in recent years. Health conditions that relate to the consumption of added sugars and energy dense foods are increasing such as HTN, metabolic syndrome, and dyslipidemia. These are often the non-Caucasian populations with Hispanic and African Americans consuming three times as much sugar as the Caucasian population. The African American population consumed more than 50% of their added sugar intake in SSB (Marriott et al., 2010). African American populations were twice as likely to consume sugared soda as compared to sodas with artificial sweeteners, and three times more likely to drink fruit juices over consuming water (Marriott et al., 2010). These numbers coincidentally fall close to the levels of co-morbidity of DM2, metabolic syndrome, CHD, DL, and HTN. “Does consuming larger amounts of sugar increase the chance of co-morbid diseases such as DM2, metabolic syndrome, CHD, dyslipidemia, and HTN?”

Definition of Terms

Throughout this paper the term added sugar will be used in context for sugar intake. Added sugar is defined as a sugar (sucrose, dextrose, glucose, fructose, or high fructose corn syrup (HFCS)) that is added to a food for increased palatability of the food. Added sugars make the food more energy dense thus increasing the caloric intake of the foods.

Theoretical Framework

This paper uses Margaret Newman's theory on Health as an Expanding Consciousness to answer the impending questions and bring understanding to how people interact, eat, and live their daily lives. There is an assumption that there is the possibility of illness without symptoms, even though a person is perceived as healthy. Prevention is the key. Through dietary education

there can be increased education on CHD perception and conscious awareness of health.

Nutritional education helps people to realize that there are consequences to the food choices that are made, like the development of diabetes or heart disease.

Purpose Statement

The purpose of this literature review is to better understand the American diet, and how added sugars can increase the incidence of CHD.

Information sources

To complete the literature review a thorough search was completed from Southern Adventist University online McKee library, databases included CINAHL MEDLINE, PubMed, and Google Scholar. Information was searched using key phrases such as: processed sugars and elevated cholesterol, glycemic index and CHD, CHD and processed sugars, CHD risk factors, DM2 and CHD, metabolic syndrome and risk factors for CHD, and lifestyle changes improving CHD and risk factors. References supplied in articles were also evaluated for potential use.

Literature Review:

American Diet

The American population is known for their easy access to energy dense foods. Energy dense diets have been linked to lower socio-economic status and have higher incidences in African American and Latino ethnicities (Chen et. al., 2010). Added sugar consumption has increased from the 1980s to more than 60% of the daily average consumption today (Welsh et. al., 2011). Reeves et al. (2014) found that those who consumed greater than 25% of their total energy intake from added sugars were on the lower end of the socio-economic scale with the

highest percentages of the population being Hispanic and African American. Ninety two percent of the Hispanic and African American populations had the poorest scores in meeting essential nutrient needs (Reeves et. al., 2014). The largest portion of added sugar consumption is from drinking sugar sweetened beverages (SSB) especially in younger populations. Those aged 2-19 years of age showed a significant increase in daily sugar consumption from 204 kcal/day in 1988-1994 to 224 kcal in 1999-2004 (Wang, Y. et. al., 2007). This was significant in relation to the increasing obesity epidemic, increased incidence of HTN, dyslipidemia and increased incidences of metabolic syndrome in adolescence and young adulthood.

Glycemic Index/Load

The glycemic index (GI) is a scale that measures how a carbohydrate raises one's blood sugar after consumption (Goto et. al., 2012). The scale ranges from 0-100 and the higher the carbohydrate containing food is on the scale the more of a spike there is in the blood glucose (Goto et. al., 2012). Glycemic load (GL) measures the impact the food has on the body and blood glucose with a scale that also ranges from 0-100 (Goto et. al., 2012). Foods that are higher on the index have more dense carbohydrates or sugars and increase the GL of the person consuming the food. The higher the GL intake the higher the response from the body causing a hyperglycemic event that then causes one to surpass their total energy intake for the day (Goto et. al., 2012). The increased glucose levels in the circulatory system create an increase in insulin productivity to counteract the hyperglycemic event (Johnson et. al., 2009). Those who consume foods on the lower end of the GI have decreased consumption of added sugars in relation to the foods being a more complex carbohydrate (Carey et. al., 2011). GL does not necessarily relate directly to the GI, because there are foods that are considered high on the GI that do not produce increased GL (Goto et. al., 2012). The GL is an important consideration and factor in relation to

how the increase affects the body and the problems that arise within the body when the GL is elevated. The extra intake of energy dense foods increases GL and thus creates the hyperglycemic event, and a repetitive action of this event has been linked to insulin resistance and increased incidence of metabolic syndrome (Carey et. al., 2009). An influx of serum glucose creates a cascade of events that leads to increased inflammation within the body and the circulatory system, and these events increase the risk of CHD according to the American Heart Association (Johnson et. al., (2009).

Goto et. al. (2012) evaluated how dietary GI and dietary GL affected the HbA1c on obese Japanese adults. Multiple linear regression models were used to adjust for confounding factors such as age, sex, abdominal adipose tissue, and HTN (Goto et. al., 2012). Goto et. al. (2012) that an increased GL had more effect on HbA1c levels than with GI. The significance for increased GL and increases in HbA1c is $p=0.044$ for those consuming the highest GL having HbA1c $>7.0\%$ compared to those with lower GL consumption for a HbA1c $<7.0\%$.

Melanson et. al. (2012) evaluated GI and GL and how they contribute to metabolic syndrome in overweight obese adults. The study compared three dietary approaches of eating low glycemic foods, portion control, and low energy dense diets (Melanson et. al., 2012). All groups had reductions of body weight and improvements in BMI that were significant ($p<0.001$) (Melanson et. al., 2012). All three diets decreased HDL levels for a significance of ($p<0.001$), with decreases also noted in saturated fat consumption and increases in protein consumption (Melanson et. al., 2012). ~~The results of the study shows that~~ there were improvements with all three dietary changes, and this gives options for improving lifestyle and metabolic syndrome.

Finley et. al. (2010) evaluated how increased GL and GI consumption increased the prevalence of metabolic syndrome. This study was a cross sectional study performed in a clinic in Dallas, TX from October 1987 to March 1999 (Finley et. al., 2010). Results of the study were that men consumed more added sugar by 15% more than women, and all had positive associations ($p < 0.05$) to increased consumption of the high food on the GI and a relation to causative factors for metabolic syndrome (Finley et. al., 2010). Positive associations were made with increased GI and increased triglyceride levels ($p < 0.0001$) with inverse associations in HDLs ($p < 0.0001$). A relation to cardiorespiratory fitness was also related to increased abdominal girth and dyslipidemia for a significance of $p < 0.05$ (Finley et. al., 2010). With improved lifestyle changes and decreased consumption of foods high on the GI an observation of improved symptoms of metabolic syndrome were seen.

Co-Morbid Diseases and Risk Factors

Metabolic Processes and Dyslipidemia

The body breaks down the easier to burn fuel first, such as carbohydrates. The body also expends these more easily metabolized fuels before expending more complex fuels such as proteins and complex carbohydrates (Porth, 2011). The simpler the carbohydrate, such as simple sugars or added sugars the higher the increase of serum glucose within the circulatory system (Porth, 2011). The glucose molecules are large molecules that can cause damage to the circulatory system, especially arterial damage (Porth 2011). The large molecules collide into arterial walls causing the inflammatory process to activate to repair the damage within the arterial vessels (Porth, 2011). This inflammatory process creates plaques over the damaged areas

to prevent further damage thus decreasing the size of the lumen of the vessel (Porth, 2011). The narrowing within the vessel increases the internal pressure of the circulatory system creating a co-morbid factor of HTN with increased cardiac workload (Porth, 2011).

Adverse changes in lipoproteins have been noted in the Coronary Artery Risk Development in Young Adults Study or CARDIA (Duffey et. al., 2010). Diets with increased added sugar intake have shown increased association with waist circumference over a twenty year in the CARDIA study (Duffey et al, 2010). Increased waist circumference had an adjusted relative risk (aRR) of 1.099; 95 % CI: 1.04,1.14; P for trend <0.0001, and increased low density lipoprotein (LDL) cholesterol results were (aRR: 1.18; 95% CI: 1.02,1.36; P=-0.018)(Duffey et. al., 2010). In a fully adjusted linear model Welsh et. al. (2011), discussed how an increase in lipid levels has coincided with an increased intake of added sugar from the NHANES III survey.

