High-fructose Corn Syrup: Does it Contribute to Obesity?

Brittney Getz
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Abstract

The increased consumption of high-fructose corn syrup (HFCS) has often been implemented in the obesity debate. The increase in consumption of HFCS occurred at the same time as the rapid increase in the rates of obesity in the United States. However, very little research lends evidence to the theory that HFCS is uniquely responsible for obesity. HFCS is similar in composition to sucrose and is most likely metabolized similarly. Also, they have similar levels of sweetness. Short-term studies on the metabolic effects of HFCS in humans has shown that it has similar effects as sucrose on levels of insulin, glucose, leptin, ghrelin, and triglycerides. While long-term studies in animals have revealed slightly different findings, it is evident that more long-term studies are necessary. It is likely that the increase in obesity is due to many factors such as increased fast food consumption and decreased amounts of physical activity. However, the increased consumption of soft drinks has also had an effect. There is more evidence relating the increase in consumption of soft drinks than the increase in consumption of HFCS to the increase in rates of obesity in the US. These soft drinks would have this effect on obesity even if they were sweetened with sucrose as the effect is due to the additional calories that are present in soft drinks as they are not as satiating as solid foods with the same amount of calories.
I. Introduction

Obesity is a major health concern in the United States. With the growing rates, 2 in 3 adults are either overweight (BMI between 25 and 29.9) or obese (BMI 30 or greater) (Ogden et al., 2006). The rate of childhood obesity is also on the rise. Severe childhood obesity has tripled in the past 25 years (Skelton et al., 2009). Obesity can lead to many negative health effects such as hypertension, coronary heart disease, strokes, type 2 diabetes, cancer, and arthritis (Haslam & James, 2005).

This rise in obesity can be attributed to many factors such as lack of exercise in a world that increasingly relies on automobiles for transportation and high calorie diets that consist of fat and sugar-rich foods. One aspect of the diet in particular that has frequently been criticized for playing a role in the obesity epidemic in the US is the increased consumption of high-fructose corn syrup (HFCS) over the past few decades (Bray, Nielsen, & Popkin, 2004). This rise in use of HFCS mirrors the rise in obesity in the US, so HFCS has often been implicated in the obesity epidemic.

HFCS is present in many foods today such as soft drinks, fruit drinks, baked goods, canned fruits, and even condiments such as ketchup (Forshee et al., 2007). However, of all added sweeteners in the diet, soft drinks are the most commonly consumed (Guthrie & Morton, 2000). The amount of soft drinks consumed has increased substantially over the past 20 years (Harrington, 2008). Adolescents especially have increased their consumption. Therefore most of the research that has been done on the negative effects of HFCS focuses on soft drink consumption.

This review aims to examine the supporting and refuting evidence that HFCS is uniquely responsible for obesity. The composition of HFCS will be explained as well as its changes in use
across the past 30 years. There is an increasing body of research that claims that HFCS does not yield different health effects than common table sugar (sucrose). If HFCS is contributing to the obesity epidemic, this is because it is present in many soft drinks and fruit juices that are commonly consumed by Americans. In order for HFCS to have a unique role in the obesity epidemic, it must be shown that it yields different effects than sucrose, and its consumption must have increased more than the consumption of other foods that are associated with weight gain.

II. How HFCS came into being and its composition

High-fructose corn syrup was developed in the late 1960s because of the invention of technology that could convert corn starch to corn syrup, which could then be converted to fructose. This is a four-step process that involves acquiring starch through the wet milling of corn, hydrolyzing this starch to yield dextrose (also known as glucose), converting this dextrose to fructose, and increasing the fructose concentration (Buck, 2001).

In the early 1980s, soft drink companies began replacing sucrose with HFCS as it was similar in sweetness to sugar, it was a cheaper alternative, and an abundance of corn products were being produced. Also, HFCS had a longer shelf-life than sucrose, so it saved money for both major soft drink companies in the US. In 1980, Coca-Cola first replaced sucrose with HFCS-55, using it as 50% of the sweetener in their products. In 1983, Pepsi-Cola made the same decision. It was not until 1984, however, that both companies decided to use HFCS-55 as 100% of the sweetener in their soft drinks (Buck, 2001).

The first type of HFCS to be created was HFCS-42, so named because it consisted of 42% fructose, 53% glucose, and 5% polymers of glucose, mainly maltose (two glucose molecules held together by a glycosidic bond) and maltotriose (three glucose molecules held together by a glycosidic bond). This version of HFCS was not widely accepted because it was
not as sweet as sucrose, the most commonly consumed sweetener at the time. Shortly after, it also became possible to manufacture HFCS-90, consisting of 90% fructose, 9% glucose, and 1% higher saccharides, such as maltose or maltotriose, by filtering HFCS-42 through an ion-exchange column which retained more fructose (Smith & Bradley, 1983). However, this version was not a viable alternative to sucrose because it was much sweeter due to the high amounts of fructose present. In order to create HFCS-55, that was closer in sweetness to sucrose, manufacturers first created HFCS-90 and mixed it with HFCS-42 (Schorin, 2005). HFCS-55 is composed of 55% fructose, 42% glucose, and 3% polymers of glucose (White, 2008). This is the most commonly consumed form of HFCS as it is the type that is commonly found in soft drinks, and therefore, the majority of research on HFCS explores the effects of HFCS-55. HFCS-55 is also found in many frozen desserts and ice cream. HFCS-42 is more commonly found in baked goods, condiments, and canned fruits. HFCS-90 is used rarely; its most common usage is in natural foods where small amounts can produce necessary sweetness (Forshee et al., 2007).

