

REVIEW ARTICLE

Sugar and Chronic Diseases

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ABSTRACT:

Recent research at the University of California San Francisco shows that added sugars are responsible for chronic diseases such as diabetes mellitus. Growing scientific evidence shows that too much added sugar, over time, is linked to diabetes, heart disease and liver disease. Overconsumption of added sugar is linked to type 2 diabetes. Our current food environment in which the vast majority of packaged foods have added sugar makes it too easy to have too much.

KEYWORDS: Added sugar, sweeteners, fructose corn syrup, diabetes mellitus.

1. INTRODUCTION:

Sugars are the sweetening agents which we use in food. They are nothing but basically carbohydrates. Sugars are the metabolites available in extremely plant species, but are most abundant in the Sugarcane. They usually consist of sucrose, galactose, fructose corn syrup etc. Historically Sugar replaced the use of honey. Initial isolation and extraction of sugars took place in India.¹ Brazil is the largest producer of Sugar while India is the second largest. In terms of usage, India is the highest consumer of sugar, miles ahead of European Union and Brazil.^{2,3}

Refined sugar is the primary product of sugarcane extraction. The consumed sugar usually is made up of glucose and fructose monosaccharides which are generally reducing in nature. Granulated sugar stands the most shared group of refined sugars. It is consumed by many people on a daily basis and is found in most households. Granulated sugar is quite commonly used to sweeten consumables, such as coffee and tea. This type of refined sugar is likewise used in baking and cooking.⁴ Sanding sugar is another form of refined sugar which is little harder than granulated sugar, which makes it suitable for cookies or cake decoration.

It likewise has the ability to add texture to a dessert, because it dissolves extremely slowly and likewise maintains a gritty feel in many serving dishes. Sanding sugar is correspondingly used with definite cold desserts to sweeten without sacrificing texture.

Powdered sugar remains to be the last type of refined sugar. It has an extremely much smoother texture than the other types of refined sugars and is commonly used in pastries and likewise dessert toppings. It can be mixed easily and a smooth product. It's likewise the type you might see coating a donut, because it's fine texture helps it stick to food surfaces.⁵

The American Heart Association in 2015 has limited the recommended daily Intake of refined sugar to 25 grams per day which is just one tenth of required daily calories.⁶

2. HEALTH EFFECTS OF SUGAR:

2.1 Fructose and its side effects:

Sucrose (polysaccharide) and its synthetic derivative fructose corn syrup consist of 2 monosaccharides, glucose and Fructose. Glucose is the monosaccharide which if polymerized produces starch, which has a huge glycemic index, stimulates the insulin response from the pancreas, and is not particularly sweet.⁷ Fructose is found in fruit, will not stimulate any insulin response, and is extremely sweet. Fructose usage has increased across the world, leading to the obesity and chronic metabolic

disorders. Sugar has been a cause of concern for doctors for ages. Never the less, Fructose is not the same as glucose. When the calorie levels are increased glycogen is in a lysis state, transitional metabolites from its break down surpasses liver mitochondrial ability, which leads to newer lipogenesis and leads to liver insulin confrontation, which drives chronic metabolic disorder. Fructose likewise leads to reactive oxygen species synthesis, which leads to cellular level damage and aging, and leads to alteration in the brain's incentive system, which drives excessive usage.⁸

2.2 Dental Caries

Dental caries is a food linked disorder which lasts to be a delinquent for certain dental patients. Frequent usage of fermentable carbohydrates which have low oral elimination rates increases the chance for enamel caries and conceivably is even more hazardous for root surfaces.⁹ Certain additives as well as sugar surrogates show great potential for the provision of between meal snack foods which reduce the chance of dental caries. Human foods, never the less, vary in food items eaten and the frequency and pattern of eating, and these factors can determine the caries forming ability of a food.

2.3 Obesity

The obesity epidemic is clinically linked with increases in total energy intake; never the less, macronutrient analysis reveals which total fat ingestion has been constant; but total carbohydrate intake, especially sugar (sucrose, high-Fructose corn syrup), makes up the majority of this increase.¹⁰ Americans consume 5 times as much sugar as they did 100 years ago (from 30 to 150 gm/day). Sugar is composed of the two monosaccharides glucose (found in starch) and fructose (found only in sugar). The Fructose moiety gives sugar its sweetness; but Fructose is likewise a dose-dependent hepatotoxin, similar to ethanol. In a situation of caloric excess, Fructose is broken down by the liver by newer lipogenesis and in the process, leads to the same dose-dependent toxic effects as does ethanol, leading to hypertension, liver and skeletal muscle insulin resistance, hyperlipidemia, and fatty liver disorder.