Table 1

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

| | % Total Energy From Added Sugars | | | | | | P Linear Trend |
|---------------------------|-----------------------------------|----------------------|----------------------|----------------------|----------------------|---------------------|----------------------|
| | 0% to 10% (referent) (n300) | 10% to 15% (n364) | 15% to 20% (n425) | 20% to 25% (n369) | 25% to 30% (n303) | 30% (n396) | |
| Model 1 | | | | | | | |
| Lipid measures, mmol/L | | | | | | | |
| HDL cholesterol | 1.40(1.36 to 1.44) | 1.35(1.30 to 1.40) | 1.31†(1.27 to 1.35) | 1.32*(1.27 to 1.36) | 1.24§(1.19 to 1.29) | 1.28†(1.23 to 1.33) | 0.001 |
| LDL cholesterol | 2.24(2.12 to 2.37) | 2.27(2.16 to 2.37) | 2.37*(2.31 to 2.44) | 2.51*(2.35 to 2.66) | 2.42(2.29 to 2.55) | 2.44(2.34 to 2.53) | 0.01 |
| TC | 4.05(3.92 to 4.19) | 4.04(3.94 to 4.15) | 4.11(4.02 to 4.19) | 4.27(4.11 to 4.43) | 4.12(3.99 to 4.25) | 4.16(4.05 to 4.27) | 0.16 |
| Triglycerides | 0.81(0.74 to 0.88) | 0.83(0.78 to 0.89) | 0.84(0.82 to 0.87) | 0.87(0.82 to 0.93) | 0.90(0.84 to 0.97) | 0.89(0.83 to 0.96) | 0.05 |
| Model 2 | | | | | | | |
| HOMA-IR | | | | | | | |
| Not overweight | 2.70(2.06 to 3.33) | 2.73(2.11 to 3.36) | 2.71(2.09 to 3.34) | 2.77(2.12 to 3.41) | 2.91(2.23 to 3.58) | 2.74(2.11 to 3.37) | 0.41 |
| Overweight | 3.49(3.02 to 3.95) | 3.65(3.15 to 4.16) | 4.17*(3.86 to 4.47) | 4.74†(4.07 to 5.41) | 4.34*(3.81 to 4.86) | 4.61†(4.08 to 5.13) | 0.004 |
| Insulin (fasting), pmol/L | | | | | | | |
| Not overweight | 78.5(59.9 to 97.0) | 80.1(62.2 to 98.0) | 78.5(62.2 to 97.1) | 80.9(62.2 to 99.5) | 84.6(79.6 to 89.6) | 80.7(62.7 to 98.7) | 0.33 |
| Overweight | 108(96.0 to 121) | 112(97.9 to 126) | 127*(122 to 136) | 140*(122 to 159) | 130*(115 to 145) | 139†(124 to 155) | 0.006 |
| Glucose (fasting), pmol/L | | | | | | | |
| Not overweight | 5.36(5.18 to 5.55) | 5.33(5.14 to 5.52) | 5.42(5.17 to 5.63) | 5.37(5.17 to 5.57) | 5.44(5.24 to 5.65) | 5.35(5.12 to 5.57) | 0.54 |
| Overweight | 5.03(4.91 to 5.15) | 5.04(4.95 to 5.14) | 5.09(5.04 to 5.15) | 5.15(5.04 to 5.26) | 5.14(5.06 to 5.22) | 5.08(4.99 to 5.18) | 0.16 |

| | | | | | | | |
|-------------------------------|------------------|------------------|------------------|------------------|-------------------|-------------------|------|
| Systolic blood pressure, mmHg | | | | | | | |
| Not overweight | 89.6(83.4to95.9) | 90.9(84.8to97.0) | 90.8(84.6to97.0) | 90.6(83.4to97.8) | 93.1†(86.9to99.2) | 91.3(85.0to97.5) | 0.07 |
| Overweight | 110(108to113) | 112(110to114) | 112(110to115) | 113(110to115) | 114*(112to117) | 114(111to116) | 0.11 |
| Waist circumference, cm | | | | | | | |
| Not overweight | 47.2(44.7to49.8) | 48.5(46.3to51) | 48.5(46.1to50.8) | 48.2(46.1to50.4) | 47.9(45.6to50.3) | 48.7‡(46.5to50.9) | 0.31 |
| Overweight | 93.6(92.3to94.8) | 94.2(92.8to95.6) | 92.6(91.5to93.8) | 94.5(93.2to95.9) | 93.7(92.4to95.0) | 92.3(90.7to93.8) | 0.52 |
| BMI, z-score | | | | | | | |
| Not overweight | 0.32(0.00to0.90) | 0.41(0.00to1.00) | 0.30(0.00to0.85) | 0.28(0.00to0.87) | 0.21‡(0.00to0.76) | 0.44(0.00to0.96) | 0.92 |
| Overweight | 1.65(1.54to1.76) | 1.80(1.67to1.92) | 1.65(1.57to1.74) | 1.72(1.60to1.85) | 1.73(1.61to1.84) | 1.88‡(1.77to2.00) | 0.07 |

BMI is adjusted for age and sex.

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

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

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

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

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

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

Welsh, J., Sharma, A., Cunningham, S., & Vos, M. (2011).

The body metabolizes different types of sugar (i.e. sucrose, fructose, dextrose, glucose, and HFCS) at a different rate creating differing amounts of free radicals upon consumption. The more concentrated the GL the higher the variation would be with a glycemic response.

Stanhope et. al. (2008) found that there were minimal differences in metabolic responses in consumption of HFCS and sucrose in a study of metabolic profiles of 34 adult participants.

Stanhope et. al. (2008) found that naturally occurring sugars had minimal increases in metabolic responses to natural occurring sugars such as fructose and glucose compared to the increased metabolic responses noted to HFCS and sucrose. This means that there is a higher GL for HFCS and sucrose containing foods compared to the natural occurring sugars creating a higher metabolic response and consequently an increased inflammatory response (Stanhope et. al., 2008). Comparisons by Stanthope et. al. (2008) found that there were no significant differences between the consumption of HFCS and sucrose for a difference of $p < 0.001$ for those <35 years

of age and $p=0.05$ for those who were >35 years of age. Consumption of both HFCS and sucrose created abnormal triglyceride levels for results of (sucrose $+28.3\pm 5.4$ mg/dL, $P<0.001$; HFCS $+18.9\pm 4.5$ mg/dL, $P<0.001$)(Stanhope et. al., 2008). Consuming natural fructose there was an observation of a significant decrease in insulin spikes ($p<0.001$) compared to those of sucrose (Stanhope et. al., 2008). The overall results showed that sucrose had a higher spike in insulin with consumption followed closely by HFCS (Stanhope et. al., 2008).

Hirshberg et. al. (2011), evaluated college students and risk factors for chronic diseases. Evaluation was performed at Rhode Island University where ~~findings were~~ most students consumed 24% of total calories in added sugar, and at least one SSB was consumed daily (Hirshberg et. al., 2011). All added sugars were correlated for $p<0.05$ with HDL levels being decreased, with a correlation in an increase in total cholesterol levels for a significance of $p<0.05$ (Hirshberg et. al., 2011). This relationship of cholesterol levels could be linked to atherosclerotic changes in young adulthood leading to problems in adulthood, such as CHD.

Welsh et. al. (2010) evaluated how caloric sweetener affected adults in the United States. This was a cross-sectional study of US adults from the NHANES surveys from 1999-2006. Welsh et. al. (2010) evaluated mean HDL cholesterol, triglycerides, and LDL cholesterol. The mean lows were: HDL (40mg/dL for men and 50 mg/dL for women), LDL (> 130 mg/dL for both men and women), and triglycerides (>150 mg/dL). Welsh et. al. (2010) found that the average consumption of added sugars for adults 15.8% of total consumed calories. Those who consumed 17.5% or more of their total energy intake in added sugar had decreased HDL levels with the average of 47.7 mg/dL adjusted mean, and had triglyceride levels that were elevated but the highest average adjusted mean was 114mg/dL for the largest consuming group of consumers who consumed more than 25% of total consumption in added sugars (Welsh et. al., (2010). LDL

levels were higher in the groups that consumed >25% of added sugar in total calories with the highest average LDL of 123mg/dL for both sexes (Welsh et. al., 2010). Those who consumed >10% of their total daily energy intake had odds of decreased HDL by 50-300% compared to the lowest consumers of <5% of total intake (Welsh et. al., 2010). This is a statistical significance of added sugar consumption and increases in dyslipidemia and increased risk for CHD.

Bantle et. al (2000) performed a 42 day study that used crystalized glucose and fructose to evaluate effects on plasma lipid levels. The crystalized fructose consisted of 17% of total caloric energy intake while the crystalized glucose provided 3% of the total caloric energy intake (Bantle et. al., 2000). Both crystalized sugars were sprinkled over regular meals to compare changes in plasma lipid levels (Bantle et. al., 2000). The plasma lipid level changes can be observed in Table 2. The most significant finding was that male subjects on day 28 had a significantly higher plasma cholesterol levels for the crystallized fructose diet compared to the glucose diet (p<0.001) (Bantle et. al., 2000). Bantle et. al (2000) learned men and women metabolize added sugar differently, and that men were more likely to have higher plasma lipid levels in relation to the added sugar consumption, (p=0.0004).

Table 2

Effectsofthe2studydietsonmeanfastingplasmalipids¹

| | | Day | | | | | |
|---|-----------------------|-------|-------|-------|--------|-------|-------|
| | | 7 | 1 | 21 | 28 | 3 | 42 |
| Plasmacholesterol(mmol/L) ^{2,3} | | | | | | | |
| | Fructosediet | 4.66 | 4.5 | 4.45 | 4.61 | 4.5 | 4.30 |
| | Glucosediet | 4.58 | 4.4 | 4.33 | 4.30 | 4.4 | 4.22 |
| | <i>P</i> ⁴ | 0.174 | 0.15 | 0.031 | <0.001 | 0.22 | 0.16 |
| PlasmaLDLcholesterol(mmol/L) ² | | | | | | | |
| Calculated ⁵ | | | | | | | |
| | Fructosediet | 2.75 | 2.67 | 2.59 | 2.69 | 2.69 | 2.49 |
| | Glucosediet | 2.69 | 2.59 | 2.51 | 2.49 | 2.62 | 2.49 |
| | <i>P</i> ⁴ | 0.399 | 0.256 | 0.122 | <0.001 | 0.174 | 0.756 |
| Measured ³ | | | | | | | |
| | Fructosediet | — | — | — | 2.75 | —2.54 | |
| | Glucosediet | — | — | — | 2.51 | — | 2.56 |
| | <i>P</i> | — | — | — | <0.001 | — | 0.658 |

| | | | | | | | |
|--|-------|-------|-------|-------|-------|-------|--------|
| Plasma HDL cholesterol (mmol/L) ^{2,6} | | | | | | | |
| Fructose diet | 1.40 | 1.35 | 1.37 | 1.37 | 1.35 | 1.30 | |
| Glucose diet | 1.42 | 1.40 | 1.35 | 1.37 | 1.35 | 1.30 | |
| <i>P</i> ⁴ | 0.363 | 0.077 | 0.516 | 0.897 | 0.488 | 0.965 | |
| Plasma triacylglycerols (mmol/L) ⁷ | | | | | | | |
| Women | | | | | | | |
| Fructose diet | | 0.96 | 0.9 | 0.9 | 1.0 | 0.94 | 0.93 |
| Glucose diet | | 0.93 | 0.8 | 0.9 | 0.9 | 1.04 | 0.97 |
| <i>P</i> ⁴ | | 0.706 | 0.29 | 0.96 | 0.81 | 0.226 | 0.63 |
| Men | | | | | | | |
| Fructose diet | | 1.34 | 1.3 | 1.2 | 1.3 | 1.27 | 1.25 |
| Glucose diet | | 1.11 | 1.1 | 1.0 | 1.0 | 1.10 | 0.95 |
| <i>P</i> ⁴ | | 0.005 | 0.01 | 0.10 | 0.00 | 0.043 | <0.001 |

¹The means for each endpoint have a common SE based on the appropriate repeated-measures ANOVA error term.

