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How Does the Consumption of Fructose and High Fructose Corn Syrup Impact the Health of Children and Adolescents?¹

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How does the consumption of fructose and high fructose corn syrup (HFCS) impact the health of children and adolescents? The media has lead consumers to believe that HFCS is detrimental to health and has aggravated obesity. The rates of obesity in school age children and adolescents have tripled in the last 30 years, but are fructose and HFCS contributing to the problem (http://www.CDC.gov, 2009)?

In the early 1980s, HFCS replaced sucrose, or table sugar, in most sweetened beverages. This coincided with the rise in obesity. Today, average daily consumption of fructose, largely as a component of HFCS, in the United States is 54.7 g or approximately 13 teaspoons of table sugar per person per day (Vos, Kimmons, Gillespie, Welsh, Michels Blanck, 2008). Consumption of fructose is significantly higher among adolescents at 72.8 g per day (Federal Interagency Forum on Child and Family Statistics, 2009; Vos et al., 2008).

To better understand the issue, it is important to understand the nature of sugars. There are two main categories of sugars—monosaccharides and disaccharides. Monosaccharides are the most basic form of sugars and include glucose, fructose, and galactose. Disaccharides consist of two monosaccharides held together by a glycosidic bond (Schorin, 2004). Sucrose, or table sugar, is composed of fructose and glucose in a 50:50 ratio. HFCS is a synthetic monosaccharide, also created from fructose and glucose, but in varying ratios based on intended use. Fructose, a core component of sucrose and HFCS, occurs naturally in fruits and vegetables; however, the majority of fructose consumption today is through sweetened beverages and packaged foods. Fructose is absorbed in the small intestine by diffusion via glucose transporter, excreted by the liver, and has minimal impact on insulin secretion. The glycemic index (GI), or how carbohydrates affect blood glucose levels, of fructose is low at 19% (Melanson, 2008). Once in the liver, fructose is rapidly converted to fructose-1-phosohate and bypasses the early, rate-limiting steps of glucose metabolism (Bantle, 2009; Murphy, 2009).

The process of fructose metabolism can lead to the build up of uric acid, which can cause cardiovascular disease including hypertension (Brown, Dulloo, & Montani, 2008). Excessive fructose intake may also cause lactate production (Tappy & Le, 2010). Large quantities of fructose have also been shown to increase postprandial triglyceride levels (Angelopoulos et al., 2009). In 2008, Livesey and Taylor published a meta-analysis evaluating fructose intake, blood glucose, and health markers specifically triglyceride levels. They found that an effect on postprandial triglycerides was not noted unless greater than 50 g fructose per day was consumed (Livesey & Taylor, 2008). Although fructose is usually consumed in liquid form, crystalline fructose can be purchased from health food stores to be used as a substitute for table sugar or diet sweeteners (White, 2009). Pediatric nurses should educate children and families about the various forms of fructose and the need to limit the amount consumed so that adverse effects will not occur.

HFCS is a synthetic monosaccharide. Varying percentages of fructose are contained in HFCS; the concentrations include 90%, 55%, and 42%. HFCS 55 (55% fructose and 45% glucose) is most similar in taste and sweetness to sucrose and is the most common form of the syrup used in sweetened beverages. HFCS is created using an isomerase technology, where the enzyme technology converts cornstarch into corn syrup, then into fructose. The GI of sucrose

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is 68%, and the GI of HFCS 55 is 63% demonstrating how similarly the sugars impact the body (Melanson, 2008).

No long-term side effects of the ingestion of HFCS have been reported. Dufault et al. (2009) found trace amounts of mercury contamination in half of the manufacturers of HFCS (Dufault et al., 2009). This may pose a significant problem in the pediatric population. Because children are ingesting large volumes of HFCS in beverages and packaged foods, they may be consuming more than trace amounts of mercury. HFCS has been approved by the Food and Drug Administration to be generally recognized as safe and therefore requires no formal investigation because of its similarity to sucrose. However, it is unknown if the synthetic composition of HFCS will impact the health of pediatric patients (Schorin, 2004).

Sugars are best consumed in their natural state. Fructose consumed in fresh fruit stimulates less insulin release than when ingested in a beverage. Fiber slows the absorption of the sugar. Fresh fruit also provides children and adolescents with essential vitamins and minerals that are not available from beverages or processed foods. Obesity is a major public health problem in the pediatric population. Education regarding healthy choices and exercise is vital to the long term heath of our children (Libuda & Kersting, 2009). Although there is no direct correlation linking HFCS as the cause of the obesity epidemic, it is clear that the excessive caloric intake from fructose and HFCS contributes to obesity.