There have also been informal claims that HFCS is sweeter than sucrose, thus making it more palatable (Bray, Nielsen, & Popkin, 2004). However, this has not been supported by scientific research (White, 2008). Sucrose and HFCS-55 have been rated as having the same relative sweetness. Although fructose itself is rated to have a higher intensity of sweetness, the small increase in the amount of fructose in HFCS-55 as compared to sucrose has not been found to have much of an effect on the difference between the sweetness of the two. This shows that the switch of soda companies from the use of sucrose to the use of HFCS-55 has not had a large effect on the sweetness of the beverage which is consumed by many Americans.

Much of the literature that attributes HFCS to obesity has been wrongly based on the assumption that HFCS is equivalent to corn syrup or fructose or that it is greatly different from
the composition of sucrose (Bray, Nielsen, & Popkin, 2004). However, the composition of HFCS-55 is actually quite similar to that of sucrose, which consists of 50% fructose and 50% glucose that are held together by a covalent bond (Forshee, Anderson, & Storey, 2007). Also, corn syrup consists of mostly glucose that may or may not be bonded, and it contains no fructose (White, 2008). The assumption that HFCS yields the same effects as fructose is also incorrect. This is a misconception that may be due to the name of HFCS. “High-fructose” implies that HFCS contains a significantly greater amount of fructose than other sweeteners, such as sucrose. This is simply not the case, as the most common form of HFCS used, HFCS-55, contains amounts of fructose that are similar to the amounts present in sucrose (Schorin, 2005).

III. Metabolism

However, the role of fructose in HFCS consumption may be important as HFCS is composed of fructose in its monosaccharide form mixed with glucose in its monosaccharide form, while sucrose contains fructose that is chemically bonded to glucose. There are many studies that suggest that fructose metabolism is different from glucose metabolism and that fructose differentially affects other mechanisms that control feeding behavior. The metabolism of sucrose is not different from that of HFCS as it too can only be absorbed by the intestinal epithelium of the small intestine when it is in its monosaccharide components. However, sucrose must be cleaved by the enzyme sucrase in the small intestine into free fructose and free glucose, a step which is missing in the metabolism of HFCS (Schorin, 2005). This however causes no differences in the absorption of fructose and glucose that have been cleaved by sucrase (as is the case with sucrose absorption) or the absorption of fructose and glucose that are already present in their monosaccharide forms (as is the case with HFCS). The absorption of fructose across the small intestine is different from that of glucose in that glucose is transported across the intestinal
epithelium via SGLUT-1 that is dependent on sodium, while fructose absorption does not depend on the presence of sodium (Riby, Fujisawa, & Kretchmer, 1993). Fructose is carried across the intestinal epithelium by the transport mechanism GLUT-5 (Skoog & Bharucha, 2004).

Digestion of carbohydrates occurs such that when food enters the mouth, it is passed to the stomach. From here it is released periodically to the small intestine where enzymes from the pancreas and small intestine help to break down polysaccharides such as sucrose into their monosaccharide forms so they can be absorbed by the small intestine. The absorbed monosaccharides are then transported in the bloodstream to the liver, where they influence the nutrients that are released into the bloodstream. When glucose is present in the bloodstream, it can be used by the brain for energy, as it is the sole source of energy in the brain. The pancreas secretes insulin when glucose is present in the bloodstream. The presence of insulin can cause glucose to be converted into glycogen by the liver for storage or it can be converted into fat by the adipose tissue. During fasting, there is less glucose present in the bloodstream, and the pancreas secretes glucagon which causes the liver to convert glycogen into glucose to provide energy to the body. There are other influences on hunger in the body such as neurochemicals. Leptin is one such chemical that can control feeding behavior. When fat levels in the body are low, leptin levels are also low. Low levels of leptin can serve to initiate feeding. Ghrelin is a hormone released by the endocrine cells of the stomach. Ghrelin levels are lowest immediately after eating, and they increase as fasting continues. The level of triglycerides present in the bloodstream after consumption is also important. When nutrients from food are being absorbed, triglycerides are present in the bloodstream, they are transported from the small intestine to adipose tissue where they are broken down into free fatty acids and monoglycerides before they can they can be reformed into triglycerides for storage in the adipose tissue. During fasting, the
adipose tissue breaks down the triglycerides into glycerol and free fatty acids, which are used by the body for energy.