²To convert to mg/dL, multiply by 38.6.

³SE=0.05.

⁴Because 6 paired comparisons of this endpoint were made, only $P < 0.008$ ($0.05/6$) should be considered significant at the 0.05 level (see Statistical analysis section).

⁵SE=0.04.

⁶SE=0.03.

⁷To convert to mg/dL, multiply by 88.5. SE=0.06.

Bantle, J. P., Raatz, S. K., Thomas, W., & Georgopoulos, A. (2000).

Lee et. al. (2014) evaluated whether added sugar consumption had an effect on lipid levels, for white females ages 10-19. Those who consumed less than 10 % of their total energy from added sugar had an increase in their HDL levels by 0.3 mg/dL compared to those who ate more than 25% of their energy from added sugars (Lee et. al., 2014). The highest consumers had a decrease in HDL cholesterol by .28 mg/dL (Lee et. al., 2014). Those with highest HDL levels were those who consumed less than 10% of their total diet in added sugar with significance of $p=0.03$ (Lee et. al., 2014). By the time the participants reached 19 years of age, those who consumed less than 10% of their diet in added sugar had HDL averages of 57.3 mg/dL (Lee et. al., 2014). Those with higher consumption of added sugar, more than 10%, had an average of 55.8 mg/dL for a significance of $p=0.04$. These results signify that those who consumed higher levels of added sugar had higher incidence of dyslipidemia. This places those with increased consumption of added sugar at higher risks for CHD.

Aeberli et. al. (2011) evaluated low to moderate consumption of impaired glucose and lipid metabolism, and how it promotes inflammation in healthy men in a random controlled trial. Low SSBs were defined as 40G of glucose, sucrose, and fructose beverages, and high levels were defined as 80G of glucose, sucrose, and fructose beverages (Aeberli et. al., 2011). The results of the random trial were a decrease in LDL particle size with consumption of high fructose and high sucrose beverages (Aeberli et. al., 2011). The smaller the LDL particle size the higher the increase incidence for atherosclerotic changes within the vessel (Aeberli et. al., 2011). Increased fasting blood glucose levels by increased 4-9% and inflammatory markers of C-reactive protein (CRP) levels increased by 60-109% with a significance of $P < 0.05$ (Aeberli et. al., 2011). This is significant because the increase in fasting blood sugar and CRP levels are causative factors for increased rates of dyslipidemia.

Hypertension

Hypertension (HTN) is increasing across the United States with more than 50% of diagnosed cases being uncontrolled (Chen et. al., (2010). The PREMIER study was performed over a time period of 18 months in four US study centers (Baltimore, MD; Baton Rouge, LA; Durham, NC; Portland, OR) (Chen et. al., 2010). Three interventions were used to evaluate improvements in blood pressure and they were: advice only, Dietary Approaches to Stop Hypertension (DASH), and a behavioral intervention group (Chen et. al., 2010). The results were evaluated at 3, 6, 12, and 18 months. Chen et. al. (2010) found that reducing SSB intake to one serving per day reduced systolic blood pressure (SBP) by an average of 1.8 mmHG with a reduction in diastolic blood pressure (DBP) by an average of 1.1 mmHG. These reductions were unrelated to weight loss, and showed that reducing SSB intake reduced blood pressure significantly ($P < 0.05$) (Chen et. al., 2010). Those with the highest amounts of SSB

consumption, especially soda, had higher incidences of HTN, metabolic syndrome, obesity, dyslipidemia, diabetes mellitus, and CHD (Chen et. al., 2010).

Obesity and Metabolic Syndrome

Obesity and metabolic syndrome have a synergistic relationship. Obesity is the new epidemic of the United States with more than one third of the population being obese (Duffey et. al., 2010). The increase in adipose tissue predispose many to metabolic syndrome and increase risk factors for the syndrome. . Criteria for diagnosing metabolic syndrome is meeting three of the five criteria of: decreased HDL, HTN, increased abdominal adipose tissue, increased serum glucose levels, and increased triglycerides (Duffey et. al., 2010). Duffey et. al. (2010) evaluated the CARDIA (Coronary Artery Risk Development in Young Adults) and found trends of increased waist circumference, triglycerides, LDL, hypertension, metabolic syndrome, and decreased HDL levels in those who consumed SSBs (Duffey et. al., 2010). Those who consumed SSB had increased symptomology related to metabolic syndrome.

SSBs have been linked to increases in body weight in adults, and this is also true for adolescents. Ebbeling et. al. (2012) studied 224 overweight obese adolescents who regularly consumed SSBs, and performed an experiment for one year to decrease the consumption of the SSBs. Ebbeling et. al. (2012) wanted to evaluate the rate of weight gain in the adolescent population in relation to consumption of SSBs. Results were significant at one year ($p=0.01$) for increased body weight and obesity for those who consumed more SSBs (Ebbeling et. al., 2012) Ebbeling et. al. (2012) also noted differences in BMI in relation to ethnicity, Hispanics and African Americans, with significance of $p=0.007$ for the Hispanic population. The reductions in

the BMI were related to reductions in SSB consumption with the change at one year being significant for $p=0.007$ (Ebbeling et. al., 2012).

Firefighters in Buffalo had significant reductions in risks for metabolic syndrome following a low glycemic diet and exercise program. These reductions were decreased rates of HTN, waist circumference, and blood glucose in 57% of all participants (Carey et. al., 2011). Initially there were 71% of firefighters over six fire houses that had a high GL with an increase in their HbA1c and waist circumference increasing the risk factors for metabolic syndrome (Carey et. al., 2011). The results had more relation to the amounts of dense sugars or carbohydrates that were being consumed rather than how the food fell in relation to the GI (Carey et. al., 2011). Improvements in dietary choices by decreasing consumption of foods high on the GI, provided significant improvements in symptoms of metabolic syndrome ($p<0.001$)(Carey et. al., 2011).

Kent et. al. (2013) evaluated how a low-fat plant based lifestyle affected HDL levels and metabolic syndrome through a cohort study. A Complete Health Improvement Program (CHIP) uses lifestyle interventions (using a low-fat plant based diet) for thirty days to evaluate changes in blood pressure, BMI, lipid profiles, and fasting plasma glucose (Kent et. al., 2013). Results of the study were decreases in HDL levels by 8.7% for a significance of $P<0.001$, but there were reductions in BMI by 3.2%, blood pressure by 5.2%, triglycerides by 7.7%, and total cholesterol by 11.1% (Kent et. al., 2013). These were important improvements with some concern over decreasing HDLs, but further evaluation should be considered for better understanding of the finding (Kent et. al., 2013). Most metabolic symptoms improved ,but the decrease in HDL levels had adverse implications in the study.

Coronary Heart Disease

Previous studies listed above have shown implications for increased risks of CHD related to increased intake of added sugar. Liu et. al. (2000) linked GL and risks for CHD in women. A cohort study of women aged 38-63 years of age was performed and followed for ten years (The Nurse's Health Study) for evaluation of CHD and other risk factors (Liu et. al., 2000). Results were that the higher the GL the higher the increase in CHD risk factors for a significance of $p < 0.0001$ (Liu et. al., 2000). This was seen mostly in women with higher BMIs $> 23 \text{ kg/m}^2$ (Liu et. al., 2000). This linked increased BMI and GI to risk factors for CHD with elimination of confounding factors such as smoking and sedentary lifestyles (Liu et. al., 2000).

Wang, H. et. al. (2013) evaluated the Minnesota Heart Survey to determine trends in added sugar intake and heart disease. The surveys were performed from 1980-1982 and then again in 2007-2009 (Liu et. al., 2013). Increases in BMI were found to occurred with increased added sugar intake, which increased by 54% for a significance of $p < 0.001$ (Liu et. al., 2013). Women had decreased added sugar intake related to men, and also had decreases in BMI in relation to men for a significance of $p < 0.01$ (Liu et. al., 2013). The results were consistent for increases in CHD, and added sugar intake with increased abdominal tissue within the study.

Fung et. al. (2009) linked SSB consumption and risks for CHD in women by evaluating results from the Nurse's Health Study. There was a 24 year follow up that evaluated BMI, energy intake, diabetes, and CHD cases (Fung et. al., 2009). Those who consumed more than 2 SSBs per day were found to having increased symptoms of metabolic syndrome, diabetes, and HTN ($p < 0.00$) (Fung et. al., 2009). Regular consumption of SSB had higher incidence of CHD risk in women even after adjustments for confounding factors (Fung et. al., 2009).

Yang et. al. (2014) examined time trends of added sugar consumption and associations to CHD and mortality. The design was a prospective cohort study of the NHANES (1988-1994, 1999-2004, and 2005-2010) for increased added sugar intake and increased rates of CHD and mortality (Yang et. al., 2014). Most adults consumed 10% or more of their daily total energy intake in added sugars which is more than the recommended daily value for added sugar (Yang et. al., 2014). The significance of this is that those who consumed higher amounts of added sugar (more than 10%) was $p < 0.001$ for increased incidence in CHD symptoms and events (Yang et. al., 2014).

