

# Sugar-a real enemy

## Abstract

Sugar and sweet consumption have been very popular worldwide. Along with modern sedentary lifestyle, this increasing trend of per capita sugar consumption assumes significance in the view of high tendency to develop insulin resistance, abdominal adiposity, and hepatic steatosis, and the increasing “epidemic” of type 2 diabetes (T2DM) and cardiovascular diseases. This article is to show the effects of different type of sugar and its effect on our physiology. We must work on various prevention strategies, encompassing multiple stakeholders (government, industry, and consumers), should target on decreasing sugar consumption. In this context, dietary guidelines show that sugar consumption should be less than 10% of total daily energy intake, but it is suggested that this limit be decreased.

**Keywords:** carbohydrate, empty calorie, refined sugar, obesity

Volume 4 Issue 4 - 2016

Pooja Shelat,<sup>1</sup> Mahendra narwaria,<sup>2</sup> Sanjay Patolia,<sup>2</sup> Arya Singh,<sup>1</sup> Binjal Shah<sup>1</sup>

<sup>1</sup>Department of Nutrition, Asian Bariatrics, India

<sup>2</sup>Department of Surgeon, Asian Bariatrics, India

**Correspondence:** Pooja Shelat, Bariatric Nutritionist, Asian Bariatrics Pvt Ltd., Ahmedabad, Gujarat, India, Email pdshelat91@gmail.com

**Received:** April 12, 2016 | **Published:** May 10, 2016

## Introduction

Overweight and obesity are major risk factors for a number of chronic diseases, including diabetes, cardiovascular diseases and certain types of cancer.<sup>1-3</sup> While the etiology of obesity is complex, increased consumption of free sugars, particularly in the form of sugar-sweetened beverages, is associated with weight gain in both children and adults.<sup>4-6</sup> Sugar-sweetened beverages contain added sugars such as sucrose or high fructose corn syrup. High fructose corn syrup (HFCS) and sucrose are the same thing because they're both highly sweet and they both contain a large amount of fructose and glucose. Sucrose is 50% fructose & 50% glucose and HFCS is 55% fructose & 45 % glucose. Our body process fructose and glucose in a different way.

## Discussion

### Glucose

It is a primary source of energy. When it's transported into the body, it stimulates pancreas to produce insulin. Our brain notices this increase, understands metabolism & gradually reduces hunger. This is mainly by the “Feed Back Mechanism”. There are many processes involved when you consume glucose, but one that occurs in your liver produces very low density lipoprotein (the bad cholesterol)(VLDL) Fortunately, only about 1 out of 24 calories from glucose that are processed by the liver turn into VLDL.<sup>7</sup>

### Fructose

Fructose is considered bad because of how it's processed by the body. Firstly, there is no hormone to remove fructose from our bloodstream (unlike glucose, which stimulates insulin production). Fructose can only be metabolized by liver. This means a greater number of calories which is about three times more than glucose which is metabolized in liver, results in much higher production of VLDL and fat.<sup>7</sup>

Fructose fools metabolism by turning off body's appetite-control system. It fails to stimulate insulin, which in turn fails to suppress

ghrelin, or “the hunger hormone,” which then fails to stimulate leptin or “the satiety hormone.” This causes you to eat more and develop insulin resistance.

High doses of fructose (>50g/day at least) in humans have been implicated in insulin resistance, postprandial hypertriglyceridemia, intra-abdominal fat accumulation, and elevated blood pressure,<sup>8-11</sup> mediated by high levels of non-esterified fatty acid (NEFAs).<sup>12</sup> Bursac et al.,<sup>13</sup> have analyzed the effects of 9-week consumption of 60% fructose solution on dyslipidemia, insulin and leptin sensitivity, and adipose tissue histology in male Wistar rats. They proposed that high-fructose-diet-induced alterations of glucocorticoid signaling in both hypothalamus and adipose tissue result in enhanced adipogenesis.<sup>13</sup> Another study by Shapiro et.al indicated that chronic fructose consumption induces leptin resistance prior to body weight, adiposity, serum leptin, insulin, or glucose increases, and this fructose-induced leptin resistance accelerates high-fat induced obesity.<sup>14</sup>

### Fruits are good though it contains fructose. Why???

This is because fruit, in its natural form, contains fiber. Fructose doesn't provide a satiety alert to let your brain know to tell you to stop eating, but fiber does this to a high degree. This is why you can eat fruit-despite the fructose content.

In a study Lydia et al.,<sup>15</sup> showed that an increase of three servings/day in total fruit and vegetable consumption was not associated with development of diabetes, whereas the same increase in whole fruit consumption was associated with a lower hazard of diabetes. An increase of 1 serving/day in green leafy vegetable consumption was associated with a modestly lower hazard of diabetes, whereas the same change in fruit juice intake was associated with an increased hazard of diabetes.<sup>15</sup>

## Conclusion

Government and health care industry should plan strategies to reduce the production as well as to plan awareness programs for consumers to reduce consumption of sugar sweetened beverages and encourage to have sugar in its natural form.

## Conflict of interest

The author declares no conflict of interest.

## References

1. Key TJ, Schatzkin A, Willett WC, et al. Diet, nutrition and the prevention of cancer. *Public Health Nutr.* 2004;7(1A):187–200.
2. Srinath Reddy K, Katan MB. Diet, nutrition and the prevention of hypertension and cardiovascular diseases. *Public Health Nutr.* 2004;7(1A):167–186.
3. Steyn NP, Mann J, Bennett PH, et al. Diet, nutrition and the prevention of type 2 diabetes. *Public Health Nutr.* 2004;7(1A):147–165.
4. Malik VS, Pan A, Willett WC, et al. Sugar-sweetened beverages and weight gain in children and adults: a systematic review and meta-analysis. *Am J Clin Nutr.* 2013;98(4):1084–1102.
5. Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. *BMJ.* 2013;346:e7492.
6. Mattes RD, Shikany JM, Kaiser KA, et al. Nutritively sweetened beverage consumption and body weight: a systematic review and meta-analysis of randomized experiments. *Obes Rev.* 2011;12(5):346–365.
7. <http://lifehacker.com/5809331/what-sugar-actually-does-to-your-brain-and-body>
8. 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 Investig.* 2009;119(5):1322–1334.
9. 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.
10. Hallfrisch J, Ellwood KC, Michaelis OE, et al. Effects of dietary fructose on plasma glucose and hormone responses in normal and hyperinsulinemic men. *J Nutr.* 1983;113(9):1819–1826.
11. Brown CM, Dulloo AG, Yepuri G, et al. Fructose ingestion acutely elevates blood pressure in healthy young humans. *Am J Physiol Regul Integr Comp Physiol.* 2008;294(3):730–737.
12. McGarry JD. Disordered metabolism in diabetes: Have we underemphasized the fat component? *J Cell Biochem.* 1994;55:29–38.
13. Bursac BN, Vasiljević AD, Nestorović NM, et al. High-fructose diet leads to visceral adiposity and hypothalamic leptin resistance in male rats-do glucocorticoids play a role? *J Nutr Biochem.* 2014;25(4):446–455.
14. Shapiro A, Mu W, Roncal C, et al. Fructose-induced leptin resistance exacerbates weight gain in response to subsequent high-fat feeding. *Am J Physiol Regul Integr Comp Physiol.* 2008;295(5):R1370–R1375.
15. Bazzano LA, Li TY, Joshipura KJ, et al. Intake of Fruit, Vegetables, and Fruit Juices and Risk of Diabetes in Women. *Diabetes Care.* 2008;31(7):1311–1317.