

Sugar: the bitter truth

Introduction

Under the reign of Maharaja Sri Gupta, the Gupta Dynasty existed at its zenith from approximately 240 to 550 CE. Innovative and resourceful, the ancient Indian empire discovered methods of turning sugarcane juice into granulated crystals that were easier to store and transport. The passing centuries ensured that the cultivation of cane sugar became a global practice, with the West Indies and tropical parts of the Americas embracing the sweet, soluble carbohydrate in the 16th century, while the 19th and 20th centuries brought the development of beet sugar, high fructose corn syrup and other sweeteners. Fast forward to the 21st century, and the excessive consumption of sugar has been linked with everything from type 2 diabetes and heart disease to high blood pressure and high triglycerides. Numerous scientific papers argue, rather persuasively, that sugar consumption is detrimental, even poisonous, to one's health. However, such vague and unremarkable assertions can detract from the fact that many different types of sugar exist (table sugar, for example, has 44 "cousins."). The aim of this paper is to objectively show the ways in which fructose, a type of sugar, is the most damaging of all.

The dangers of fructose

In stark contrast to glucose, which tends to pass through the liver, fructose is metabolized almost completely in the glandular organ. Used mostly for glucoses, or the production of energy, glucose differs to fructose, which is mainly used to generate nucleic acid. From salad dressings to soft drinks, high fructose corn syrup (HFCS), a manmade sweetener, is found in a number of processed foods, and has been since the late 1970's. HFCS is inexpensive to produce, thus making it attractive to the food industry. Created by changing the glucose in corn starch to fructose, high-fructose corn syrup comes in three forms: HFCS-90, HFCS-55 and HFCS-42. Sugar is addictive, and some scientists believe that fructose rivals heroin for its addictive-like effects¹ although fruit does contain fructose, the amounts are relatively low, and the fiber content of apples, for example, slows down fructose absorption.

As sales of soft drinks containing the sweetener continue to reach new highs, the obesity epidemic continues to intensify, which suggests that a very intimate relationship exists between the two.² According to a number of studies, dietary fructose not only promotes cardiovascular disease but appears to have adverse effects on postprandial serum.³ In three separate studies, scientists split participants into two groups; those who would consume beverages made with high fructose corn syrup and those who would consume diet beverages made with aspartame. At the end of the study, which ran for 10 weeks, each participant was weighed. Those who had consumed the beverages containing high-fructose corn syrup gained weight, and those who didn't did not.⁴ The intake of dietary fructose has increased every year since the late 1970's, with modern day processed foods containing 25% more sugar than processed foods from the 70's.⁵ In 1970, according to United States Department of Agriculture (USDA) reports, HFCS consumption was virtually non-existent.⁶ In 2016, globally, HFCS now represents close to 44% of today's total added sweeteners, with sucrose, glucose syrup, pure glucose, and honey accounting for the other 56%.⁷

Volume 4 Issue 5 - 2016

John Glynn

Ludwig Von Mises Institute, USA

Correspondence: John Glynn, Ludwig Von Mises Institute, Auburn, Alabama 36832, United States, Tel +353874656871, Email john.glynn.gev@gmail.com

Received: July 29, 2016 | **Published:** August 05, 2016

In the U.S., for example, HFCS consumption has continuously increased over the past four decades, now accounting for more than 42% of total caloric sweetener consumption - The figure was 16% in 1978.⁸ What is even more worrying, perhaps, is the fact that 50% of children in the U.S. - between the ages of 3 and 5 - consume beverages containing manmade sweeteners.⁹ The rapid metabolism of fructose in liver cells can result in a rare condition called hereditary fructosuria. Or HFI¹⁰ An inborn error of fructose metabolism caused by a deficiency of the enzyme aldolase B, HFI renders individuals affected asymptomatic until the ingestion of fructose, sucrose, or sorbitol occurs.