Fructose metabolism has been found to have different effects on insulin, leptin, ghrelin, triglycerides, and free fatty acids compared to metabolism of equivalent amounts of glucose. This has been found in a study by Teff et al. (2004), which highlights many of the findings with regards to the differential metabolism of fructose and glucose. They took blood samples from lean women throughout day one of the study. Sampling occurred most frequently around the three meal times, so that levels of circulating ghrelin, leptin, insulin, and glucose after consumption of a meal with either a glucose or fructose sweetened beverage could be properly analyzed. In day two of the study, participants were given the chance to eat as much as they desired of the food that was presented at each meal. They found that they levels of circulating glucose in the blood were smaller after consumption of a meal with a fructose-sweetened beverage, with the level of plasma glucose getting progressively lower with each ingestion of a meal with a fructose-sweetened beverage. Plasma insulin levels were also lower after consumption of a meal with a fructose-sweetened beverage. The production of leptin was also found to be lower for the day when the fructose-sweetened beverages were consumed compared with the days that the glucose-sweetened beverages were consumed. Measurements of ghrelin in the bloodstream were lower after consumption of a meal with a glucose-sweetened beverage than after consumption of a meal with a fructose-sweetened beverage indicating that ghrelin was suppressed more by the presence of glucose. This means that ghrelin levels are smaller after ingestion of a beverage that contains more glucose so the participant’s hunger is suppressed longer after the ingestion of a beverage with higher amounts of glucose. They found that the amount of triglycerides present in the bloodstream did not decrease as much after the
consumption of a meal with a fructose-sweetened beverage as it did after a meal with a glucose-sweetened beverage and the level of triglycerides remained higher throughout the night when fructose-sweetened beverages were consumed. High levels of triglycerides can lead to negative health effects such as a greater risk for heart attacks because too many triglycerides in the bloodstream can thicken the blood and make it more susceptible to clotting. There were no changes found in the amount of free fatty acids present in the blood stream. Also, no difference was found between the amount of calories, fat, or carbohydrates consumed between the glucose and fructose testing days.

In line with previous research, this study supports the finding that fructose is unable to promote insulin secretion from the pancreas. This is thought to be caused by the lack of GLUT-5 transporters in the pancreas. The authors point out that the decrease in leptin that results from the consumption of fructose-sweetened beverages is most likely caused by the lesser amounts of insulin and glucose in the body causing glucose to not be used by adipose tissue. Therefore, leptin is not released in sufficient amounts from the adipose tissue, so the body does not receive sufficient information about how much fat is present. The decreased suppression of ghrelin by fructose-sweetened beverages implies that fructose is not as satiating as glucose, as higher amounts of ghrelin are related to increased appetite.

It has also been found that in the presence of glucose, fructose absorption is facilitated. So while pure fructose alone has been found to be incompletely absorbed in many people, the presence of glucose or other carbohydrates allows fructose to be absorbed (Riby, Fujisawa, & Kretchmer, 1993). This has been shown because fructose malabsorption does not occur when glucose is present or with the ingestion of sucrose, which results in free fructose and glucose (Rumessen & Gudmand-Høyer, 1986). The characteristics of fructose malabsorption are
flatulence, abdominal pain and distention, rumbling, and diarrhea. Rumessen and Gudmand-Høyer (1986) measured the responses of healthy adults to different concentrations of ingested sucrose, fructose, glucose, and fructose with glucose using hydrogen breath tests, which indicate that unabsorbed fructose has reached the large intestine where nutrients travel when they are unable to be digested (Riby, Fujisawa, & Kretchmer, 1993). They found no malabsorption of sucrose or glucose. However when they compared the malabsorption of fructose to equivalent amounts of fructose that would be hydrolyzed from sucrose, significant differences were found. Fructose malabsorption was found when all concentrations of pure fructose were ingested, however, there was more malabsorption at higher concentrations. When fructose was combined with increasingly higher concentrations of glucose, the percentage of participants who suffered from malabsorption decreased. When fructose and glucose were ingested at a 1:1 ratio, no malabsorption was observed. This study also revealed that some individuals may not even be able to absorb fructose at very small amounts.

Considering the evidence that fructose is metabolized better in the presence of glucose and that sucrose, like HFCS, can only be absorbed in its monosaccharide components, it is necessary to examine the effects of HFCS itself, not glucose and fructose separately, in order to fully understand its effects on metabolism and how it is similar to and different from sucrose. The following studies that examine the effects of HFCS directly as compared to other sweeteners provide further support for the theory that the effects of HFCS cannot be examined using fructose because there are significant differences found between the effects of HFCS compared to fructose. The research that compares the metabolic and endocrine effects of HFCS to sucrose does not reveal such significant differences as are found between glucose and fructose.
It is important to note that sucrose in soft drinks often hydrolyzes to the monosaccharide form of fructose and glucose by the time it is consumed, so it is similar in composition to HFCS (Schorin, 2005). However, the amount of sucrose that is hydrolyzed depends on many factors such as the temperature that it is stored at, the amount of time since the soft drink was manufactured, and the pH content of the beverage. Monsivais, Perrigue, and Drewnowski (2007) accounted for this possibility in their comparison of the effects of a beverage sweetened with sucrose, a beverage sweetened with high-fructose corn syrup, diet soda, and milk on participants’ ratings of satiety, sweetness, and the amount consumed at a meal after ingestion of each beverage. They analyzed the extent to which the sucrose sweetened beverage they were using had hydrolyzed to glucose and fructose monosaccharides at the time of consumption. They found that the amount of sucrose present at the time of administration (10% of the total sugar present) of the sucrose-sweetened beverage had declined substantially from the amount present at the time of its manufacture (36% of total sugar present). The amount of fructose present in its monosaccharide form was 32% of total sugar at the time of manufacture to 45% at the time of consumption. This provides further evidence that sucrose sweetened soft drinks are similar in composition and thus, metabolism to high-fructose corn syrup sweetened soft drinks.