Fructose bypasses many of the body's satiating signals, which can lead to over consumption and excessive calorie intake. Moran (2009) reported that gastric emptying of fructose is more rapid than glucose. Rapid gastric emptying may cause diarrhea and flatulence (White, 2009). Ghrelin is the hormone that stimulates hunger, and leptin is a hormone that helps to regulate appetite and metabolism. Teff et al. (2004) found that studies comparing the consumption of high-glucose to high-fructose beverages demonstrated that high-fructose beverages stimulate lower levels of leptin. In addition, the ingestion of fructose does not suppress Ghrelin, suggesting that it does not satisfy hunger to the same degree as glucose (Teff et al., 2004). These physiologic and hormonal responses demonstrate that fructose has less potential than glucose to decrease satiety. This can be aggravated with long-term fructose consumption.

Bray (2008) analyzed five studies that demonstrated a positive association between soft drink consumption and weight gain. In addition, interventions directly limiting sugar-containing beverages have been shown to improve body mass index in a group of adolescents (Ebbeling et al., 2006). Limiting sweetened beverages in pediatric patients will decrease caloric consumption, thus helping to prevent obesity. Pediatric nurses must provide education that sweetened beverages should be consumed in moderation.

References

- Angelopoulos, T. J., Lownder, J., Zukley, L., Melanson, K. J., Nguyen, V., Huffman, A., & Ripple, J. M. (2009). The effect of high-fructose corn syrup consumption on triglycerides and uric acid. *The Journal of Nutrition*, 139, 1242S–1245S.
- Bantle, J. P. (2009). Dietary fructose and metabolic syndrome and diabetes. *The Journal of Nutrition*, 139, 1263S–1268S.
- Bray, G. A. (2008). Fructose should we worry. *International Journal of Obesity*, 32, s127–s131.
- Brown, C. M., Dulloo, A. G., & Montani, J. -P. (2008). Sugary drinks in the pathogenesis of obesity and cardiovascular diseases. *International Journal of Obesity*, 32, S28–S34.
- Dufault, R., LeBlanc, B., Schnoll, R., Cornett, C., Schweitzer, L., Wallinga, D., Hightower, J., Patrick, L., & Lukiw, W. J. (2009). Mercury from chlor-alkali plants: Measured in food product sugar. *Environmental Health*, 8, 1–6, doi:10.1186/1476-069X-8-2.
- Ebbeling, C. B., Feldman, H. A., Osganian, S. K., Chromitz, V. R., Ellenbogen, S. J., & Ludwig, D. S. (2006). Effects of decreased sugar— Sweetened beverage consumption on body weight in adolescents: A randomized, controlled poplit study. *Pediatrics*, 117, 673–680, doi: 10.1542/peds.2005-0983.
- Federal Interagency Forum on Child and Family Statistics. (2009). America's Children: Key National Indicators of Well-Being. Washington, DC: U.S. Government Printing http://www.cdc.gov/Features/ dsOverweightChildren.
- Libuda, L., & Kersting, M. (2009). Soft drink and body weight development in childhood: Is there a relationship? *Current Opinion in Clinical Nutrition and Metabolic Care*, 12, 596–600.
- Melanson, K. J. (2008). High-fructose corn syrup, energy intake, and appetite regulation. *American Journal of Clinical Nutrition*, 88, 1738S-1744S.
- Moran, T. H. (2009). Fructose and satiety. *The Journal of Nutrition*, 139, 1253S–1256S.
- Murphy, M. P. (2009). The state of the science on the dietary sweeteners containing fructose: Summary and issues to be resolved. *Journal of Nutrition*, 139, 1269S–1270S.
- Schorin, M. D. (2004). High fructose corn syrups, part 1. Nutrition Today, 40, 248–252.
- Tappy, L., & Le, K. A. (2010). Metabolic effects of fructose and the worldwide increase in obesity. *Physiological Reviews*, 90, 23–46, doi: 10.1152/physrev.000192009.
- Teff, K. L., Elliott, S. S., Tcshop, M., Kieffer, T. J., Rader, D., Hieman, M., Townsend, R. R., Kiem, N. L., D'Alessio, D., & havel, P. J. (2004). Dietary fructose reduces circulating insulin and leptin, attenuates postprandial suppression of ghrelin, and increases triglycerides in women. *The Journal of Clinical Endocrinology and Metabolism*, 89, 2963–2972.
- White, J. S. (2009). Misconceptions about hight-fructose corn syrup: Is it uniquely responsible for obesity, reactive dicarbonyl compounds, and advances glycation end products? *Journal of Nutrition*, 139, 1219S–1227S.
- Vos, M. B., Kimmons, J. E., Gillespie, C., Welsh, J., & Michels Blanck, H. (2008). Dietary fructose consumption among US children and adults; The Third National Health and Nutrition Examination Survey. *Medscape Journal of Medicine*, 10, 160.