High fructose corn syrup, satiety, and the obesity epidemic

High fructose corn syrup (HFCS) is abundant in numerous foods that Americans consume every day. If one reads the ingredients list on virtually any processed food, he or she will see that HFCS is frequently near the top of the list. This is important because HFCS consumption can result in overeating because it signals satiety, or fullness, to the brain differently than naturally occurring carbohydrates such as sucrose and glucose. Overeating can cause weight gain, obesity, and a multitude of health conditions. In addition to the negative effects on individuals, HFCS may be contributing to the American obesity epidemic. This is significant because HFCS may be partly responsible for America's increasingly overweight and obese population.

First, HFCS is a substitute for sucrose, and both are carbohydrates. While they are similar in composition, they are different in structure. Carbohydrates are classified into categories based on the number of sugar molecules they contain. Monosaccharides are composed of one sugar molecule, disaccharides have two, and polysaccharides have many (Venes, 2001). Disaccharides and polysaccharides are broken down into monosaccharides by the body in order to be utilized. Regular table sugar is the disaccharide sucrose. Equal amounts of glucose and fructose are released when sucrose is metabolized by sucrase in the small intestine (2001). According to Wikipedia, HFCS provides the body with these same two monosaccharides: glucose and fructose. However, HFCS is structurally different from sucrose. HFCS is composed of the two individual monosaccharides glucose and fructose, while sucrose is a disaccharide composed of glucose and fructose linked together (2006). Therefore, HFCS does not have to be broken down by the body because its two component sugars are already in their simplest forms (Wikipedia,

2006). This seemingly simple distinction has many negative implications for satiety, weight, obesity, and society.

In addition, the sources of sucrose and HFCS are different. While sucrose occurs naturally, HFCS does not. Sucrose is obtained from sugar cane or sugar beet (Venes, 2001). Wikipedia notes that Japanese researchers developed the production process of HFCS in the 1970s. Then, between 1975 and 1985, the chemical was introduced into many processed foods in the United States (Wikipedia, 2006). Since that time, consumption has increased tremendously. In 2004, the average American consumed 19.2 kilograms of HFCS (Wikipedia, 2006). Lempert explains that like regular corn syrup, HFCS is derived from cornstarch (2006). As part of an extensive series of reactions, cornstarch is processed to yield glucose, which is then converted to fructose by several different enzymes (Wikipedia, 2006). This process ultimately causes the converted product to be much sweeter than the original product, which is one of its key benefits (Severson, 2004).

Next, the application of HFCS in foods is incredibly vast. The most notable and perhaps most damaging use of HFCS is in regular sodas. However, it is also used in sweet foods such as baked goods, syrups, jellies, and desserts (Severson, 2004). In addition to foods commonly expected to include a form of sweetener, bread, crackers, ketchup, pasta and barbeque sauces often contain HFCS (Lempert, 2006). HFCS is 55 percent fructose and 45 percent glucose in soda, which is the product where most of the chemical is found. However, Severson states that the ratio of the two monosaccharides can vary in different foods, and common ratios of fructose to glucose are 55:45 and 90:10 (Wikipedia, 2006). These different ratios contribute to the versatility of HFCS.

Moreover, there are both benefits and drawbacks to the sugar substitute. First, HFCS is much less expensive than sugar, which is a primary reason for its widespread use. Although the production process of HFCS is extensive, the high import tariffs on sugar cause the latter to be more expensive. For the food industry, cost is important and makes HFCS an attractive alternative to the more expensive sucrose. Next, the higher ratio of fructose to glucose results in a sweeter taste than that produced by the disaccharide sucrose. Therefore, less HFCS is needed to produce the same degree of sweetness produced by regular sugar (Severson, 2004). The lower cost and smaller quantity mean that it is much less expensive to sweeten a food with HFCS than sucrose. In addition, HFCS is liquid at room temperature, so it mixes into foods better than regular sugar (Lempert, 2006). The liquid form is also easier to transport (Wikipedia, 2006). Furthermore, it extends the shelf-life of processed food and prevents freezer burn. These features benefit both the food industry and consumers. All of these factors make HFCS an attractive alternative to sucrose. As a result, it is included in many foods as a substitute for sugar. Despite the numerous benefits, there is one principal drawback: HFCS does not send satiety signals to the brain in the same way as sucrose. As a result, one can consume an enormous amount of calories without feeling full. Clearly, this contributes to obesity and all of the health risks with which it is associated.

Additionally, HFCS is digested, absorbed, and metabolized differently than glucose. Most importantly, it does not signal satiety because insulin secretion and leptin production are not stimulated. These homeostatic hormones are adiposity signals that act on the central nervous system (CNS) to stimulate satiety**.** More specifically, these hormones act on the medial hypothalamus, which plays major role in metabolism, energy balance, and caloric intake needs. They are also thought to act on the ventral tegmental area and limbic system, which mediate

reward and motivation (Figlewicz, 2003). Leptin is synthesized and secreted primarily by adipocytes (fat cells), and its production is stimulated by insulin (Bray, 2004). Leptin reduces food intake by binding to receptors in the ventral medial hypothalamus. Once bound to leptin, the receptors produce a cascade effect to signal satiety (Wikipedia, 2006). Insulin is a hormone secreted by the beta cells in the pancreas and is transported throughout the body. Like the leptin receptors, insulin receptors are present throughout the CNS, with higher concentrations in the hypothalamus (Figlewicz, 2003). Insulin controls the ability of glucose to enter cells, specifically myocytes and adipocytes (Wikipedia, 2006). Low levels of insulin or a resistance to insulin will prevent glucose from entering cells.