Obesity

As excess fructose is quickly converted into fat, the scientific evidence linking the consumption of sugary drinks with weight gain is difficult to ignore.¹¹ Beverages high in sugar contribute to weight gain more than solid foods, and two soft drinks a day increases a child's risk of becoming obese by more than 60%.¹² Hormonally, fructose reduces leptin levels, meaning an individual feels less full, and, by increasing the expression of the hormone ghrelin, an individual feels hungrier.¹³ As the excessive consumption of sugary beverages can cause metabolic damage and trigger the early stages of diabetes and heart disease .replacing sugar with artificial sweeteners appears to be a healthier alternative.¹⁴

Diabetes is now the seventh-leading cause of death in the United States, with \$245billion spent each year on treatment.¹⁵ A 2010 meta-analysis study found that sugar-sweetened beverages, or SSBs, increase the risk of type 2 diabetes, and in some cases cause insulin resistance and β -cell dysfunction.¹⁶ Previous studies have shown that the ingestion of fructose increases subsequent food intake.¹⁷ Which suggests that, when it comes to suppressing the desire to consume food, fructose is less efficient than glucose? Another study saw participants subjected to a 10-wk supplementation with either glucose or fructose. Researchers found that fructose sparked an increase in body weight.¹⁸ However glucose did not cause an increase in body weight. Ever since the 60's and the seminal work of Sir Philip Randle,

the eminent British medical researcher, the link between disturbed lipid metabolism and insulin resistance has been scrutinized, with several studies suggesting that a high-fructose diet leads to hepatic insulin resistance in healthy males.¹⁹ Furthermore, insulin-resistant individuals have higher ectopic lipid deposition, which can severally damage insulin signal transduction pathways, thus affecting glucose storage and uptake.²⁰ Fructose and hyperinsulinemia, a condition in which there are excess levels of insulin circulating in the blood relative to the level of glucose, appear to possess a close relationship.²¹ Hyperinsulinemia, where the amount of insulin in the blood is higher than considered normal amongst non-diabetics, leads to the accumulation of intracellular glyceraldehydes and dihydroxyacetone phosphate.²² Research has also shown that high-fructose diets increase hypertension levels.²³ On the basis of specific studies evaluating the relationship between fructose consumption and obesity, the effect of fructose on total energy intake seems to be a pertinent issue. Several studies, including an independent meta-analysis, identified a direct link between fructose and increased energy intake.²⁴

Fructose intake and cardiovascular risk factors

Recent studies have highlighted the dangers of diets enriched with fructose. Essentially, along with weight gain, high-fructose diets can cause several adverse metabolic and cardiovascular diseases, including dyslipidemia, insulin resistance, hypertension, and hyperuricemia.²⁵ Dyslipidemia, where an abnormal amount of lipids (e.g. cholesterol and/or fat) are present in the blood, can result from a high-fructose diet, as the sugar can increase plasma total- and VLDL-triglycerides, both in healthy volunteers and in patients with insulin resistance.²⁶ Even in the case of healthy volunteers, an increase in total cholesterol is not just possible, it's probable.²⁷ Numerous studies have examined the hyperlipidemic effects of fructose. One meta-analysis, which examined eating habits from 1977 to 2004, concluded that fructose in large amounts becomes toxic.²⁸

More than 6,000 volunteers took part in a study examining the relationship between soft drink consumption and cardiovascular risk factors. The final results were conclusive: Consuming more than one soft drink (330ml can) per day significantly increases the risk of developing high blood pressure.²⁹ A subsequent study of 74 Swiss children, aged between 6 and 14years-old, showed that fructose promoted the early signs of atherosclerosis, a condition in which plaque builds up inside the arteries.³⁰ High doses of fructose can result in postprandial hypertriglyceridemia and intra-abdominal fat accumulation, along with elevated blood pressure.³¹ Along with obesity, diabetes and dental cavities, excess sugar consumption has been associated with cardiovascular disease.³² Individuals consuming 10% or more of their total energy from added sugar are three times more likely to develop heart disease than those who consume less than 10%.³³ A single 355 ml can of sugar-sweetened soda contain - on average - 40grams, or 10 teaspoons, of sugar.³⁴ Is it really a shock that obesity levels are on the rise In Canada, for example, 3.5billion litres of sugar-sweetened soda is available for consumption each year.³⁵ That works out at roughly 110litres per person. Recent research shows that the average American child, between the ages of 4 and 8, consumes 68grams of sugary drinks a day, with this number quintupling between the ages of 14 and 18.³⁶