The AHA has taken on a stance against added sugar and has released guidelines and statements related to reduction of added sugar intake. Johnson et. al. (2009) released a scientific statement related to decreasing added sugar intake related to increased incidence of obesity, dyslipidemia, diabetes, metabolic syndrome, and HTN. The statement focused on added sugar consumption being ten percent or less of total dietary intake related to total energy intake for the day (Johnson et. al., 2009). Van Horn et. al. (2010) also released a translation of implementation on added sugar consumption and limitations and improvements in health status related to decreased consumption to added sugar. This translation evaluated many forms of added sugar from SSBs to confectionary treats such as cakes, candy, and other forms of sweetened goods. The conference for the translation and implementation of added sugar consumption challenged food producers to add new labels for added sugar intake for consumers to review before purchasing (Van Horn et. al., 2010).

Limitations

There were several limitations to the study with one of the main weaknesses being the majority of the studies were second hand surveys, but they were from nationally accredited sources. Another limitation is not having enough randomized trials for the effects of added sugars or processed sugars to the body. More information should be obtained and studied to assess direct effects on added sugars and the inflammatory process over longer trial periods, and what damage occurs during the process. Additionally research should also be undertaken to evaluate how long term use of added sugar effects the system and increases chances for CHD. Randomized control trials should be further utilized to assess the symptomology of CHD and the prevalence of co-morbidities in relation to CHD. Further research should be performed on the pitfalls of the American diet, and on how lifestyle modifications can improve CHD risk factors with a relation to co-morbid conditions. Not every sugar is created equal and further research could be important on evaluating the concept that not every sugar is created equal. Differences on how different types of sugar and whether they are natural or processed should be evaluated further in-depth studies to determine the differences of consuming natural and unnatural added sugar.

Additional studies on reducing symptomology of CHD and all co-morbidities would be beneficial for combined treatment and improved education on relation of co-morbid diseases. A multitude of factors has to be considered when evaluating lifestyle and how to modify and improve lifestyles for better results. It is becoming more and more apparent that prevention is the key to longevity and better health outcomes rather than treatment after the fact. More research is needed to evaluate the system of how we prevent chronic disease especially CHD that is the highest rated killer in the Western world today. Overall there is a very large correlation of added sugars and processed foods with the development of CHD. With DM2 and CHD being more and

more prevalent together there is now a larger concern of what and how we eat and what this consumption does to our bodies. Eating more fresh fruits, vegetables, whole grains, and consuming lessening amount of animal protein is the most current and effective prevention at this time and added to regular physical activity regimen can prevent and reverse damage from risk factors already in progress. A decrease of the glycemic load could be a better descriptor in prevention of not only CHD, but many co-morbid conditions such as metabolic syndrome and DM2. A further evaluation should be performed on how fresh fruits, vegetables, legumes, nuts, and berries effect the body as a whole and if some are more cardio protective than others giving a better idea on how to educate and push further into the future for better prevention of diseases that are secondary to poor dietary behaviors and sedentary lifestyles. This information could help increase knowledge on what to eat and what not to eat to give more guidelines and broaden the understanding of dietary prevention as a whole.

Conclusion

Added sugars have been associated with increased risk factors for CHD. Increases in dyslipidemia, HTN, metabolic syndrome, and obesity have been linked to increases in CHD. The AHA is identifying how added sugars affect the body and increase the chance for CHD, and is releasing recommendations for dietary intake for prevention. With CHD being the leading cause of death in America, taking a closer look at what we consume and the effects they have on our body is something to plan for in the future. Guidelines should be evaluated for adults and children to help prevent co-morbid factors leading to CHD. Further evaluation should be made on how prevention provides better outcomes than modern medicine. Many studies have evaluated the effects of added sugar especially added sugar intake through SSBs, such as the Minnesota Heart Study and the Nurses' Health Study. These studies have provided results

significant to causation of CHD in relation to increased added sugar intake and how the increased intake creates symptoms that are causative factors for CHD. Reductions in symptomology can create reductions in CHD, so by reducing the causative factors such as dyslipidemia, metabolic syndrome, HTN, and obesity a reduction is seen in CHD. The disease process can be controlled by reducing added sugar intake to a level of recommended daily values and focus should shift towards educating the public to improve dietary habits for improved incomes. Education and lifestyle modifications are important in improving outcomes. Improving lifestyles and education should become the primary focus for care of the person, not only for prevention of CHD, but also other co-morbid diseases. CHD is a disease that can be prevented and more research and education should be provided to the American public. Considerations should be made for those at greatest risk for CHD, and education should be provided for consumption of foods and added sugar intake. The United States spends billions of dollars on diseases that are preventable, and only a small portion of this money focuses on prevention. Prevention is the key to success, and through education and pro-active prevention, a preventable disease can be avoided.

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Welsh, J., Sharma, A., Cunningham, S., & Vos, M. (2011). Consumption of Added Sugars and Indicators of Cardiovascular Disease Risk Among US Adolescents. *Circulation*, 123(3), 249-257.

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Wolff, E., Vergnes, M. F., Portugal, H., Defoort, C., Amiot-Carlin, M., Lairon, D., & Nicolay, A. (2011). Cholesterol-absorber status modifies the LDL cholesterol-lowering effect of a mediterranean-type diet in adults with moderate cardiovascular risk factors. *Journal of Nutrition*, 141(10), 1791-1798.

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[Yang, Q., Zhang, Z., Gregg, E., Flanders, W., Merritt, R., & Hu, F. \(2014\). Added Sugar Intake and Cardiovascular Diseases Mortality Among US Adults. *JAMA Internal Medicine*, 174\(4\), 516-524.](#)

doi:10.1001/jamainternmed.2013.13563

Capstone Matrix

| Reference | Purpose Objective Hypothesis | Patients Populations Sample | Interventions Identify Independent and Dependent Variables | Outcomes/Findings | Level of Evidence |
|---|--|--|---|--|----------------------|
| <p>Aeberli, I., Gerber, P., Hochuli, M., Kohler, S., Haile, S., Gouni-Berthold, I., & ... Berneis, K. (2011). Low to moderate sugar-sweetened beverage consumption impairs glucose and lipid metabolism and promotes inflammation in healthy young men: a randomized controlled trial. <i>American Journal Of Clinical Nutrition</i>, 94(2), 479-485. doi:10.3945/ajcn.111.013540</p> | <p>The aim of the study was to investigate the effect of five different SSBs containing fructose, glucose, or sucrose, in amounts likely to be consumed in everyday life and over a limited time period, on lipid and glucose metabolism with a particular focus on LDL particle size and inflammatory markers in healthy young men.</p> | <p>29 healthy normal-weight male volunteers between 19-25 years of age. Selected through advertisements in Zurich on college campuses and volunteers could not take regular medication or consumed more than 60g SSB a day.</p> | <p>Intervention was adding SSB to the diet with fructose, glucose, or sucrose and in this five different variations of low levels to high levels were used.</p> <p>The dependent variable is the blood sugar testing and inflammatory makers.</p> <p>The independent variable is the amount of sugar and type of sugar consumed by the participant over the four week trial period, with the types of sugar being sucrose, fructose, and high fructose corn syrup (HFCS) in medium and high concentrations. Medium was 40 g of glucose, fructose, and HFCS and 80g was high consumption of glucose, fructose, and HFCS.</p> | <p>Whereas a significant change in waist to hip ratio was significantly higher in all interventions containing fructose (range: 0.92 ± 0.05 to 0.93 ± 0.05) compared with baseline ($0.92 + 0.06$) ($p < 0.0083$).</p> | <p>Level 1</p> |
| <p>Chen, L., Caballero, B., Mitchell, D., Loria, C., Lin, P., Champagne, C., & ... Appel, L. (2010). Reducing consumption of sugar-sweetened beverages is associated with reduced blood pressure: a prospective study among United States adults. <i>Circulation</i>, 121(22), 2398-2406. doi:10.1161/CIRCULATIONAHA.109.911164</p> | <p>The primary objective of this study is to prospectively examine a relationship between changes in SSB consumption and BP among US adults and monitor whether a change in consumption of diet beverages is associated with BP.</p> | <p>Is a prospective study that participated in the PREMIER study as a randomized trial designed with ages ranging from 25-79 yrs of age from four separate cities across the US (Portland, New Orleans, Raleigh Durham, and Baltimore)</p> | <p>The intervention is one of three ranging from advice only and behavioral intervention groups involved n DASH (dietary approaches to stop hypertension) and then a behavior group involving both DASH and physical activity.</p> <p>The independent variable is blood pressure and the relation it has to SSBs and lifestyle are the dependent variables.</p> | <p>Change in SSB consumption was strongly and positively associated with SBP and DBP in both age-adjusted and multivariate-adjusted models. Increase in BP was seen even in study groups who did not have caffeine in the SSBs and wt increases were also seen across the board in the non-intervention group. With reduction of 1 SSB/day of 12 floz there was a SBP decrease of 10.9 average with a p score of <0.001 and DBP decrease of the same with a p score of</p> | <p>Level 1</p> |

