Sweetened Beverages and Health: Current State of Scientific Understandings[1,2]

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ABSTRACT

This article summarizes the presentations from the "Sweetened Beverages and Health: Current State of Scientific Understandings" symposium held at the ASN Annual Meeting in Boston, MA on April 23, 2013. The metabolic and health effects of sugar-sweetened beverages were discussed from a variety of points of view by 5 different presenters. Dr. David Allison drew a distinction between conjecture and proof related to sweetened beverages and obesity. Dr. Richard Mattes discussed differences between solid and liquid calories. Dr. Miguel Alonso-Alonso reviewed potential contributions of functional neuroimaging, particularly as they relate to whether sugar is potentially "addictive." Dr. Kimber Stanhope discussed work related to experiments comparing fructose to glucose. Dr. James Rippe presented evidence from randomized controlled trials from his research organization showing no differences among high-fructose corn syrup, sucrose, glucose, or fructose at normal human consumption amounts. *Adv. Nutr. 4: 527–529, 2013.*

Introduction

The metabolic and health effects of sugar-sweetened beverages (SSBs)[6] are controversial and are the subject of intense scientific debate. These potential effects span important scientific questions and also are of great interest to the public and potentially to regulatory bodies. Recent epidemiologic studies have associated the consumption of SSBs with increased risk of obesity, heart disease, hypertension, possible inflammatory responses, and decreased dietary quality. Based on these considerations, the AHA has recommended limiting consumption of added sugars to no more than 150 kcal/d for the average adult man and 100 kcal/d for the average adult woman. These recommendations are substantially different and more restrictive from those of the Institute of Medicine and the Dietary Guidelines for Americans, which establish a safe upper limit of consumption of up to 25% of energy in the human diet.

The debate among scientists and the public is further complicated by confusion over the terms "fructose," "high-fructose corn syrup (HFCS)," and "sucrose." Scientific experiments conducted by investigators that compare pure fructose to pure glucose have further clouded the issue, because neither is consumed to any appreciable degree in isolation in the human diet. Although there is broad scientific consensus on the metabolic equivalence of HFCS and sucrose, considerable controversy exists about whether any health hazards result from consumption of either sucrose or HFCS because of the fructose moiety found in both of these sugars.

Several recent papers have utilized functional MRI (fMRI) findings to suggest that fructose stimulates hypothalamic blood flow responses differently than glucose and may also stimulate reward pathways differently. Several scientists and members of the media have suggested that these differences may lead to a form of sugar "addiction" and overeating, which could contribute to the modern pandemic of obesity. Very little data are available on brain responses to commonly consumed sugars such as sucrose or HFCS.

The purpose of this symposium was to explore current and emerging scientific understandings of SSBs with an emphasis on both what is known and what is not known.

[1] This article is a summary of the symposium "Sweetened Beverages and Health: Current State of Scientific Understandings" held April 23, 2013 at the American Society for Nutrition Scientific Sessions and Annual Meeting at Experimental Biology 2013 in Boston, MA. The symposium was sponsored by the ASN Medical Nutrition Council and supported in part by an educational grant from the Corn Refiners Association. The organizer has indicated that related reviews of this symposium will be submitted for publication in an upcoming issue of *Advances of Nutrition*.

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[6] Abbreviations used: HFCS, high-fructose corn syrup; fMRI, functional MRI; SSB, sugar-sweetened beverage.

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The symposium's first speaker was Dr. David Allison, Distinguished Professor and Quetelet Endowed Professor of Public Health at the University of Alabama at Birmingham. Dr. Allison discussed available evidence to support the conjecture that SSBs might be associated with obesity but not showing that this proposition was true.

Dr. Allison provided 6 different reasons why there might be confusion related to whether sweetened beverages cause obesity. These include:

- The use of causal language in observational studies of obesity and nutrition when these studies establish association rather than cause and effect.
- Emotion-raising language, including such phrases that SSBs represent a "threat to global health," the "toxic" truth about sugar, or fructose as "pure, white and deadly."
- The perils of ignoring history such as comparing tobacco to sugar.
- Exaggerations and distortions in press releases and public statements.
- Reporting bias, where the reporting of the study is inconsistent with the trial registration.
- Citation bias, where subsequent papers citing published work exaggerate observed effects.

Dr. Allison concluded that although though there was a sound basis for conjecturing that increased consumption of SSBs might be associated with weight gain or that reducing SSBs could reduce weight, the actual studies in these areas provide only equivocal effects.