This study also revealed no differences between HFCS-42, HFCS-55, and sucrose on measures of satiety and perceived sweetness. Participants were given a HFCS-42 sweetened soft drink, a HFCS-55 sweetened soft drink, a sucrose sweetened soft drink, a diet soft drink, milk, or no beverage, and two hours and twenty minutes later, they were given a test meal and allowed to consume as little or as much as they desired. Every 20 minutes throughout the test day, participants rated their hunger, desire to eat, and fullness. They also rated the sweetness of the beverage they consumed. It was found that the beverages sweetened with HFCS-42, HFCS-55,
and sucrose were rated equally on perceived sweetness. Also, there was no difference in the ratings of these three beverages on satiety, hunger, or desire to eat at any time during the study. However, these results should be interpreted with caution, as they are based on participants’ subjective ratings of these beverages and not the physiological effects that these various sweeteners may have.

However, further research has been done examining the endocrine and metabolic effects of HFCS compared to sucrose and other sweeteners. In one study by Akhavan and Anderson (2007), subjective satiety was measured as well as responses of satiety hormones after the consumption of different ratios of glucose to fructose solutions. Beverages contained glucose to fructose ratios that were similar to the ratios of HFCS-55 and sucrose as well as solutions containing either more glucose or more fructose. Sucrose itself was also used. Participants consumed a beverage sweetened with one of four glucose to fructose ratio solutions, a flavored water, or regular water, and 75 minutes after the consumption of the beverage, they were allowed to eat as much pizza as they desired. Blood samples were taken and satiety questionnaires were completed every fifteen minutes after the consumption of the beverage. It was found by looking at the amount of pizza consumed by each participant that caloric compensation was greatest after consumption of the beverage containing the highest glucose to fructose ratio (80% glucose, 20% fructose). So participants compensated more by consuming fewer calories after consumption of a beverage with a high glucose to fructose ratio. However, all beverages suppressed the amount of food eaten at the test meal more than water except for the solution with the lowest glucose to fructose ratio (20% glucose, 80% fructose). The levels of plasma uric acid were increased above the control for all ratios with the exception of the 80% glucose/20% fructose solution. High levels of uric acid can cause gout and kidney problems. The similar effects of sucrose and HFCS
on uric acid levels is important because it implies that neither is more capable than the other of inducing metabolic syndrome. Blood glucose levels were higher than all other solutions at each measurement after consumption of the 80% glucose/20% fructose solution and lower than all other solutions for the 20% glucose/80% fructose after consumption. Consumption of the 80% glucose/20% fructose solution also yielded the highest insulin concentrations, while consumption of the 20% glucose/80% fructose and 35% glucose/65% fructose solutions yielded the lowest insulin concentrations. All sweetened solutions resulted in ghrelin concentrations that were lower than ghrelin concentrations following consumption of water. As noted by the authors, this study provides evidence that the immediate physiological effects of sucrose (containing chemically bonded glucose and fructose) do not differ from the effects of the 50% glucose/50% fructose solution (containing glucose and fructose in their monosaccharide forms). The authors attribute the finding that concentrations of ghrelin in the blood are not significantly different to the finding that ghrelin may respond similarly to all sugars, regardless of the concentrations of glucose to fructose. The physiological effects of HFCS and sucrose do not seem to differ in the short term; however this study is limited by its sample size of young males.

Further evidence of the similarities between HFCS and sucrose can be found in a study by Melanson et al. (2007). Lean women were given controlled meals with a beverage sweetened with HFCS-55 or sucrose during the first day of the study. Blood samples were taken this day as well as appetite ratings. In day two of the study participants were able to eat as little or as much as they desired. This study found similar results to Akhavan and Anderson (2007) in that there were no differences found between consumption of HFCS sweetened beverages and sucrose sweetened beverages on circulating blood levels of glucose, insulin, and ghrelin. This study also looked at the effects of HFCS and sucrose sweetened beverages on circulating leptin levels.
Appetite ratings were similar between the two as well. This is to be expected due to their similar metabolic effects. A study by Soenen and Westerterp-Plantenga (2007) also found that the compensatory food intake after ingestion of a HFCS or sucrose sweetened beverage did not differ. However, this compensatory food intake was not enough to account for the additional calories consumed from either of the beverages, suggesting that soda intake in general may cause overconsumption of calories.