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| | | | | <0.01. Change at the 18 month mark was average of SBP decrease of 5.4±6.5 and DBP decrease of 5.6±6.8. | |
| Eriksson, K., Westborg, C., & Eliasson, M. (2006). A randomized trial of lifestyle intervention in primary healthcare for the modification of cardiovascular risk factors. <i>Scandinavian Journal Of Public Health, 34</i> (5), 453-461. | To prove lifestyle interventions are the primary healthcare modification for improvement of cardiovascular risk factors. | Subjects were chosen from Bjorknas primary healthcare center in Boden, Sweden and were required to have a diagnosis of dyslipidemia, type 2 diabetes, obesity, or any combination thereof in patients aged 18-65 y/o, but exclusions were CHD, stroke, TIA, severe hypertension or crisis, dementia, or severe psychiatric disease. | The intervention is lifestyle changes for health improvement with diet and exercise and stress management. The independent variable is improved health from lifestyle changes where the dependent variables are dietary modifications and exercise especially cardio and strength training. | The study had significant results (BMI had decrease of -0.5±1.0, wtredctioin of -1.5±2.8. Bp reduction of sBP -4.7±10.5, DBP -3.8±5, Triglyceride decrease of -.28±11.07, FBS decrease of .08±.49, A1C decrease of .54±.74, HDL decrease of .03±.19, LDL decrease of .36±.83 percentages in diet and exercise) across the board with improvements and favorable outcomes related with the lifestyle interventions especially with the combination of diet and exercise therapy and very significant improvements compared to the control who received one day education and pamphlets. | Level 1 |
| Fung, T., Malik, V., Rexrode, K., Manson, J., Willett, W., & Hu, F. (2009). Sweetened beverage consumption and risk of coronary heart disease in women. <i>American Journal Of Clinical Nutrition, 89</i> (4), 1037-1042. doi:10.3945/ajcn.2008.27140 | To examine whether with CHD, and whether the relation is independent of obesity and diabetes, we prospectively assessed intake of sweetened beverages and CHD in middle-aged women with detailed measures of lifestyle and dietary factors. | The Nurses' Health Study were females aged 30-55 y/o living in the US and responding to a questionnaire regarding lifestyle, medical, and other health related information. | The intervention is does the fewer SSBs consumed decrease chances of CHD. The independent factor is whether or not the nurse will develop CHD. The dependent factors are how many SSBs are consumed and lifestyle of the particular person. | The results were a significant positive association between regular consumption of SSBs and the risk of CHD. This association remained significant even after adjustment for a multitude of dietary and lifestyle factors. Results were that those who consume >2SSB/day had an increased BMI of 25.3±.01, HTN 15±0.06, and increased consumption of fructose 20±0.03, and sucrose 35±0.04 greater than those who consumed <1SSB/month for a p trend of 0.0001. | Level 2 |
| Joyce, T., & Gibney, M. (2008). The impact of added sugar consumption on overall dietary quality in Irish children and | The study aimed to assess the impact of added sugars on overall diet quality by examining macronutrient, | The population sample were Irish children and teen ranging from 5-17 y/o. | The intervention is the overall impact of added sugar consumption by Irish children and teenagers. | The study found that with the more sugar was consumed by the lower socio-economic classes and in those who | Level 2 |

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| <p>teenagers. <i>Journal Of Human Nutrition & Dietetics</i>, 21(5), 438-450.</p> | <p>micronutrient, fiber, and food group intakes and compliance with macronutrient recommendations in Irish children and teenagers.</p> | | <p>The independent variable is the amount of sugar consumed by the individual.</p> <p>The dependent variables were related to age, location, and socio-economic status.</p> | <p>consumed the most sugar had lower intakes of fiber, fat, and especially protein. This thus reduced the intake of micronutrients in children and especially needed vitamins in the diet being replaced by sugar consumption. Increased SSB intake also followed a trend of increased consumption of cakes, chocolate, fruit juice, and other added sugars for a decrease in nutrients such as fiber for a p score of <0.001, but there was a less significant correlation to those who consumed added sugars with no SSB so they had a decrease in added sugar consumption for a p score of <0.05.</p> | |
| <p>Marriott, B., Olsho, L., Hadden, L., & Connor, P. (2010). Intake of added sugars and selected nutrients in the United States, National Health and Nutrition Examination Survey (NHANES) 2003-2006. <i>Critical Reviews In Food Science & Nutrition</i>, 50(3), 228-258. doi:10.1080/10408391003626223</p> | <p>The purpose of the study is to update expand the estimates of selected nutrient intake as related to dietary added sugars.</p> | <p>Children and adults in the US aged 4y/o and up and were involved in the NHANES what we eat in America.</p> | <p>The intervention is the survey that is performed to evaluate the daily intake of the average US family.</p> <p>There are no independent variables although it could be seen that BMI is in direct relation of intake of added sugar and calories from added sugar.</p> | <p>The results are that more than 50% of adults and 94% of total individuals did not meet their intake of micronutrient needs of vitamins and mineral but exceeded the amount of intake for sodium. 87% of the population had added sugar intake between 0-25% of energy and these had higher reports of micronutrient intake and fiber intake in relation to individuals who yielded >25% of their total energy intake from added sugars.. Individuals with intakes of energy >25% from added sugars appear to be at greater risk for nutrient inadequacy based on NHANES. On average the US ingested 83 grams of added</p> | <p>Level 1</p> |

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| | | | | sugar/day with the higher total energy intake coming from SSB. Those with >25% total energy intake in added sugar had 150.8 grams of their sugar from SSB. | |
| <p>Stanhope, K., Griffen, S., Bair, B., Swarbrick, M., Keim, N., & Havel, P. (2008). Twenty-four-hour endocrine and metabolic profiles following consumption of high-fructose corn syrup-, sucrose-, fructose-, and glucose-sweetened beverages with meals. <i>American Journal Of Clinical Nutrition</i>, 87(5), 1194-1203</p> | <p>The objective of this study is to compare the metabolic and endocrine effects of consuming HFCS and sucrose sweetened beverages and to determine whether responses are affected by sex and adiposity. An additional objective is to compare the effects of consuming HFCS and sucrose sweetened beverages with the consumption of beverages sweetened with fructose or glucose.</p> | <p>Participants were randomly selected through local advertisements and thirty four participants were selected with age ranges from 20-50 y/o</p> | <p>The intervention is consuming HFCS, sucrose, and glucose with a meal provided by the examiners over a 24 hr period to examine glucose effects on the metabolic system.</p> <p>The independent variable is the initial metabolic reading before meal consumption.</p> <p>The dependent variables are the SSBs consumed with each meal provided.</p> | <p>Comparing the HFCS, Sucrose and glucose results were: HFCS had a significant increase in triglyceride levels of 18.9±5.4 for a p<0.001. Sucrose increased triglycerides by 28.3±5.4 for a p score <0.001. Glucose did not have a significant noted increase but held a p score for 0.10 for triglycerides, but had a significant increase in insulin for a p<0.01 compared to the p<0.001 increase in fructose. All four sugars increased triglycerides, LDL, and insulin for a fasting blood sugars for sucrose a change of 222.2±27.2 and HFCS 226.3±25.2. Triglyceride changes for sucrose were 793.3±135.8 and HFCS 611.1±132.1. Total cholesterol changed 28.3±5.4 with sucrose and HFCS 18.9±4.5. Numbers were significant for both but higher for the added sucrose levels.</p> | <p>Level 2</p> |
| <p>Liu, S., Willett, W., Stampfer, M., Hu, F., Franz, M., Sampson, L., & ... Manson, J. (2000). A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. <i>American Journal Of Clinical Nutrition</i>, 71(6), 1455-1461.</p> | <p>The objective of this study was to prospectively evaluate the relations of the amount and type of carbohydrates with risk of CHD.</p> | <p>The population was the Nurses Health Study where 121700 female registered nurses aged 30-55 y/o were used and since has had a 2 yr follow up to learn of participants health condition</p> | <p>There is no intervention only observation and surveying for a relationship between consumption of SSBs and CHD.</p> <p>The independent variable is health status either good or bad in relation to CHD and diabetes.</p> <p>The dependent variables are diet and sugar consumption especially the consumption of</p> | <p>The comparison is between SSBs with HFCS, sucrose, and glucose and the effects on the metabolic system over a twenty four hour period and between sexes with two experimental trials more than a month apart. With the energy adjusted glycemic load results were 1.57(95% with CI 1.27,1.95; p<0.0001) which were</p> | <p>Level 3</p> |

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| | | | SSBs. | significant for this risk factor but no other factors. Shows that using glycemic index for foods could increase prediction of CHD with the intake of sugars. This was not specifically to processed sugars though. | |
| Wang, Y., Bleich, S., & Gortmaker, S. (2008). Increasing caloric contribution from sugar-sweetened beverages and 100% fruit juices among US children and adolescents, 1988-2004. <i>Pediatrics</i> , 121(6), e1604-14. | The aim is to provide a timely update on the current pattern of beverage consumption among US youth with specifics on SSBs and the excess calories consumed when drinking SSBs and FJs that contribute to excess weight gain among US children. Also an examination in age, gender, race, income, and overweight status. | Information was used from the National Health and Nutrition Examination Survey (NHANES III) 1988-2004. A cross sectional survey was used with children who completed the 24 hour dietary recall. | The independent variable is weight and health status of the child with the intervention being a survey of dietary status. The dependent variables are consumption of SSBs and how that impacts their weight and health status. | In 1999–2004, US youth consumed an average of 224 kcal per capita per day from SSBs (~11% of their daily energy intake). On any given day, 84% of the adolescents drank SSBs, and these youth consumed an average of 30 oz throughout the day, equivalent to 356 kcal or 16% of their total energy intake. To burn off ~360 extra kcal, an average 15-year-old boy who weighs 50 kg (110 lb) would need to replace sitting by 3.25 hours of walking or 1 hour of jogging. The youngest SSB drinkers (aged 2–5 years) consumed an average of 15.5 oz or 176 kcal from SSB on a typical day (1 regular 12-oz soda contains 140 kcal), ~10% of their total energy intake in 1999–2004. This is more than twice the dietary guideline, which suggests no more than 4 to 6 oz/day for those in this age group. Over the study period, we observed an overall increase in per-capita SSB and FJ consumption (Table 2). This increase parallels secular trends in total energy intake and prevalence of obesity during this decade. Given that the percentage in SSB drinkers changed little over this period, we believe that | Level 3 |

