

Fructose study fails to show real world significance

John S White (2 June 2009)

We are writing to express concern that the conclusion of Havel and coworkers (1) in their manuscript titled, "Endocrine and Metabolic Effects of Consuming Fructose- and Glucose-Sweetened Beverages with Meals in Obese Men and Women: Influence of Insulin Resistance on Plasma Triglyceride Responses" is not relevant to real world human diets. The authors concluded that "over consumption of dietary fructose may exacerbate the adverse metabolic profiles in obese individuals, particularly those with existing insulin resistance and may therefore increase the risks for developing diabetes and cardiovascular disease"; but their experimental design, in which 30% of calories are fed as fructose in comparison to glucose, was highly artificial. Virtually no one consumes a diet in which 30% of calories—literally all their simple sugars—are composed entirely of a single sugar (glucose or fructose) in a beverage. The ratio of fructose:glucose in the contemporary human diet was recently estimated at 0.79 by Forshee et al. (2) and has remained unchanged since 1966. The introduction of high fructose corn syrup (HFCS) did not change the fructose:glucose ratio because a) HFCS and sucrose have the same composition and b) HFCS displaced sucrose in foods and beverages on a nearly one-for-one basis.

In the context of commonly used nutritive (caloric) sweeteners— which are half fructose and half glucose—30% of calories as fructose would require the intake of 60% of calories as sucrose, i.e., table sugar, HFCS, honey or fruit juice concentrates. With such exaggerated intakes, Havel et al. have inappropriately shifted the discussion from food safety to toxicology. It is possible the authors are of metabolic changes induced under conditions of extreme fructose loading not present at normal exposure levels.

The authors justify their study design by citing the "dramatic increase in [fructose] consumption over the past four decades" and the parallel between increased consumption and increased incidence of obesity in the U.S.; but this thesis is substantially weakened by consideration of the omitted perspective that all caloric macronutrients have increased similarly over the same time period and that each equally well parallels the increased incidence of obesity (3).

It would be ill advised to base predictions of real world medical outcomes, formulate dietary advice or articulate public health policy on experiments with exposures so highly distorted from actual consumption patterns of fructose.

References

1. Teff KL, Grudziak J, Townsend RR, Dunn TN, Grant RW, Adams SH, Keim NL, Cummings BP, Stanhope KL, Havel PJ 2009 Endocrine and metabolic effects of consuming fructose- and glucose-sweetened beverages with meals in obese men and women: influence of insulin resistance on plasma triglyceride responses. *J Clin Endocrinol Metab* 94:1562-1569
2. Forshee RA, Storey ML, Allison DB, Glinsmann WH, Hein GL, Lineback DR, Miller SA, Nicklas TA, Weaver GA, White JS 2007 A critical examination of the evidence relating high fructose corn syrup and weight gain. *Crit Rev Food Sci Nutr* 47:561-582
3. White JS 2008 Straight talk about high-fructose corn syrup: what it is and what it ain't. *Am J Clin Nutr* 88:1716S-1721S