The second speaker, Dr. Richard Mattes, Professor in the Department of Foods and Nutrition at Purdue University, discussed issues related to solid compared with liquid calories and their effects on appetite. Dr. Mattes discussed the common assumption that a "calorie is a calorie" by comparing ingestive processes of beverages with solid foods. Ingestive processes such as cognition, orosensory factors, gastric emptying, intestinal transit, rate of absorption, and endocrine response show differences when comparing beverages to solid calories. Moreover, general properties, including such issues as hunger and thirst, distinguish between beverages and solid foods. Dr. Mattes concluded that consumption amounts of beverages was more consistent with weight trends than consumption of solid calories; that beverages evoke weaker appetitive and compensatory responses than do solids; and that beverages contribute to snacking and are associated with increased food energy, possibly due to increased energy density. He concluded that obese individuals may be at the greatest risk for adverse consequences due to high intake of fluid calories and may stand to realize the greatest benefits from moderating intake.

The third speaker, Dr. Miguel Alonso-Alonso, neurologist and Instructor in Neurology at Harvard Medical School, spoke about the emerging understandings from functional neuroimaging with particular reference to the issue of whether sweetened beverages might be addictive. Dr. Alonso provided insight into how the brain perceives food as a goal, reward, and as a needed fuel for the body. The issue of whether the brain's perception of reward, such as responding to pleasure, motivation, reinforcement and learning, leads to addiction was discussed. Dr. Alonso evaluated the evidence supporting the food addiction model of obesity and concluded that the evidence is insufficient to make a broad connection and the model fits only in limited contexts (e.g., binge eating disorder).

Dr. Alonso also discussed how human neuroimaging might help in this area and concluded that fMRI studies could play a role in understanding neuromechanisms but on its own was not useful to the diagnosis of addiction. Dr. Alonso concluded with a review of recent fMRI studies in the area of consumption of various foods and the neuroresponse to them. Included in this discussion was a recently conducted pilot study involving his team at Harvard in collaboration with investigators at Rippe Lifestyle Institute looking at the effects of various sugars on the brain. Pilot data from this study suggested no differences among HFCS, sucrose, fructose, and glucose in terms of their effects on hypothalamic blood flow. Dr. Alonso concluded that there were limited and insufficient data in humans on the links between food/obesity and addiction and that the model fits well only in a subset of cases, such as obesity with binge eating disorder. The contribution of specific foods (e.g., sweetened beverages) is largely unexplored and deserves further research.

The fourth speaker, Dr. Kimber Stanhope, Associate Project Scientist, Department of Molecular Biosciences at the School of Veterinary Medicine at the University of California Davis, discussed work from her laboratory and others related to comparisons of fructose to glucose on various metabolic parameters and health considerations. She provided data that fructose is metabolized differently than glucose in the human body and may lead to accumulation of fat in the liver, insulin resistance, abdominal weight gain, and increased risk factors for metabolic syndrome, heart disease, and diabetes. She acknowledged that much fewer data are available in the area of HFCS and sucrose, although she cited a recently published study suggesting that sucrose, when consumed in SSBs, increased liver fat and ectopic deposition of fat in muscles compared with milk, diet soft drinks, and water. She concluded that while limited data are available to support the more restrictive guidelines from the AHA compared with those of the Dietary Guidelines for Americans in the area of the health effects of added sugar consumption, she still believes that the AHA Guidelines are more appropriate.

The fifth speaker at the symposium, Dr. James Rippe, cardiologist and Professor of Biomedical Sciences at the University of Central Florida, presented evidence from recent randomized controlled trials from his research organization comparing HFCS to sucrose at amounts up to the 90th percent population levels for fructose as well as these 2 sugars compared with pure glucose and pure fructose at the 50th percent of population. These studies have demonstrated that by every variable measured thus far in humans, HFCS and sucrose are metabolically equivalent. He also presented

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evidence that studies comparing pure fructose to pure glucose do not accurately represent human nutrition, because neither of these substances is consumed to any appreciable degree by itself in the human diet.

Dr. Rippe presented evidence from randomized controlled trials showing no unique relationship between HFCS and obesity and no adverse effect on lipids from HFCS, sucrose, glucose, or fructose except for a modest increase in TGs driven largely through greater sugar consumption. He presented further evidence that there were no differences between fructose-containing sugars and glucose at average population consumption amounts of fructose and no increase in fatty infiltration of the liver at consumption amounts up to the 90th percentile of population consumption for fructose. Dr. Rippe concluded with data from a pilot study that suggested that there were no differences in hypothalamic or cerebral blood flow between fructose-containing sugars and glucose but cautioned that larger studies are required to find more definitive proof in this area.

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