As previously mentioned, HFCS is structurally different from glucose and sucrose. Therefore it is not metabolized in the same way. In fact, it is only broken down in a multi-step chemical process in the liver. Disaccharides such as sucrose are cleaved in the intestine, and the glucose produced is absorbed by a sodium-glucose transporter. In contrast, fructose is absorbed in the small intestine, and it is not absorbed by a sodium-dependent process. After absorption, glucose and fructose are either transported to the liver or passed into the general circulation. Circulating glucose stimulates insulin release from the pancreas, whereas fructose does not. This is because beta cells lack the fructose transporter (Bray, 2004). Fructose metabolism differs from that of glucose in three main ways. First, insulin controls the transport mechanism for glucose, whereas fructose is insulin independent. Second, glucose provides satiety signals to the brain by stimulating insulin and leptin production. Fructose does not provide this signal because pancreatic beta cells and neurons lack its receptor. Third, fructose provides carbons for long-chain fatty acid synthesis, which facilitates triacylglycerols more efficiently than glucose.

Because of this, fructose is thought to contribute to obesity and elevated LDL cholesterol levels (Wikipedia, 2006.)

Furthermore, the major drawback of HFCS is that its consumption is linked with obesity. Obesity is a condition in which excessive fatty tissue becomes a risk factor for certain health conditions (Wikipedia, 2006). It is measured by Body Mass Index (BMI) and waist circumference (AOA, 2005). According to the American Obesity Association, it is the second leading cause of preventable death after smoking (2005). Over-consumption of HFCS and sucrose is a major contributor to obesity. Interestingly, elevated glucose levels may lead to type II diabetes, whereas fructose has no effect on diabetics due to its very low glycemic index. Therefore, obese people may either have low concentrations of leptin or may have high concentrations while being resistant. Leptin resistance is similar to the reduced insulin sensitivity that is characteristic of type II diabetes. Obesity occurs after prolonged periods of overeating, despite the leptin and insulin signaling. The relentlessly high concentrations eventually desensitize cells that used to signal satiety (Wikipedia, 2006).

Finally, obesity is a global epidemic in developing and developed countries, especially the United States. Both environmental and genetic factors contribute to obesity. The environmental changes over the past few decades, including the widespread availability of food and reduced physical activity, have clearly contributed to the obesity epidemic (Centers for Disease Control and Prevention, 2006). Currently, researchers are constructing a human obesity gene map in an attempt to identify genes that contribute to obesity (AOA, 2005). Bray argues that the rapidity of the obesity epidemic in the United States makes environmental factors the more likely explanation. The increased consumption of HFCS may be a primary factor to the environmental link with obesity (2004). Obesity is an urgent crisis because it affects the entire

body, especially the cardiovascular, pulmonary, and muscular systems. Cancer, high blood pressure, type II diabetes, coronary heart disease, and osteoarthritis are a few examples of obesity-related medical conditions. There are 300,000 deaths nationwide each year from obesity. In addition, obesity carries annual healthcare costs of $100 billion (AOA, 2005). Clearly, the obesity epidemic involves dire consequences.

In conclusion, HFCS is a common sugar substitute present in nearly all processed foods. It is composed of the two monosaccharides glucose and fructose, and its structure and metabolism are different from that of table sugar. It has many commercial benefits, including its vast applications for use and relative inexpensiveness. However, HFCS does not signal satiety to the brain in the same way as sucrose because it does not stimulate insulin and leptin production. Therefore it has been linked to the obesity epidemic and its affiliated health problems. It is important for consumers to be aware of these implications and how they may factor into their lifestyle.

Glossary

Glucose: Monosaccharide with the chemical formula $C_6H_{12}O_6$, the major free sugar circulating in the blood of higher animals. Glucose triggers the release of insulin.

Fructose: Monosaccharide with the chemical formula $C_6H_{12}O_6$, an isomer of glucose. Fructose does not require insulin to be used by the body, but rather is absorbed by the GLUT-5 and GLUT-2 transporters.

Sucrose: Disaccharide composed of glucose and fructose with the chemical formula $C_{12}H_{22}O_{11}$, table sugar. Sucrose is hydrolyzed into a 50:50 mixture of glucose and fructose by the enzyme sucrase.

High fructose corn syrup (HFCS): Mixture of the two individual monosaccharides glucose and fructose in varying proportions, much sweeter than sucrose. It may be contributing to the American obesity epidemic because of its different metabolic pathway.

Sucrase: Enzyme that catalyzes the hydrolysis of sucrose into its component monosaccharides, glucose and fructose.

Insulin: A hormone that regulates carbohydrate metabolism. It is produced by the pancreatic beta cells, and its release is stimulated when blood glucose levels are elevated. Insulin serves as a transporter to move glucose into cells. It also promotes the release of leptin. Both of these hormones act to signal satiety; neither is stimulated by fructose.

Leptin: A hormone that regulates energy intake and expenditure (i.e. satiety) by binding to receptors in the CNS. It is produced by adipocytes. HFCS does not stimulate leptin production like glucose does.

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