Recommendations

As individuals we can reduce fructose consumption by preparing meals using fresh produce, and the consumption of ready-to-eat and

processed foods should be minimized. If and when buying fruit, make sure it is fresh or frozen, and if purchasing it in canned form, ensure it comes without artificial sweeteners. Governments across the globe should make a conscious effort to ensure that food industries decrease free sugar content in foods. By grouping all sugars together when listing ingredients on product packaging, the labelling of content in the Nutritional Contents table of all packaged foods become less cryptic. Furthermore, restaurant menus should list the free sugar, sodium, Trans fat, saturated fat, and calorie content of their produce. Finally, if we are to minimize the risk posed by sugar, fructose included restrictions on the marketing of all foods and beverages, especially to children, must be considered. Provincial governments may consider placing an excise tax on beverages high in free sugars, which are largely energy dense and nutrient deficient. The indirect tax could be based on free sugars per unit, with created revenues being used to educate and make healthy eating more economical.

Conclusion

To label fructose chronically toxic seems appropriate. When consumed intermittently, fructose appears to cause minimal damage; however, frequent, excessive consumption appears to cause serious damage. Fructose, the sweetest tasting of all sugars, is so addictive that Dr. Robert Lustig famously called it “alcohol without the buzz.” Moderately higher insulin and blood sugar levels help our bodies feel more satiated after eating, but fructose, due to the way it's metabolized, doesn't appear to help with satiety.

Acknowledgements

None.

Conflict of interest

Author declares that there is no conflict of interest.

References

1. Avena NM, Rada P, Hoebel BG. Evidence for sugar addiction: Behavioral and neurochemical effects of intermittent excessive sugar intake. *Neurosci Biobehav Rev.* 2008;32(1):20–39.
2. Bray GA, Nielsen SJ, Popkin BM. Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *Am J Clin Nutr.* 2004;79(4):537–543.
3. Nakagawa T, Hu H, Zharikov S, et al. A causal role for uric acid in fructose-induced metabolic syndrome. *Am J Physiol Renal Physiol.* 2006;290(3):F625–F631.
4. Le MT, Frye RF, Rivard CJ, et al. Effects of high-fructose corn syrup and sucrose on the pharmacokinetics of fructose and acute metabolic and hemodynamic responses in healthy subjects. *Metabolism.* 2001;61(5):641–651.
5. Havel PJ. Dietary fructose: implications for dysregulation of energy homeostasis and lipid/carbohydrate metabolism. *Nutr Rev.* 2005;63(5):133–157.
6. United States Department of Agriculture. Sugar and Sweeteners Yearbook Tables. *Other Recommended Data Products.* 2009.
7. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *Am J Public Health.* 2007;97(4):667–675.
8. Marriott BP, Cole N, Lee E. National estimates of dietary fructose intake increased from 1977 to 2004 in the United States. *J Nutr.* 2009;139(6):1228–1235.