Fructose induces alterations in central nervous system energy signalling which lead to a vicious cycle of excessive usage.¹¹ These effects may be a direct effect of the monosaccharide, or indirect, through increased triglycerides in blood and reduced insulin uptake. Finally, initial information postulates which fructose may act directly on the brain to alter mitochondrial energetics, promoting neuronal damage, which may manifest as altered cognition and decision-making ability, even dementia.¹² These effects may likewise be direct or indirect through insulin resistance. Finally, maternal fructose consumption may have distinct effect

on the foetus before birth, through epigenetic alterations in this energy balance pathway.

2.4 Diabetes

While experimental and observational research postulates which sugar usage is linked with the development of type 2 diabetes, it is not sure whether alterations in sugar usage can account for differences in diabetes prevalence among overall populations. No other food has proved significant individual linking with diabetes incidence after controlling for obesity. The impact of sugar on diabetes was independent of sedentary lifestyle and alcohol consumption, and the effect was changed but not unapproved by other parameters such as obesity and overweight. Duration and degree of sugar exposure correlated significantly with diabetes incidence in a dose-dependent manner, while declines in sugar exposure correlated with significant subsequent declines in diabetes rates independently of other socioeconomic, food and obesity incidence alteration.

Differences in sugar availability statistically explain variations in diabetes incidence rates at a population level which are not explained by physical activity, overweight or obesity.¹³

In retrospect, the argument which Fructose corn syrup is uniquely linked to obesity failed to take into consideration a number of important issues. Although usage of Fructose corn syrup in the World drastically increased from the initial 1970s if it first came into consumption until about 1999, over the past decade the usage of Fructose corn syrup has decreased, whereas obesity has increased or remained at the same levels.¹⁴ Moreover, as fructose corn syrup usage increased in the World, drastic decrease in the amount of sucrose that is consumed is recorded.

Although total caloric sweetener usage in the World has increased since 1970, sucrose remains the leading added sugar consumed in the American food and the leading source of Fructose. According to the many studies, sugars and sweeteners available for usage increased approximately 80 kcal/d per person from 420 kcal to 480 kcal.¹⁵ Furthermore, across the world usage of sucrose is nine times as much as Fructose corn syrup, and there are epidemics of obesity and diabetes in areas where little or no Fructose corn syrup is available.

Certain researchers say that fructose corn syrup is metabolically different from sucrose. Research by Stanhope et al. showed reports similar to those which has been reported in both men and women and likewise demonstrated no difference in post-prandial triglycerides after usage of either Fructose corn syrup or

sucrose.¹⁶ Research showed no differences in satiety or energy usage after fructose corn syrup, sucrose, or milk preloads.

Perhaps the reigning scientific reports relating to the metabolic diseases equate the fructose corn syrup and sucrose the hypothesis which the replacement of sucrose by fructose corn syrup in beverages is an affirmative factor in obesity is not supported on the basis of its conformation, biologic activities, or short-term effects on food usage.¹⁷ Had the hypothesis been phrased in the converse, namely which replacing fructose corn syrup with sucrose in beverages would be seen as a solution to the obesity epidemic, its merit would have been seen more easily. An offer which a return to sucrose containing beverages would be an incredible answer to the obesity epidemic would have been met with outright dismissal.¹⁸

Although this debate has largely been resolved within the scientific community, multiple articles in the lay press and Internet postings still maintain which fructose corn syrup is somehow uniquely linked to obesity. Furthermore, a number of food and beverage manufacturers had touted removal of fructose corn syrup from their products as though it somehow makes these products healthier.¹⁹ These examples serve as a reminder which scientific debate on issues which the public cares about does not take place in a vacuum and which misperceptions may linger long after the scientific debate has largely been resolved.

Several research had compared the breakdown, endocrine response, and health effects of pure Fructose with those of pure glucose. Often this research had compared amounts of either Fructose or glucose delivered as 25% of energy as components of mixed nutrient foods.²⁰ The theoretical justification for such research trials rests on the well-established difference in liver breakdown of fructose and glucose in the liver. The routes of liver breakdown of Fructose and glucose are depicted.²¹

Fructose breakdown differs from which of glucose in 2 important ways. First, there is initial complete liver extraction of Fructose.

Stanhope et al. reported which consuming Fructose-sweetened, but not glucose-sweetened beverages at the same levels of energy usage (25% of energy), increased visceral adiposity and lipids, and decreased insulin sensitivity in overweight or obese individuals.¹⁶ Recent research reviews by Dolan et al. reported which no adverse effect on triglycerides or weight was observed in multiple trials using Fructose at up to the 95th percentile population usage level.²² Likewise documented which no

increases in blood pressure or propensity toward obesity occurred at up to the 90th percentile population usage levels of Fructose. It should be emphasized which these meta-analyses included only research exploring usage of Fructose in isolation. Stanhope et al. Likewise did not find increases in blood pressure if obese individuals consumed as much as 25% of energy as either Fructose or glucose.¹⁶

It should be noted which Fructose and glucose are rarely consumed in isolation in the human food. Thus, research comparing pure Fructose with pure glucose, particularly at high levels, should be treated with caution, particularly consumption research comparing the more commonly consumed sucrose and fructose corn syrup had yielded different results.

