

Fructose, Sucrose, and High Fructose Corn Syrup: Modern Scientific Findings and Health Implications^{1,2}

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Added sugars have increased in diets in the United States and many other industrialized countries over the past 30 y. Controversies have arisen, because some investigators have suggested that sugars, in general, and sugar-sweetened beverages, in particular, may be associated with increased risk of obesity, metabolic syndrome, heart disease, and other serious health conditions. Many of these arguments have been based on epidemiologic studies, randomized clinical trials, and theoretical constructs.

Results of randomized clinical trials have been inconsistent. The Dietary Guidelines for Americans and the AHA have published similar recommendations for added sugars. The debate among the public is complicated by confusion over the terms “fructose,” “high fructose corn syrup (HFCS),” and “sucrose,” with many people believing that there are metabolic differences between HFCS and sucrose. This confusion persists despite the fact that both the American Medical Association and the Academy of Nutrition and Dietetics have issued statements reporting that both of these sugars are essentially equivalent. Furthermore, there is a broad scientific consensus on the metabolic equivalence of HFCS and sucrose.

With this as a backdrop, 5 leading experts in the metabolism and health effects of fructose, HFCS, and sucrose gathered for this symposium to present data and discuss and debate relevant scientific findings related to the metabolism and health effects of these sugars.

The symposium’s first speaker was John White, a sugar biochemist and Founder and President of White Technical

Research. Dr. White provided detailed evidence concerning the metabolic equivalence of HFCS and sucrose. He reminded the audience that fructose is rarely consumed by itself in the human diet. It is virtually always consumed in combination with glucose, whether it is in carbonated soft drinks, fruit juices, or many fruits and vegetables. Dr. White presented a detailed discussion concerning the biochemistry and metabolism of sucrose and HFCS and contrasted this with studies done on pure fructose compared with glucose, often delivered in dosages above normal population consumption levels. Dr. White provided data demonstrating that the amount of glucose in the human diet from all sources far exceeds the amount of fructose from all sources. He concluded that fructose is not increasing in the human diet and that good cause and effect evidence does not exist that uniquely links the metabolism of fructose from normally consumed sugars at typical dosages to a variety of adverse health conditions.

The second speaker, Dr. George Bray, Chief of the Division of Clinical Obesity and Metabolism at the Pennington Biomedical Research Center, presented data documenting that sugar consumption has risen dramatically in the United States in the last 100 y and particularly in the last 30 y. He raised a special concern about soft drink consumption, which has also risen dramatically during this period and is the leading source of fructose in the American diet delivered either through sucrose or HFCS used to sweeten beverages.

Dr. Bray provided a summary of epidemiologic evidence showing an association with soft drink intake and obesity, metabolic syndrome, gout, and fatty liver disease. He then reviewed recent studies that suggest that soft drink calories are less satiating than calories from solid foods. Dr. Bray concluded by reviewing 3 randomized controlled trials of sugar-sweetened beverages. One 10-wk trial demonstrated weight gain in the group consuming sucrose-sweetened beverages compared with Aspartame-sweetened beverages. The second study demonstrated weight gain and increased blood pressure in sucrose-sweetened beverages compared with Aspartame-sweetened beverages. Dr. Bray noted that trials comparing fructose to glucose showed increases in both

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TG and de novo lipogenesis in the fructose arm. Dr. Bray reviewed findings from a recent study that compared sugar-sweetened beverages to diet beverages, milk, and water that reported increased visceral fat and increased fat deposition in liver and muscle.

The third speaker, Dr. Robert Lustig, Professor of Clinical Pediatrics at the University of California, San Francisco, presented evidence supporting his theory that sugar at high levels may be “toxic” and is metabolized in the liver in ways that are similar to ethanol and different from glucose. Dr. Lustig depicted detailed biochemical pathways to support his assertion that fructose may be preferentially metabolized into fat by the liver, thereby increasing the risk of heart disease, obesity, type 2 diabetes, nonalcoholic fatty liver disease, and hypertension. Dr. Lustig also presented animal data in support of his theory that sugar is “addictive.”

The fourth 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 comparing HFCS to sucrose at levels up to the 90th percentile of population consumption for fructose. These studies demonstrated that by every parameter measured thus far in humans, HFCS and sucrose are metabolically equivalent. He also presented evidence that studies comparing pure fructose to pure glucose do not accurately represent human nutrition, because neither of these substances is consumed by itself to any appreciable degree in the human diet. Dr. Rippe presented evidence from randomized clinical trials that consumption of neither HFCS nor sucrose led to increases in blood pressure, dyslipidemia (with the exception of slight

increases in TG), obesity, metabolic syndrome, nonalcoholic fatty liver disease, or ectopic fat deposition in muscles.

Dr. Rippe challenged Dr. Lustig’s and Dr. Bray’s interpretation of de novo lipogenesis and provided data from a variety of researchers demonstrating that de novo lipogenesis amounted to a minor pathway in the overall human energy economy (on the order of 1–5% of fat generated compared with the 100–125 g of fat consumed). Dr. Rippe concluded that despite widespread scientific agreement on many issues such as the equivalence between HFCS and sucrose, the public remains very confused on these issues. He called for further randomized clinical trials to resolve the remaining controversies.

The final speaker, Dr. David Klurfeld, National Program Leader, Human Nutrition, USDA-Agricultural Research Service, provided an overview of what government agencies consider in the debate over added sugars. He noted that federal agencies rely heavily on evidence-based guidelines such as the Dietary Guidelines for Americans and RDA from the Institute of Medicine when developing nutrition policy. He noted that current levels of added sugar consumption are within the guidelines from the Dietary Guidelines for Americans and the Institute of Medicine. Dr. Klurfeld concluded that portion size and overall caloric consumption were more likely causes of obesity than simply blaming sugar-sweetened beverages. He stated that the claim that sugar is “toxic” does not pass the test of face validity and that although Americans consume too much sugar, it is only one factor in a poor dietary pattern. He noted that regulating or taxing sugars is a political decision and that insufficient nutritional data exist to justify such a decision.