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| | | | | <p>population-wide increases in SSB consumption are likely driven by increases in daily consumption level.</p> <p>Our results indicate that carbonated sodas represented less than half of all SSBs consumed by the children aged 2 to 11. In contrast, carbonated sodas comprised approximately two thirds of the SSBs consumed by adolescents. These results suggest the importance of focusing on other SSBs in addition to soda, such as fruit drinks, fruit punch, and sports drinks. Our analysis also points to the rising importance of FJ in children's diet guidelines. Preschool-aged children who drink 100% juice consumed, on average, 10 oz daily, almost twice the APA-recommended amount of 4 to 6 oz/day. This finding echoes previous reports that documented that children aged 1 to 5 years who were enrolled in the Supplemental Nutrition Program for Women, Infants, and Children program received twice the recommended amount of fruit juice (~9.5 floz/day).^{19,20} Average consumption of 100% juice among teens also exceeded the recommended 8 to 12 oz/day for this age group. This proved that SSB contributed to average of 204kal/day from 1988-1994 to 224kal/day from 1999-</p> | |
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| | | | | 2004 with an increase of 9.8% in consumption of SSB. Of this increase the individual increase in consumption was 55% came from soda, 37% came from fruit juice, and 8% came from sports drinks. | |
| Wang, H., Steffen, L. M., Zhou, X., Harnack, L., & Luepker, R. V. (2013). Consistency Between Increasing Trends in Added-Sugar Intake and Body Mass Index Among Adults: The Minnesota Heart Survey, 1980-1982 to 2007-2009. <i>American Journal Of Public Health, 103</i> (3), 501-507. doi:10.2105/AJPH.2011.30056 | The purpose of the study is to show consistency between increasing added sugar intake and body mass index among adults and in relation CHD. | The study population included independent probability samples on non-institutionalized adults in a defined geographic area of Minneapolis St Paul and the surrounding area. | There is no present intervention but it is a survey of regular dietary intake and the trends in CVD and their risk factors in surveys conducted in the Minnesota Heart survey 190-1982, 2007-2009 | Age-adjusted added sugar intake (%kcal) increased concurrently with level of BMI in both men and women over 27 survey years ($p_{trend} < .001$). Women consumed less added sugar than did men in each survey ($p_{trend} < .001$) although the proportion of calories from added sugar were similar between genders. Men consumed approximately 10.9% of energy from added sugar in 1980-1982 and 15.1% in the last survey 2007-2009, representing a 38.5% increase throughout the survey. BMI in women paralleled their added-sugra intake decreasing in the last survey, whereas BMI in men continued to increase after 2000-2002 $p_{interaction} < .001$ between survey year and gender in relation to BMI. | Level 2 |
| Welsh, J., Sharma, A., Cunningham, S., & Vos, M. (2011). Consumption of Added Sugars and Indicators of Cardiovascular Disease Risk Among US Adolescents. <i>Circulation, 123</i> (3), 249-257. doi:10.1161/CIRCULATIONAHA.110.972166 | The purpose was to determine if there is an association between the consumption of added sugars and indicators of CVD risk among US adolescents and to determine if body weight modifies this association. | Data came from NHANES and the National Health consisting of adolescents aged 12-18 y/o living in the US between 1999-2004. | The intervention is whether decreasing added sugar consumption will decrease risk factors for heart disease and decrease BMIs of adolescents. The independent variable is the health status and BMI of adolescents involved in the study. The dependent variables are the increase or decrease of sugar consumption as to | Results demonstrate that intake of added sugars is positively associated with known CVD risk factors. Increases in dyslipidemia among adolescents regardless of body size and increased insulin resistance among those overweight or obese with higher intake of added sugars. Average daily intake for sugar consumption was 118.9 gram for a $p < 0.0001$. Intake of | Level 2 |

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| | | | whether it improves BMI and health status. | fats, fiber, protein, and sodium were negatively associated with increased intake of added sugar for a $p < 0.0001$. For those who consumed >30% of total energy from added sugar there was a negative association of HDL levels and a positive association in LDL levels for a p trend of < 0.001 . | |
| Go, A., Mozaffarian, D., Roger, V., Benjamin, E., Berry, J., Blaha, M., & ... Turan, T. (2014). Executive summary: heart disease and stroke statistics--2014 update: a report from the American Heart Association. <i>Circulation</i> , 129(3), 399-410. doi:10.1161/01.cir.0000442015.53336.12 | Is a purpose statement from the AHA and Circulation related to current statistics over CHD and stroke. | Current US statistics over CHD, stroke, and risk factors relating to each. | n/a | An estimated 31.9 million adults > 20 yrs of age have total serum cholesterol levels > 240 mg/dl with a prevalence of 13.8%. In 2010 1 in 9 deaths were attributed to heart failure. 1 in 3 deaths in 2010 were related to cardiovascular disease while 1 in 6 deaths were related to coronary heart disease. | Level 5 |
| Maki, G., Akemi, M., Atsushi, G., Satoshi, S., Naomi, A., Takuro, S., & ... Shaw, W. (2012). Dietary glycemic index and glycemic load in relation to HbA1c in Japanese obese adults: a cross-sectional analysis of the Saku Control Obesity Program. <i>Nutrition & Metabolism</i> , 9(1), 79-97. doi:10.1186/1743-7075-9-79 | The purpose of this study was to evaluate whether dietary glycemic index or load affected blood glucose especially HbA1c in obese adults in Japan. | In total, 237 women and men participated. We used the baseline data for the analysis. Of the 237 people participated in the study, 10 participants did not complete the study, and 227 participants were included in the analysis. | There was analysis of the glycemic load and index to determine the relation to blood glucose with no intervention. This was an analysis of the relation of the sugar intake and obesity on the glycemic scale and glycemic load amounts. | After adjustments for potential confounding factors, GI was not associated with HbA1c, but GL was positively associated with HbA1c. For increasing quartiles of GI, the adjusted mean HbA1c were 6.3%, 6.7%, 6.4%, and 6.4% (P for trend = 0.991). For increasing quartiles of GL, the adjusted mean HbA1c were 6.2%, 6.2%, 6.6%, and 6.5% (P for trend = 0.044). In addition, among participants with HbA1c $\geq 7.0\%$, 20 out of 28 (71%) had a high GL (\geq median); the adjusted odds ratio for HbA1c $\geq 7.0\%$ among participants with higher GL was 3.1 (95% confidence interval [CI] = 1.2 to 8.1) compared to the participants with a lower GL (<median). Further, among 16 | Level 3 |

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| | | | | <p>participants with FPG \geq 150 mg/dL, 13 participants (81.3%) had a higher GL; the adjusted odds ratio for FPG \geq 150 mg/dL among participants with a higher GL was 8.5 (95% confidence interval = 1.7 to 43.4) compared to those with a lower GL. In contrast, GI and GL were not associated with metabolic risk factors other than glycemia.</p> | |
| <p>Carey, M. G., Al-Zaiti, S. S., Liao, L. M., Martin, H. N., & Butler, R. A. (2011). A low-glycemic nutritional fitness program to reverse metabolic syndrome in professional firefighters. Results of a pilot study. <i>Journal Of Cardiovascular Nursing</i>, 26(4), 298-304.</p> | <p>The purpose of this study was to quantify MetS prevalence and evaluate the effect of a low-glycemic nutritional fitness program on the reduction of MetS risk factors among firefighters.</p> | <p>Professional firefighters were screened for MetS then enrolled in a low-glycemic nutritional fitness program for a 12-week period. Anthropometric and physiological measurements were obtained at the start and end of the program.</p> | <p>The intervention is the fitness program and the low glycemic nutritional diet.</p> <p>The independent factor is whether the metabolic syndrome improves with the dependent factors of exercise and change of diet to the low glycemic index diet.</p> | <p>Seventy-five firefighters (aged 42 [SD, 8] years, mostly white men) had a total MetS prevalence of 46.7% ($P < .05$ vs normal population). One platoon (10 men, aged 48 [SD, 5] years) was enrolled in the 12-week program. Most (7/10) had MetS at the baseline, but this prevalence decreased significantly after 12 weeks to 3 subjects ($P = .02$). On average, subjects had 3.2 (SD, 1.6) versus 1.9 (SD, 1.7) MetS risk factors ($P < .01$) at baseline and the 12-week interval, respectively.</p> <p>The prevalence of MetS and MetS risk factors is higher among professional firefighters compared with the general population. A short-duration, low-glycemic fitness program can successfully improve anthropometric and physiological measures and reduce the prevalence of MetS.</p> | <p>Level 1</p> |
| <p>Kent, L., Morton, D., Rankin, P.,</p> | <p>The purpose of this study was</p> | <p>Participants participating in</p> | <p>The intervention is was the</p> | <p>Results were</p> | <p>Level 1</p> |