With the recognition which Fructose versus glucose experiments does not reflect typical human nutrition, increased scrutiny has been consumption on the Fructose moiety of both fructose corn syrup and sucrose.

Sucrose is composed of 50% glucose and 50% Fructose, whereas the producers of fructose corn syrup consumption in most foods and beverages are typically composed of 55% Fructose and 45% glucose (this is the common form of consumption in beverages) or 42% Fructose and 58% glucose the form commonly consumption in baked goods and other food applications.²³

A number of research had explored sugary drink usage and its potential association with a variety of metabolic and health issues. Cross-sectional research in humans had linked soft-drink usage with less optimum nutrition, greater body weight, and higher energy usage.²⁴ It has likewise been postulated which excessive Fructose usage from added sugars may play a factor in epidemics of heart disorder, insulin resistance, type 2 diabetes, hypertension, hyperlipidemia, and obesity.²⁵ In addition, some research had postulated which Fructose ingestion may lead to increased indices of inflammation and free radical release, whereas other research had not confirmed these findings.²⁶

With this body of synthesis as background, the American Heart Association has issued a scientific statement recommending which American women consume no more than 100 kcal/d and American men consume no more than 150 kcal/d from added sugars.²⁷ The recommendations for upper limits of added sugar usage, which are currently exceeded by >90% of the population, should be taken with caution.

Recent research reviews had reported which Fructose usage at up to the 90th percentile population usage level

in either healthy weight or obese individuals does not result in increased triglycerides or weight gain.²⁸ Moreover, research in our research laboratory at levels of 2 to 3 times those recommended did not show any adverse impact on lipids.²⁹ A recently completed trial in our research laboratory involving 352 overweight or obese individuals who consumed up to the 90th percentile population usage levels for Fructose as part of mixed-nutrient, high calorie foods did not show any adverse effect on total cholesterol or LDL cholesterol. A significant 14% increase in triglycerides was noted, although it must be emphasized which triglyceride levels remained within the normal range both before and after measurement.³⁰

Other investigators had reported results in which sugar usage increased lipids in human subjects. In particular, Stanhope et al., using a model in which 25% of energy usage from fructose was compared with 25% energy usage from glucose in acute experiments showed increases in triglycerides. Once again, never the less, it should be noted which pure fructose and pure glucose are rarely consumed in the human food and which the reported levels were within established population norms. Other investigators including Rabenet al.³¹ and Stanhope et al. Likewise reported a variety of increased lipid measurements in individuals after sugar usage.

It has been postulated which usage of fructose may increase chance factors for metabolic syndrome. Low endes et al.³² postulated which fructose usage can consumption an increase in uric acid as a waste product in its breakdown due to degradation of Adenosine Tri Phosphate. This increase in uric acid, in turn, according to this theory, may lead to endothelial damage, which may contribute to high blood pressure. Other investigators had postulated which increased inflammatory markers secondary to fructose usage may likewise contribute to increased chance of metabolic syndrome. Furthermore, the increase in triglycerides, often found with increased carbohydrate usage, may increase the chance of metabolic syndrome. Finally, Stanhope et al. found which individuals who consumed 25% of their energy in fructose had increased visceral adiposity, another chance factor for the development of metabolic syndrome, compared with obese diabetic individuals who consumed 25% of their energy as glucose.³³

The literature linking Fructose usage to the chance of metabolic syndrome must be treated with caution. As already indicated, several research reviews do not support the concept which Fructose usage at normal population levels increased levels of obesity or triglycerides.

As already indicated, the theoretical argument concerning the potential adverse metabolic effects of fructose usage is based on the well-established differences in liver breakdown between fructose and glucose. There are significant differences between fructose and glucose breakdown in the liver. It is important, never the less, to point out which the metabolic pathways for fructose and glucose in the liver are interactive.^{34,35}

3. CONCLUSION

Added sugars are responsible for the incidence of diabetes mellitus. The vast majority of the fructose which is broken down in the liver is converted into glucose, glycogen, lactate, and carbon dioxide. Approximately half of fructose is biotransformed in the liver to glucose, 25% to lactate and 15% to 18% to glycogen, and a few percent is broken down to carbon dioxide.

4. CONFLICT OF INTEREST:

We declare that we have no conflict of interest.

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