A similar study was conducted to compare the metabolic effects of HFCS to fructose and glucose, as well as sucrose by Stanhope et al. (2008). Women and men with a range of body weights, including normal, overweight, and obese, consumed beverages sweetened with HFCS or sucrose. A small sample of men in the study also consumed beverages sweetened with pure fructose or pure glucose. No differences were found between HFCS and sucrose on levels of glucose, leptin, ghrelin, triglycerides, or free fatty acids in the blood. However there was an effect of gender. The level of leptin in the blood of women was about 3 times higher than that of men during consumption of both the HFCS and sucrose sweetened beverages. Low levels of leptin can cause the initiation of feeding. The triglyceride level in men was 3 times higher than that of women during consumption of the sucrose sweetened beverage and 7 higher than that of women during consumption of the HFCS sweetened beverage. Again, high levels of triglycerides can make the blood more susceptible to clotting. The authors point out that these gender differences have been found in other studies. The level of insulin in the blood was slightly higher following consumption of the sucrose sweetened beverage, but this difference was found only in subjects under the age of 35. This could be due to the fact that sucrose contains more glucose than HFCS, but it may simply be due to variability as both Melanson et al. (2007) and Soenen and Westerterp-Plantenga (2007) did not find differences between HFCS and
sucrose on insulin levels. Further research is necessary to confirm these differences. No differences were found in the small subset of men in the study between HFCS, sucrose, fructose, or glucose sweetened beverages on leptin, ghrelin, or free fatty acids. The plasma glucose and insulin levels were lower after consumption of a fructose sweetened beverage compared to a glucose sweetened beverage with the levels after consumption of HFCS or a sucrose sweetened beverage in between those of the glucose and fructose sweetened beverages. This is to be expected due to the similar fructose and glucose contents of HFCS and sucrose. However, it was found that the level of plasma triglycerides was higher after consumption of a fructose, HFCS, or sucrose sweetened beverage compared to a glucose sweetened beverage. These triglyceride responses of HFCS and sucrose are larger than would be expected due to their fructose and glucose content. The authors suggest that this may be the result of the metabolism of glucose in the liver, which may have been stimulated by fructose. They base this off of research that has found that ingesting fructose can lower the plasma glucose responses to ingested glucose. Further research needs to be done to determine whether the similar levels of triglycerides after consumption of a fructose, HFCS, or sucrose sweetened beverage are maintained. However, this study provides further evidence for the similar responses caused by ingestion of a HFCS or sucrose sweetened beverages and that they yield different responses than ingestion of a fructose or glucose sweetened beverage. Also, the similar physiological responses between HFCS and sucrose sweetened beverages can be generalized to normal weight, overweight, and obese people.

While studies on humans have revealed no differences between HFCS and sucrose sweetened beverages, one study on female rats has found differences (Light, Tsanzi, Gigliotti, Morgan, & Tou, 2009). In this study, rats drank a water control or water sweetened with either
Those rats drinking the glucose sweetened water drank the most, followed by the rats drinking the sucrose sweetened solution, and then followed by the rats drinking the HFCS-55 sweetened solution, the fructose sweetened solution, and the water control. Rats drinking the water control consumed the most food, and rats drinking glucose consumed the least amount of food. There was no difference in food intake found between rats drinking the glucose solution or the sucrose solution, between rats drinking the sucrose solution or the HFCS solution, between the rats drinking the HFCS solution or the fructose solution, or between rats drinking the fructose solution or the water control. The rats drinking the glucose sweetened solution had the greatest total energy intake, while the rats drinking the water control had the lowest total energy intake. There was no difference in total energy intake between the rats drinking the HFCS, sucrose, or fructose solutions. The body weights of the rats after 8 weeks were only higher than the control for rats drinking the HFCS solution. These rats also had higher body weights than rats drinking the glucose solution. However, no differences were found between the body weights after 8 weeks of the rats drinking the HFCS, sucrose, or fructose solutions. As can be seen in Figure 1, the final body weights of the rats drinking the HFCS solution were higher than the final body weights of the rats drinking the water control and the glucose solutions, but there was no difference between the rats drinking the HFCS, sucrose, or fructose solutions. Unlike what has been found in previous studies, there were no differences between rats drinking sucrose, fructose, glucose, HFCS, or the water control on levels of plasma leptin, cholesterol, or triglycerides. This could mean that responses to HFCS, sucrose, fructose, and glucose are different in rats compared to humans. A replication of this study would be useful to determine if these results are upheld. Also, the use of male rats is necessary in future research. The physiological effects of sucrose and HFCS in this study were not intermediary
between glucose and fructose as has been found in previous research. The authors noted that the increased total intake of the rats drinking the glucose solution but their lack of overall body weight gain compared to other groups may have been due to their decreased feed efficiency caused by their increased urine excretion and thirst. Also, more time may be necessary before a difference between the body weights of rats drinking fructose or sucrose can be compared to rats drinking the water control. This study also did not examine the amount of energy expended by these rats, as it is possible that the amount of energy expended was lowest by the rats drinking the HFCS solution.

