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MAYBE NOT SO SWEET, AFTER ALL…
Not all sugars are equal, at least when it comes to weight gain and health

Philadelphia, PA -- Researchers at the Monell Chemical Senses Center, the University of California, Davis and other collaborating colleagues report that drinking beverages containing fructose, a naturally-occurring sugar commonly used to sweeten soft drinks and other beverages, induces a pattern of hormonal responses that may favor the development of obesity.

It is estimated that consumption of fructose has increased by 20-30% over the past three decades, a rate of increase similar to that of obesity, which has risen dramatically over the same time span. Data from the present study suggest a mechanism by which fructose consumption could be one factor contributing to the increased incidence of obesity.

In the study, reported in the June 4 issue of the Journal of Clinical Endocrinology and Metabolism, 12 normal-weight women ate standardized meals on two days. The meals contained the same number of calories and the same distribution of total carbohydrate, fat and protein. On one day the meals included a beverage sweetened with fructose. On the other day, the same beverage was sweetened with an equal amount of glucose, another naturally-occurring sugar that is used by the body for energy.

Following meals accompanied by the fructose-sweetened beverage, circulating levels of insulin and leptin were decreased compared to when the women ate the same meals accompanied by the glucose-sweetened beverage. Lower levels of insulin and leptin, hormones that convey information to the brain about the body's energy status and fat stores, have been linked in other studies to increased appetite and obesity.

In addition, levels of ghrelin, a hormone thought to trigger appetite that normally declines following a meal, decreased less after meals on the day the women drank the fructose-sweetened beverage. And, the fructose also resulted in a long-lasting increase of triglycerides, fatty molecules in the blood that are indicators of risk for cardiovascular disease.

Together, the hormonal responses observed after drinking beverages sweetened with fructose suggest that prolonged consumption of diets high in energy from fructose could lead to increased caloric intake and contribute to weight gain and obesity. Lead author Karen Teff, Ph.D., a physiologist at Monell, comments, "Fructose consumption results in a metabolic profile of hormones which would be predicted to increase food intake, thereby contributing to obesity in susceptible

populations." Teff notes that this pattern of hormonal responses is similar to that observed after consuming a high-fat meal, and continues, "Based on our previously published work, this metabolic profile resembles that of fat consumption. Thus, despite the fact that fructose is a sugar, metabolically the responses are similar to those seen following fat ingestion." The elevated levels of plasma triglycerides observed after fructose consumption further suggest that frequent fructose consumption could also contribute to the development of atherosclerosis and cardiovascular disease.

According to co-author Dr. Peter Havel, a research endocrinologist at the University of California, Davis, "Although this short-term experiment provides important new data, additional research is needed to investigate the long-term impact of consuming fructose in humans, particularly its effects on lipid metabolism and on endocrine signals involved in body weight regulation. New studies should also be conducted in subjects who are at increased risk for metabolic diseases such as type-2 diabetes and cardiovascular disease and who may be more susceptible to the adverse effects of overconsuming fructose".

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Citation: Karen L. Teff, Sharon S. Elliott, Matthias Tschoep, Timothy J. Kieffer, Daniel Rader, Mark Heiman , Raymond R. Townsend , Nancy L. Keim , David D'Alessio and Peter J. Havel. Dietary Fructose Reduces Circulating Insulin and Leptin, Attenuates Postprandial Suppression of Ghrelin and Increases Triglycerides in Women. *Journal of Clinical Endocrinology and Metabolism*, 2004, 89, 2963-2972.

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