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| <p>Ward, E., Grant, R., Gobble, J., & Diehl, H. (2013). The effect of a low-fat, plant-based lifestyle intervention (CHIP) on serum HDL levels and the implications for metabolic syndrome status--a cohort study. <i>Nutrition & Metabolism</i>, 10(1), 58-63. doi:10.1186/1743-7075-10-58</p> | <p>to further explore the changes in CVD risk factors, especially HDL, in a large cohort of individuals participating in a lifestyle intervention that advocated a low-fat, plant based eating pattern- the complete health improvement program</p> | <p>the CHIP lifestyle intervention within the U.S. for thirty days.</p> | <p>CHIP lifestyle and reducing added sugar, sodium, and cholesterol intake o 40g, 2000 mg, and 50 mg respectively. This was incorporated with thirty minutes of daily moderate physical activity. The intervention was performed for thirty days.</p> <p>The independent factor is how the five risk factors for Metabolic Syndrome are affected by the intervention.</p> | <p>HDL-reduction of 323 participants from 41.7% to 34.6%. HDL did decrease as much as 21% for a p<0.001 without explanation during the intervention but LDL decreased 15% p<0.0001 and TC < 12% P<0.0001. Mean changes were SBP -6.95, DPB -4.14, BMI -0.98, TC -21.46, HDL -4.77, LDL -17.10, TG -11.05, FPG -6.43, TC-HDL -0.13. LDL-HDL -0.14 all with a p value <0.001</p> | |
| <p>Van Horn, L., Johnson, R. K., Flickinger, B. D., Vafiadis, D. K., & Yin-Piazza, S. (2010). Translation and implementation of added sugars consumption recommendations: A conference report from the American Heart Association added sugars conference 2010. <i>Circulation</i>, 122(23), 2470-2490. doi:10.1161/CIR.0b013e3181ff dcb0</p> | <p>A conference report from the American Heart Association added Sugars Conference 2010 with consumption recommendation</p> | <p>Conference report on AHA recommendations on added sugar consumption and associated factors of heart disease</p> | <p>No factors or study. States recommendations by the AHA</p> | <p>Recommendations by the AHA for added sugars are on average for females no more than a 100 calories and for men no more than 150 calories from added sugars. This breaks down to six teaspoonful for women and 9 teaspoonful for men. Reference is a 12 ounce can of soda contains 140 calories or 9 tsps of added sugars. One 16 ounce bottle of sugar sweetened ice tea contains 184 calories (11.5 tsp) of added sugar and one candy bar contains 120 calories (7.5 tsp) from added sugar.</p> | <p>Level 1</p> |
| <p>Chiu, C., Liu, S., Willett, W., C., Wolever, T., M.S., Brand-Miller, J., Barclay, A., W., & Taylor, A. (2011). Informing food choices and health outcomes by use of the dietary glycemic index. <i>Nutrition Reviews</i>, 69(4), 231-242. doi:10.1111/j.1753-4887.2011.00382.x</p> | <p>The purpose is to provide information on the glycemic index and how food choices affect health</p> | <p>A guide to food choices and how they are glycemicly and how the glycemic index and glycemic load affect health in particular cardiovascular health.</p> | <p>How the glycemic index affects the glycemic load, but no experiment is performed it is simply a statement.</p> | <p>Using the glycemic index to eat results in a lower glycemic load when avoiding higher glycemic foods. This can help decrease inflammation and blood sugar and help improve HgbA1C in those who are metabolically compromised. High glycemic load diets are strongly associated with risk factors for CHD with low HDL levels and increased insulin resistance with increased metabolic syndrome and</p> | <p>Level 5</p> |

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| <p>Melanson, K., J., Summers, A., Nguyen, V., Brosnahan, J., Lowndes, J., Angelopoulos, T., J., & Rippe, J., M. (2012). Body composition, dietary composition, and components of metabolic syndrome in overweight and obese adults after a 12-week trial on dietary treatments focused on portion control, energy density, or glycemic index. <i>Nutrition Journal</i>, 11, 57-57. doi:10.1186/1475-2891-11-57</p> | <p>The aim of this trial was to compare the effects of these three different dietary approaches on body weight, components of the metabolic syndrome, and diet composition within the context of a comprehensive weight loss program. The focus was on chronic disease prevention in overweight and obese adults who are otherwise healthy, and did not meet criteria for MetS except waist circumference.</p> | <p>In Orlando, FL subjects were recruited through newspaper, advertisements, and were screened by telephone. Criteria were sedentary lifestyle, BMI 27-35, and were not on any prescribed medications for weight loss or recently lost weight.</p> | <p>The intervention was three separate diets one with portion control, one with decreased energy density, and one with glycemic control.</p> <p>The independent variables are the changes in LDL, HDL, HgbA1C, BMI, waist circumference, blood pressure, body fat percentage, and C reactive protein.</p> | <p>levels of C-reactive protein.</p> <p>Changes in the twelve week program was LED: BMI -4.14, BMI -1.36, Body fat % -3.87, fat mass -4.98, waist circumference -4.06, SBP -2.44, DBP -0.78, Triglycerides -0.15, HDL -0.06, Insulin -9.31, Glucose AUC-1.03, CRP -0.39, HOMA-IR-0.30.</p> <p>LGI: Body mass -3.39, BMI -1.11, Body percent body fat -2.65, Fat mass -3.64, waist circ -3.31, SBP -0.05, DBP 1.91, Triglycerides -0.06, HDL -0.11, Glucose 0.06, Insulin -5.35, Glucose AUC -0.54, HOMA-IR -0.14, CRP -0.54.</p> <p>PC: Body Mass -3.73, BMI -1.32, Body fat % -2.91, fat Mass -4.00, Waist Circ -2.87, SBP -3.71, DBP -2.29, Triglycerides -0.06, HDL -0.05, Glucose -0.07, Insulin -9.45, Glucose AUC-0.69, HOMA-IR -0.38, CRP -0.52.</p> <p>Similar findings found in each group with LED foods having the highest success, but only with minimal margins.</p> | <p>Level 2</p> |
| <p>Lee, A., K., Binongo, J. N. G., Chowdhury, R., Stein, A., D., Gazmararian, J., A., Vos, M., B., & Welsh, J., A. (2014). Consumption of less than 10% of total energy from added sugars is associated with increasing HDL in females during adolescence: A longitudinal analysis. <i>Journal of</i></p> | <p>The purpose of the study was to investigate the association between added sugar intake and HDL levels in females from early adolescence to early adulthood.</p> | <p>10 yr prospective cohort performed in three cities in the U.S.. Cincinnati, Ohio, Richmond, CA, and Washington, D.C.. Non-Hispanic Caucasian girls and non-Hispanic African American girls were used for the study with a total of n=1166 and n=1213</p> | <p>No intervention was proposed</p> <p>Plasma lipid levels were assessed at visits 1,3,5,7, and 10 for the present analysis.</p> | <p>At baseline the percent of normal-wt girls remained stable over time 68.8% at baseline to 67.5% at visits 10. The percent of obese girls increased from 12.6% at baseline to 17.0% at last visit. HDL remained steadily the same from 54.3 at the first visit and 54 at last. Average</p> | <p>Level 4</p> |

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| <p><i>the American Heart Association</i>, 3(1), e000615-e000615. doi:10.1161/JAHA.113.000615</p> | | <p>respectively.</p> | | <p>added sugar consumption increased from 17% at the first visit to 20.6% at the last visit. Among consumers of added sugar HDL was 2mg/dL higher in African Americans than Caucasians at all visits. Over the ten year study low consumption of added sugars was associated with a 2.2 mg/dL increase in HDL, from 55.1 to 57.3 mg/dL (p=0.04).</p> | |
| <p>Hirshberg, S., E., Fernandes, J., Melanson, K., J., Dwiggin, J., L., Dimond, E., S., & Lofgren, I., E. (2011). Dietary sugars predict chronic disease risk factors in college students. <i>Topics in Clinical Nutrition</i>, 26(4), 324-334. doi:10.1097/TIN.0b013e318237d026</p> | <p>The aim of this project was to examine the impact of dietary sugar components on risk factors for obesity, CVD, and T2D in young adult college students</p> | <p>College students at the University of Rhode Island were used with a sample size of 261 males and females.</p> | <p>A nutrition assessment and chronic disease risk factor identification study was a cross-sectional study of first year students performed at University of Rhode Island. Phone recall was used to evaluate dietary data with 2 consecutive weekdays of lab work occurring at the end of the semester.</p> | <p>A mean of total sugar consumption was 24% of daily allotment of kilocalories with 17% of those being added sugars. Mean total kilocalorie intake was 2100 with 53% coming from carbohydrates, 15% from protein, and 31% from fat. All sugar components were negatively correlated with HDL-C (P<0.05) and all but fructose were positively correlated with TC:HDL C ratio. Chronic fructose consumption is postulated to increase energy intake by activating neural reward pathways such as opioid and dopamine pathways. HDL decreased with total sugars and added sugars with equal amounts. The LDL increased across the board with a p<0.006 for total sugars.</p> | <p>Level 4</p> |
| <p>Duffey, K. J., Gordon-Larsen, P., Steffen, L. M., Jacobs, D., J., & Popkin, B. M. (2010). Drinking caloric beverages increases the risk of adverse cardiometabolic outcomes in the coronary artery risk development in young adults (CARDIA) study. <i>American Journal of Clinical Nutrition</i>, 92(4), 954-959.</p> | <p>To examine associations between intake of select beverages and continuous and categorical incident cardiometabolic factors and the metabolic syndrome in a sample of black and white young adults.</p> | <p>The CARDIA study is a prospective study of cardiovascular risk factors in 5115 persons using data from 1985-1986, 1992-1993, and 2005-2006.</p> | <p>A nutritional assessment which consisted of a short questionnaire on general dietary practices followed by a comprehensive food frequency questionnaire which asked about the previous month's food intake.</p> | <p>SSBs were the most significant in the assessment. WC increased significantly for $P_{trend} < 0.001$, high triglycerides increased for a $p_{trend} < 0.033$ and increases in LDL cholesterol for a $p_{trend} = 0.018$, hypertension increases for a $p_{trend} = 0.023$ with a low HDL $P < 0.05$. Fruit juice and whole</p> | <p>Level 4</p> |