Figure 1. Body weight at each weekly measurement of rats consuming the water control, glucose, sucrose, fructose, and HFCS-55

Another study has examined the effects of drinking a HFCS sweetened solution in mice. This study attempted to develop a mouse model of obesity for nonalcoholic steatohepatitis (NASH), a disease characterized by excess fat in the liver that can cause cirrhosis. Mice were
given a Western lifestyle for 16 weeks by consuming HFCS-55 sweetened water, a diet high in trans-fat, and no cage racks were included to prevent them from exercising. Another group of mice experienced the same conditions without a trans-fat diet and another group without HFCS-55 sweetened water. A control group was also included without the trans-fat diet, without HFCS-55 sweetened water, and with cage racks. They found that the mice in the HFCS-55 and trans-fat condition consumed 13.2% more calories than the mice who consumed water without HFCS-55. Mice who consumed the trans-fat diet without HFCS-55 sweetened water were found to be more tolerant of glucose and more insulin sensitive than mice who consumed the HFCS-55 sweetened water. Also, mice who consumed both the HFCS-55 sweetened water and the trans-fat diet weighed significantly more than the control group as well as the group that consumed the trans-fat diet without the HFCS-55 sweetened water. The greater consumption of food by mice who consumed HFCS-55 is noted as a potential role for HFCS in the obesity epidemic. However, the amount of HFCS consumed in this experiment would be the equivalent of a human drinking 8 soft drinks a day, which is quite a bit higher than what is commonly consumed (Nielsen & Popkin, 2004). The typical amount of soft drinks consumed is 7% of the daily caloric intake, amounting to 140 calories of a 2,000 calorie diet. This is equal to about one soft drink consumed per day. Also, the higher body weights of the mice in the experimental conditions must be interpreted with caution as this study did not provide a sufficient control group. The access to the cage rack provided the control group with the opportunity to exercise while none of the experimental groups were provided with this option. However, this study does provide evidence of what can occur with long term heavy consumption of HFCS-55, but it is necessary to explore the effects of long-term moderate consumption of HFCS-55 as that is more in line with current soft drink consumption patterns. Also, the role of sucrose sweetened
beverages was not explored in this study, and it is possible that sucrose sweetened water would have had the same effects as HFCS-55 sweetened water on the amount of weight gained and the insulin and glucose responses (Tetri, Basaranoglu, Brunt, Yerian, & Neuschwander-Tetri, 2008).

Little evidence is available about the effects of long-term HFCS consumption in humans. One study has been conducted examining the effects of consuming a HFCS sweetened beverage for 3 weeks (Tordoff & Alleva, 1990). Participants who drank a HFCS-sweetened beverage for 2 weeks gained significantly more weight than the control (no beverage consumed) and the difference was even larger at three weeks. This study, however, compared the effects of drinking a HFCS sweetened beverage to an aspartame sweetened beverage. Therefore, it cannot be determined if HFCS sweetened beverages result in significantly more weight gain than beverages sweetened with other caloric sweeteners such as sucrose. There is also evidence from this study that consuming sweetened beverages whether with HFCS or aspartame decreases consumption by about 179 to 195 kcal per day. Although, this is not enough compensation to account for the additional calories from the HFCS sweetened beverage so weight gain occurs. Another study has compared the long term effects of drinking a sucrose sweetened beverage and an artificial sweetened beverage (Raben, Vasilaras, Møller, & Astrup, 2002). In this 10 week study of overweight participants, increases in blood pressure, fat mass, and body weight were found in the participants who drank the sucrose sweetened beverages. The findings from these studies indicate that consumption of calorically sweetened beverages in general whether they are sweetened with HFCS or sucrose can lead to weight gain.

It is important to note that the research that has been discussed thus far has focused mainly on HFCS as it is found in beverages. This is because HFCS is most commonly consumed in soft drinks (Guthrie & Morton, 2000). It also may be due to the finding that caloric beverages
are not as satiating as solid foods and they are not adequately compensated for in later consumption (DiMeglio & Mattes, 2000). Research must be conducted on the effects of consuming other food sources that contain HFCS, as well as the prevalence of consumption of other food sources that contain HFCS. Based on the data from HFCS in solution it is likely that pastries sweetened with HFCS would have the same metabolic and endocrine effects as pastries sweetened with sucrose.

Research has shown that the similar compositions of HFCS-55 and sucrose make it unlikely that the switch by major soft drink companies from the use of sucrose to HFCS-55 would have had little difference on the development of the obesity epidemic in America. Although sucrose contains fructose and glucose in equal amounts and they are chemically bonded, it must be broken down into its monosaccharide components before it can be metabolized. Therefore, the small intestine absorbs fructose and glucose from HFCS and sucrose in the same manner. As studies that have been conducted on humans show that HFCS and sucrose produce similar metabolic and endocrine effects, it seems unlikely that one would be more satiating than the other. Studies that have been done on rodents that have revealed different effects between HFCS and sucrose need to be replicated as they contrast with the data that has been found from human studies. The majority of the studies that have been conducted with humans have been short-term studies. Long-term studies of the effects of HFCS and sucrose on weight gain must be conducted in humans before any more definitive conclusions can be made.

