History Of Fructose Use in Food Products

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The History of High Fructose Corn Syrup

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Food can be classified in ways other than by the nutrients they contain. In today’s fast moving culture, foods are characterized as having “magical” qualities that can turn consumers “superstars”, or reduce them to a less than optimal state of health and wellness. Sugars appear to be one of those vulnerable foodstuffs that can be enjoyed in moderation, but also contribute chronic health issues. This article will discuss the history of high fructose corn syrup in our diet, and how its metabolism may or may not be related to obesity.

HISTORY OF HFCS

The innate preference for sweet foods has been well established, in fact, the decoding of the genome has shown the exact chromosomal location for the gene that drives our consumption of sweet foods. Food manufacturers were aware of the desire for sweet foods well before the decoding of the genome and satisfied this instinctive food preference with sucrose derived from sugar cane or sugar beets. The fluctuating price in sugar cane, usually grown in tropical climates outside the United States (US), spurred American ingenuity and in the 1950s ground-breaking work in food chemistry yielded HFCS. This liquid alternative to sucrose began appearing in food products in the 1960s and existed in obscurity until the 1980s when evidence appeared, though inconclusive, implying HCFS promoted metabolic anomalies.

When it was developed, HFCS was an appealing food additive. It was generally recognized as safe (GRAS) with a Type 2 conclusion on safety by the Food and Drug Administration, added little cost, dissolved easily, and delivered a well-accepted sweet flavor. In addition, fructose, which makes up 42-55% of HFCS, was a naturally occurring product found in honey, fruits, and vegetables. These favorable characteristics allowed HFCS to emerge as a one-to-one replacement for sucrose from 1970-1998.
From 1998 to the present, sucrose and HFCS have existed in relatively equal amounts in the American diet. In 2004, Bray, Nielsen and Popkin ignited discussion in the scientific community with their article linking HFCS to the growing rate of obesity in the US. ⁶

**METABOLISM OF HFCS**

The science of fructose metabolism appears clear cut from the standpoint of biochemistry, overconsumption results in storing the energy from HFCS as fat and glycogen. Yet the real question is not only the metabolic pathways, but also what happens when we eat beyond our metabolic needs.

In any sugar over-feeding, more insulin is required to maintain a normal level of blood glucose. For many years, the similarity in structure between fructose and sucrose suggested that it would be similarly metabolized. However, meals containing high amounts of dietary fructose did not decrease the insulin and leptin levels in women and the study subjects reported lower satiety leading to an increase in food intake.¹ In hepatic insulin resistance, additional insulin is needed to achieve normal blood sugar in the presence of fructose, yet, high circulating insulin did not suppress further gluconeogenesis. The important piece of information here is not that fructose was included in the carbohydrate mix with negative results but rather that the mix was in excess of metabolic needs.⁷

In clinical studies, the individual response to varying amounts of fructose should be considered. Since any unusual effects of fructose are intermediated by interactions with glucose (metabolically a large amount of fructose converted to glucose), underlying genetic differences in the way glucose is metabolized should be considered in study interpretations. The genetic uniqueness of study subjects makes it difficult to tell if results were due to the fructose, or preexisting issues with glucose metabolism.⁶
Variations in individual response to sweetener intake make it difficult to use population level data (i.e. ecological and epidemiological data) in making a case for or against HFCS. However, the correlation between metabolic disease and HFCS exists: by the year 2002 HFCS sweeteners represented >56% of the US nutritive sweetener market, and obesity and Type 2 diabetes were on the rise.8

**OBESITY, SOFT DRINKS, AND HFCS**

That the American diet has changed over the last century is impossible to refute. The creep of HFCS into foods and into our refrigerators and pantries was only one of these changes. During the first six decades of the 20th century, consumption of dietary carbohydrates decreased. This change was largely due to a decrease in whole grains, as meat rose to the center of the plate bringing with it a 30% increase in fat consumption. Since the mid-1960s, carbohydrate intakes rose back to 19th century levels; although the mix of carbohydrates was different than it was pre-1960. Specifically, fiber intake decreased as refined foods became the preferred source of carbohydrates. It was during this same time period that caloric sweeteners gained a strong foothold in American diets with an overall increase of over 87%.8

With these changes in dietary consumption, chronic disease rates began to reflect the changes in food intake. In the 1980s prevalence of heart disease and high cholesterol increased due to the years of increased meat consumption and led to a scientific discussion about the role of fats in the diet. However, it wasn’t until 2004 that attention was acutely focused on obesity, diabetes type 2 and metabolic syndrome and the foods containing HFCS.8

Today, the controversy bubbles around the soft drink industry and obesity. At the heart of the issue is HFCS. According to White et al., HFCS levels in our diet have been in decline since 2002 yet the US obesity crisis continued to worsen during that time.9 While on the surface this data does not support an impact of HFCS on obesity, controversy continues to exist around this topic.10
The role soft drinks play in appetite stimulation is one additional effect of HCFS that is currently under investigation. However, research in this area is complex, contradictory, and unclear. Several feeding studies showed that individuals who drank caloric beverages shortly before or with a meal ate the same amount of calories as those who had a calorie free drink. Yet, moving that drink to one hour before a meal gave quite different results. In another gender stratified study, women who drank a caloric beverage one hour before a meal significantly increased their calorie load at the next meal but the opposite was seen in males. 11

CONCLUSION

Since sucrose and HFCS are similar in calories, would it make sense to restrict or eliminate HFCS from the diet only to replace it with sucrose? Or, should dietetic professionals advocate for more fiber and less refined carbohydrates as a general approach to the sweetener dilemma?

While HFCS may not be the villain it has been painted to be, looking at the total amount of sweeteners consumed in the diet is still valid. Whether it is sucrose, fructose, or a blend of the two as found in HFCS, Americans could do well to heed the adage of “everything in moderation”.

References