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| doi:10.3945/ajcn.2010.29478 | | | | milk had lower risks of high triglycerides $p_{trend}=0.046$, hypertension $p=0.0007$. | |
| <p>Bantle, J. P., Raatz, S. K., Thomas, W., & Georgopoulos, A. (2000). Effects of dietary fructose on plasma lipids in healthy subjects. <i>American Journal of Clinical Nutrition</i>, 72(5), 1128-1134. Retrieved from https://ezproxy.southern.edu:444/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=ccm&AN=2001025192&site=ehost-live&scope=site</p> | <p>The objective of this study was to determine effect of dietary fructose on plasma lipids.</p> | <p>Twenty four healthy subjects participated where there were twelve males and females with six of each being < 40 y/o and six of each being > 40 y/o.</p> | <p>The intervention was two study diets in a randomized balanced crossover design, that is where each participant ate the prescribed diet for 42 days each. The diets were isoenergetic composed of common foods, and identical except crystalline fructose was added to one diet and crystalline glucose to the other.</p> <p>The independent variable was the plasma lipid levels and how they reacted to the diet.</p> | <p>The plasma lipids and triacylglycerol responses were different for men and women $p=0.04$. Serum apolipoprotein B concentrations were 0.80 ± 0.04 g/L for fructose on day 42 and for glucose were 0.77 ± 3mg/L on day 42 of the glucose diet. The response of plasma triacylglycerol during the 24-h metabolic profiles on day 42 of the study diets also differed by sex $p=0.008$. On day 42 of the fructose diet 32% in men had higher triacylglycerol than the glucose diet.</p> | <p>Level 2</p> |
| <p>Ibero-Baraibar, I., Cuervo, M., Navas-Carretero, S., Abete, I., Zulet, M., & Martinez, J. (2014). Different postprandial acute response in healthy subjects to three strawberry jams varying in carbohydrate and antioxidant content: A randomized, crossover trial. <i>European Journal of Nutrition</i>, 53(1), 201-210. doi:10.1007/s00394-013-0517-7</p> | <p>The aim of this study was to investigate the acute consumption effect of three different types of strawberry jams, high-sugar (HS), low sugar (LS) and low sugar antioxidant (LSA), with different carbohydrate and antioxidant content, on postprandial glucose metabolism, lipid profile, antioxidant profile, and satiety in healthy adult men and women.</p> | <p>Six men and ten women (BMI 23.99 ± 3.05 kg/m², age 25.94 ± 3.02 y/o) were enrolled in the study. A double blind randomized crossover, double blind study with three arms.</p> | <p>The intervention was the three different types of jams and how they affect blood sugar response with (HS, LS, and LSA).</p> <p>The independent variable is how the body is affected by the jams by evaluating postprandial glucose metabolism, lipid profile, antioxidant profile, and satiety in healthy adult men and women.</p> | <p>Jams used were HS contained natural and added sugars at 41.8 ± 1.6g/100g. LS contained only natural sugars at 2.6 ± 0.1g/100g, and LSA at 2.7 ± 0.1 g/100g but the LS varieties were w/o added sugar. LSA were free of polyphenols with the antioxidant being naturally occurring in the pulp of the fruit. Post prandial glycemic response with LSA and LS being significantly lower $p<0.001$ compared to the HS jam. HOMA-IR values decrease at 30 min after HS intake showing significant ($P<0.001$) higher levels at 30 and 60 min compared to LS and LSA jams. TC and HDL-C and TG decreased $p<0.05$ between 0-30min with no differences between jams.</p> | <p>Level 2</p> |

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| <p>Finley, C. E., Barlow, C. E., Halton, T. L., & Haskell, W. L. (2010). Glycemic index, glycemic load, and prevalence of the metabolic syndrome in the cooper center longitudinal study. <i>Journal of the American Dietetic Association, 110</i>(12), 1820-1829. doi:10.1016/j.jada.2010.09.016</p> | <p>To address the hypothesis that higher levels of glycemic index and glycemic load are directly associated with prevalence of the metabolic syndrome and its components, this study examines the cross-sectional association between glycemic index, glycemic load, and the metabolic syndrome and its components with adjustment for cardiorespiratory fitness, an objective and reliable measure of recent physical activity habits, and other potential confounding factors in women and men enrolled in the Cooper Center Longitudinal Study.</p> | <p>The Cooper Center Longitudinal Study is a prospective cohort study of men and women who received a comprehensive medical examination at the Cooper Clinic, a preventive health clinic in Dallas, TX. Non-hispanic whites for 9,137 ,men and 1,775 women.</p> | <p>There was no intervention it was a study to evaluate how glycemic index and glycemic load affect metabolic syndrome. Factors that were factored in were physical fitness and factors affecting men and women.</p> | <p>Energy glycemic index was higher in men with metabolic syndrome compared to men without metabolic syndrome (p<0.0001). There was no observed difference in women between glycemic index and glycemic load. Men in upper quintiles of glycemic index had increased odds of metabolic syndrome, large waist girth, elevated triglycerides, and low HDL-C with a significant trend seen across quintiles (p<0.05).A significant interaction was observed between glycemic index and fitness with the blood pressure component as the outcome variable (P=0.03). Association between glycemic index and blood pressure with cardiorespiratory fitness in men in the high fitness category showed an inverse association between glycemic index and high blood pressure p=0.0009 for trend. Glycemic load was positively associated with elevated triglycerides (P for trend <0.0001) in the multivariate model. HDL-C and glucose had inverse relationships for a p for trend <0.0001 for all.</p> | <p>Level 4</p> |
| <p>Johnson, R. (2009). Dietary sugars intake and cardiovascular health: A scientific statement from the american heart association. <i>Circulation (New York, N.Y.), 120</i>(11), 1011; 1011-1020; 1020.</p> | <p>Scientific statement from the American Heart Association</p> | <p>None</p> | <p>None</p> | <p>None</p> | <p>Level 5</p> |
| <p>Welsh, J., Sharma, A.,</p> | <p>To assess the association</p> | <p>Cross sectional study among</p> | <p>None</p> | <p>A mean of 15.8% of</p> | <p>Level 2</p> |

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| <p>Abramson, J., Vaccarino, V., Gillespie, C., & Vos, M. (2010). Caloric sweetener consumption and dyslipidemia among US adults. <i>JAMA: Journal Of The American Medical Association</i>, 303(15), 1490-1497. doi:10.1001/jama.2010.449</p> | <p>between consumption of added sugars and blood lipid levels in the U.S.</p> | <p>US adults from NHANES 1999-2006. Respondents were grouped into amounts of intake of added sugar and dietary recommendations.</p> | | <p>consumed calories was from added sugars. Among participants consuming less than 5% to less than 10%, 10% to less than 17.5%, 17.5% to 25%, and 25% or greater of total energy as added sugars, adjusted mean HDL-C levels were, respectively , 58.7, 57.5, 53.7, 51.0, and 47.7 mg/dL (p<0.001), geometric mean triglyceride levels were 105,102, 111, 113, and 114 mg/dL (p<0.001). and LDL-C levels modified by sex were 116, 115, 118, 121, and 123 mg/dL among women (p=0.047). No significant trends were seen for men. Among higher consumers (>10% added sugars) the odds of low HDL-C levels were 50% to more than 300% greater compared with the reference group (<5% added sugars).</p> | |
| <p>Ebbeling, C., Feldman, H., Chomitz, V., Antonelli, T., Gortmaker, S., Osganian, S., & Ludwig, D. (2012). A randomized trial of sugar-sweetened beverages and adolescent body weight. <i>New England Journal Of Medicine</i>, 367(15), 1407-1416. doi:10.1056/NEJMoa1203388</p> | <p>Consumption of SSBs may causes excessive weight gain. To assess the effect on weight gain on an intervention that included the provision of noncaloric beverages at home for overweight and obese adolscents.</p> | <p>Random trial of those who were obese and drank SSB regularly. The experiment group was a decrease in SSB while the control group had no intervention.</p> | <p>The experimental group had decreases in added SSB intake while the control group was only observational.</p> | <p>Retention rates were 97% at 1 year and 93% at 2 years. Reported consumption of sugar sweetened beverages was similar at baseline in the experimental and control groups, declined to nearly 0 in the experimental group at 1 year and remained lower in the experimental group than in the control group at 2 years. At 1 year there were significant differences for changes in BMI (-0.57, p=0.045) and weight (-1.9 kg, p=0.04). We found evidence of effect modification according to ethnic group, among Hispanic participants there was a significant</p> | <p>Level 2</p> |

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| | | | | <p>between-group difference in the change in BMI at 1 year (-1.79, p=0.007) and 2 years (-2.35, p=0.01) but not among non-Hispanic participants (p>0.35 at years 1 and 2). The change in body fat as a percentage of total body weight did not differ significantly between groups at 2 years (-0.5%, p=0.40). There were no adverse events related to study participation.</p> | |
| <p>Yang, Q., Zhang, Z., Gregg, E., Flanders, W., Merritt, R., & Hu, F. (2014). Added Sugar Intake and Cardiovascular Diseases Mortality Among US Adults. <i>JAMA Internal Medicine</i>, 174(4), 516-524. doi:10.1001/jamainternmed.2013.13563</p> | <p>To examine time trends of added sugar consumption as percentage of daily calories in the United States and investigate the association of this consumption with CBD and mortality.</p> | <p>Prospective cohort study that evaluated the NHANES surveys from 1988-1994, 1999-2004, and 2005-2010 for increases in added sugar consumption and increased incidence of mortality and CHD.</p> | <p>None</p> | <p>Among US adults the adjusted mean percentage of daily calories from added sugar increased from 15.7% (95% CI, 15.0%-16.4%) in 1988-1994 to 16.8% (16.0%-17.7%), p=0.02) in 1999-2004 and decreased to 14.9% (14.2%-15.5%; p<0.001) in 2005-2010. Most adults consumed 10% or more of calories from added sugar (71.4%) was approximately 10% consumed 25% or more in 2005-2010. During a median follow up period of 14.6 years, we adjusted hazard ratios of CVD mortality across quintiles of the percentage of daily calories consumed from added sugar were 1.00 (ref), 1.09 (95% CI, 1.05-1.113), 1.23 (1.12-1.34), 1.49 (1.24-1.78), and 2.43 (1.63-3.62, p<0.01), respectively. After additional adjustment for socio-demographic, behavioral, and clinical characteristics, HRs were 1.00 (ref) 1.07 (1.02-1.12), 1.18 (1.06-1.31), 1.38 (1.11-1.70), and 2.03 (1.26-3.27,</p> | <p>Level 2</p> |

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| | | | | <p>p=0.004), respectively. Adjusted HRs were 1.30 (95% CI, 1.09-1.55) and 2.75(1.40-5.42;p=0.004). Comparing participants who consumed 10% 224.9% or 25% or more of calories from added sugar with those who consumed less than 10% of calories from added sugar. These findings were largely consistent across age, group, sex, race/ethnicity, educational attainment, physical activity, health eating index, and BMI.</p> | |
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