IV. Changes in use of HFCS and soft drink consumption

One study in particular is responsible for the theory that the consumption of HFCS is related to the obesity epidemic. Bray, Nielsen, and Popkin (2004) conducted a review of the
literature on fructose metabolism as it compares to glucose metabolism, and used this as evidence that HFCS metabolism differs from sucrose metabolism. They use findings that fructose is sweeter than sucrose to support their theory that HFCS sweetened beverages are sweeter than sucrose sweetened beverages. The similarities in the increase in consumption of HFCS are associated with the increase in the obesity epidemic, as both have markedly increased since the 1970s. This article attributes the negative effects of fructose consumption to HFCS consumption because it contains more than 50% fructose (which is more than sucrose), although the increased amount of fructose that is present in HFCS is relatively small and the composition of HFCS is much closer to that of sucrose than to pure fructose. However, this is not considered.

The increase in the consumption of HFCS and sweetened beverages is proposed to play a role in the increase in fructose consumption. However, if sucrose were present in sweetened beverages as opposed to HFCS, the fructose consumption of Americans would still have increased. A more likely contributor to the obesity epidemic is the increased use of sugar sweetened beverages.

There is a great deal of evidence linking consumption of soft drinks to weight gain. It has been found that from 1977-2001, Americans have increased their consumption of soft drinks from 50 kcal per day to 144 kcal per day, almost three times as much, while milk consumption has decreased from 143 kcal per day to 99 kcal per day which comes from a decrease in both volume consumed as well as the use of milk that is lower in fat. Increases in other beverages such as coffee, tea, and alcohol have been minimal. Americans aged 2-18 experienced the biggest decrease in milk consumption between 1977 and 2001. The largest increase in soft drink consumption occurred in Americans aged 2-18 and 19-39 (Nielsen & Popkin, 2004). This suggests that the increase in soft drink consumption may play a greater role in obesity than HFCS. Even when soft drinks are sweetened with sucrose, people are unable to compensate
enough for these extra calories (Soenen and Westerterp-Plantenga, 2007). This study implies that children are consuming fewer valuable nutrients (due to their decreased milk consumption) and consuming more unnecessary calories that provide little or no nutritional benefit (due to their increased consumption of soft drinks).

Soft drinks may be a more likely culprit in the obesity epidemic in America than HFCS. It is true that the presence of HFCS in soft drinks provides calories that can promote obesity when they are persistently consumed without enough compensation or energy expenditure, but sucrose also provides these extra calories. There are many studies that provide evidence that liquid calories do not provide as much satiation as the same amount of calories in solid form. One study has compared the effects of consuming a soft drink with the effects of consuming jelly beans which provide similar nutritional benefits and were matched for energy content (DiMeglio & Mattes, 2000). It was found that after consumption of the jelly beans, participants were able to adequately compensate for the extra energy consumed. However, participants did not compensate for the additional calories after compensation of the soft drink. This study shows that consumption of sugar in a liquid form is not as satiating as consumption of sugar in a solid form, and thus people are more likely to overconsume after consumption of a sugar sweetened beverage.

There is a reported increase in fructose consumption since 1978, but this is due to the effects of consumption of HFCS, consumption of sucrose, as well as the increased consumption of soft drinks. The peak daily availability of sweeteners was in 1999 at 187.9 g/day, but it has declined slightly since then and has remained steady since 2003 at 176 g/day. The ratio of HFCS consumption to sucrose consumption has remained constant since 1999 at 44:42 respectively. The availability of sweeteners increased by 16.6% between 1978 and 2003, while HFCS
availability increased by 60.8% and the availability of sucrose decreased by 32.7%. It was estimated that men had greater intakes of fructose, however men aged 15-22 had the highest mean fructose intake. In women, those aged 15-22 also had the highest mean fructose intake. Nonalcoholic beverages (including soft drinks and fruit drink) account for the largest percentage of fructose in the diet (54.3%) followed by grain products (20.3%) such as flour and cereal products. Men and women aged 15-22 also had the highest intake of fructose from nonalcoholic beverages (Marriott, Cole, & Lee, 2009). It has also been found that regular soft drinks specifically constitute the largest percentage of added sweeteners to the diet (Guthrie & Morton, 2000). French, Lin, & Guthrie (2003) found that the majority of soft drink consumption in children occurs at home. From 1994-1998 it has been reported that children aged 6-17 consumed an average of 12 oz. of soft drinks a day, this was increased from estimates from 1977-1978 of 5 oz. of soft drinks a day. Also, it was estimated that from 1994-1998, 56% of all children drank soft drinks on a particular day, while from 1977-1978, 37% of all children drank soft drinks on any particular day. This provides evidence that the greatest concern for overconsumption of beverages that contain fructose is in teenagers and young adults.