9. Harnack L, Stang J, Story M. Soft drink consumption among US children and adolescents: nutritional consequences. *J Am Diet Assoc*. 1999;99(4):436–441.
10. Hommes FA. Inborn errors of fructose metabolism. *Am J Clin Nutr*. 1993;58(5):788S–795S.
11. Malik VS, Willett WC, Hu FB. Sugar-sweetened beverages and BMI in children and adolescents: reanalysis of a meta-analysis. *The American Journal of Clinical Nutrition*. 2009;89(1):438–439.
12. Ludwig DS, Peterson KE, Gortmake SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective. *Lancet*. 2001;357(9255):505–508.
13. Janne C de Ruyter, Margreet Olthof, Jacob C. A Trial of Sugar-free or Sugar-Sweetened Beverages and Body Weight in Children. *N Engl J Med*. 2012;367(15):1397–1406.
14. Ebbeling CB, Feldman HA, Chomitz VR, et al. A Randomized Trial of Sugar-Sweetened Beverages and Adolescent Body Weight. *N Engl J Med*. 2012;367(15):1407–1416.
15. <https://www.cdc.gov/diabetes/pubs/statsreport14/national-diabetes-report-web.pdf>
16. Sluijs I, van der Schouw YT, van der A DL, et al. Carbohydrate quantity and quality and risk of type 2 diabetes in the European Prospective Investigation into Cancer and Nutrition–Netherlands (EPIC-NL) study. *Am J Clin Nutr*. 2010;92(4):905–911.
17. Rodin J. Pure sugar vs. mixed Effects starch fructose loads on food intake. *Appetite*. 1991;17(3):213–219.
18. Stanhope KL, Schwarz JM, Keim NL, et al. Consuming fructose-sweetened, not glucose-sweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans. *J Clin Invest*. 2009;119(5):1322–1334.
19. Faeh D, Minehira K, Schwarz J, et al. Effect of fructose overfeeding and fish oil administration on hepatic de novo lipogenesis and insulin sensitivity in healthy males. *Diabetes*. 2005;54(7):1907–1913.
20. Shulman GI. Cellular mechanisms of insulin resistance. *J Clin Invest*. 2000;106(2):171–176.
21. Grimble RF. Fructose is associated with inflammation of adipose tissue, and this is particularly evident in obese patients. Inflammatory status and insulin resistance. *Clin Nutr Metab Care*. 2002;5(5):551–559.
22. Eifert KC, McDonald RB, Stern JS. High sucrose diet and exercise: effects on insulin-receptor function of 12- and 24-mo-old Sprague-Dawley rats. *J Nutr*. 1991;121(7):1081–1089.
23. Reaven GM, Lithell H, Landsberg L. Hypertension and associated metabolic abnormalities: the role of insulin resistance and the sympathetic-adrenal system. *New Engl J Med*. 1996;334(6):374–381.
24. Fields M, Lewis CG. Dietary fructose but not starch is responsible for hyperlipidemia associated with copper deficiency in rats: effect of high-fat diet. *J Am Coll Nutr*. 1999;18(1):83–87.
25. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *Am J Public Health*. 2007;97(4):667–675.
26. Bizeau ME, Pagliassotti MJ. Hepatic adaptations to sucrose and fructose. *Metabolism Exp*. 2005;54(9):1189–1201.
27. Bantle JP, Laine DC, Thomas JW. Metabolic effects of dietary fructose and sucrose in types I and II diabetic subjects. *JAMA*. 1986;256(23):3241–3246.
28. Crapo PA, Kolterman OG. The metabolic effects of 2-week fructose feeding in normal subjects. *Am J Clin Nutr*. 1984;39(4):525–534.
29. Marriott BP, Cole N, Lee E. National estimates of dietary fructose intake increased from 1977 to 2004 in the United States. *J Nutr*. 2009;139(6):1228–1235.
30. Dhingra R, Sullivan L, Jacques PF, et al. Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. *Circulation*. 2007;116(5):480–488.
31. Aeberli I, Zimmermann MB, Molinari L, et al. Fructose intake is a predictor of LDL particle size in overweight schoolchildren. *Am J Clin Nutr*. 2007;86(4):1174–1178.
32. Stanhope KL, Havel PJ. Fructose consumption: Potential mechanisms for its effects to increase visceral adiposity and induce dyslipidemia and insulin resistance. *Curr Opin Lipidol*. 2008;19(1):16–24.
33. Yang Q, Zhang Z, Gregg WE, et al. Added sugar intake and cardiovascular diseases mortality among US adults. *JAMA Internal Med*. 2014;174(4):516–524.
34. Liu S, Willett WC, Stampfer MJ. A prospective study of dietary glycemic load, carbohydrate intake and risk of coronary heart disease in US women. *Am J Clin Nutr*. 2000;71(6):1455–1461.
35. WHO opens draft consultation on draft sugars guideline: note for media. World Health Organization; 2014.
36. Buhler S, Raine KD, Arango M, et al. Building a strategy for obesity prevention one piece at a time: The case of sugar-sweetened beverage taxation. *Can J Diabetes*. 2013;37(2):97–102.
37. Garriguet D. Beverage consumption of children and teens. *Health Rep*. 2008;19(4):1–6.