There is much more research that relates consumption of soft drinks containing HFCS to obesity than research that relates HFCS itself to obesity. In a study examining the soft drink consumption of schoolchildren, it was found that there was a greater chance of becoming obese for each additional sugar sweetened beverage consumed every day (Ludwig, Peterson, & Gortmaker, 2001). Increased consumption of diet soft drinks was associated with a decrease in obesity. One randomized controlled study of adolescents aged 13 to 18 has also found links between sugar sweetened beverage consumption and body weight (Ebbeling et al., 2006). In this 25 week study, children were provided with either noncaloric or caloric beverages and their
changes in weight and BMI were monitored. It was found that decreasing intake of sugar sweetened beverages was associated with a lower body weight. Of the children tested, those who were in the top third of the BMI at baseline had the largest change in BMI due to consumption of noncaloric beverages as compared to caloric beverages. These studies suggest that a good way in which to decrease childhood obesity is to replace sugar sweetened soft drinks with noncaloric sweetened soft drinks. However, again, the effects of drinking a soft drink sweetened with HFCS do not seem to be different from the effects of drinking a soft drink sweetened with sucrose.

There is also evidence linking the consumption of soft drinks to increased risks for diabetes type II (Schulze et al., 2004). Like previously mentioned research, this study also found increased weight gain for women who increased their consumption of sugar sweetened soft drinks. Increased consumption of sugar sweetened soft drinks correlated with an increased risk for type II diabetes. The authors attributed this increased risk to the fact that liquid carbohydrates are more absorbed more quickly than solid carbohydrates (DiMeglio & Mattes, 2000).

The theory that HFCS is a strong contributor to obesity is very weak because it can easily be accounted for by the increased consumption of soft drinks since the 1970s. Children especially have begun consuming more sugar sweetened beverages and fewer beverages such as milk that are nutritionally helpful. Soft drinks have also been associated with negative health consequences such as diabetes type II. Also, while there has been increased consumption of fructose over the past 30 years, this increase is not due to HFCS alone as sucrose also contains fructose and the higher consumption of soft drinks in general would lead to a greater use of fructose.
V. Conclusions

High-fructose corn syrup has often been given a negative reputation in America. With obesity being such a huge problem in the United States, people are quick to look for culprits. HFCS is an easy target the increases in its use mirrored the increases in the numbers of overweight and obese people in the US. This correlation is easy to interpret as causality. However, many other changes occurred during this time period that can better account for the increases in obesity in the US. It is possible that obesity can partially be accounted for by decreases in physical activity that occurred as people became ever more reliant on automobiles for transportation. There has been a decrease in smoking which has increased the rate of obesity. The increase in portion sizes and in average numbers of calories consumed per day also has been playing a role (Bray, Nielsen, & Popkin, 2004).

Ever since soft drinks were introduced into the US diet, people were reluctant to attribute obesity to this frequently consumed and enjoyed beverage and more willing to attribute it to the new product used by the soft drink companies, high-fructose corn syrup. However, there is more evidence linking the increase in obesity to the increase in use of soft drinks than the increase in the use of HFCS. Although it is still possible that consumption of soft drinks is not a strong factor in the development of obesity and that its influence may be accounted for by other factors (Sun & Empie, 2007; Bremer, Auinger, & Byrd, 2009). However, in any case the use of sugar sweetened beverages must be monitored because it has been shown that sugar in liquid form cannot be adequately compensated for like sugar in solid form.

The media has been a strong influencing factor on the negative perception of HFCS. The name “high-fructose” corn syrup has led people to believe that HFCS yields similar effects to fructose. Therefore the negative research that surfaces about fructose has been attributed to
HFCS, this has been publicized by the media causing consumers to become weary of it. However a good body of research has revealed that HFCS has similar effects to sucrose at least in the short term, as both yield similar effects of levels of insulin, glucose, leptin, ghrelin, and triglycerides in the bloodstream. However, many consumers are not aware of this research and are flocking towards “natural” sugars such as common table sugar (sucrose). However, “healthy” sugar does not exist. The reality is that sugar provides little nutritional benefits in any processed form. The increased use of sugar in the United States is also a major contributor to obesity, as sugars are less satiating than proteins. If obesity is to be avoided, processed sugar consumption needs to decrease and an increase in healthier eating must occur.

The long-term effects of HFCS consumption as compared to sucrose consumption must be studied in more detail so that any differences can be determined. As these studies are not always ethical in humans, more animal research is necessary. However, these results must be interpreted with caution as it seems that the effects of HFCS consumption in animals may not be identical to the effects of HFCS consumption in humans. Also, research on the differential effects of HFCS and sucrose in diabetes patients may be useful, although again, this may not be practical. An animal model of diabetes should be developed to explore this.

It is also interesting to note the increased use of HFCS in the diet as a preservative. Many breads and cereal products now contain HFCS to increase shelf-life. HFCS is now found in foods that it has not been used in previously. Americans are consuming higher amounts of HFCS because of this. The effects of this additional consumption of HFCS need to be examined. The presence of this added sweetener could be causing weight gain simply because of the additional calories.
In summary, there is no convincing evidence that HFCS is uniquely responsible for the obesity epidemic in the US. Studies have shown that sucrose would likely have the same effects as HFCS in soft drinks. Soft drinks are a more probable culprit for the obesity epidemic because research has shown the liquid calories are not compensated for as adequately as solid calories. Therefore, the intake of sugar sweetened beverages such as soft drinks should be limited to help appease the obesity epidemic.
References:


