

Calorie Control Council Response to Sanchez-Lozada et al. “How safe is fructose for persons with or without diabetes?”

Sanchez-Lozada LG, Le M, Segal M, Johnson RJ. Am J Clin Nutr 2008;88:1189-90.

Livesey and Taylor (1) have published a meta-analysis which proposed beneficial uses of fructose at low-to-moderate intake levels. This proposal was summarily dismissed as premature in a companion editorial by Sánchez-Lozada et al (2), in which the authors argued that a) increased consumption of fructose parallels the rise in obesity, diabetes, hypertension and kidney disease, and b) the unique metabolism of fructose is a significant cause of the metabolic syndrome. Their arguments fail to convince, however, because c) the consumption of *all* macronutrients has increased in parallel with fructose over the past three decades and d) there is no hard evidence that fructose at typical consumption levels constitutes a credible risk in humans for metabolic syndrome and the constellation of contributing conditions.

While there is no question that fructose intake has increased over the past 35 years, it has not done so disproportionately from the rest of the diet. A recent article in this journal (3) demonstrated unequivocally that consumption of added sugars—including the principal fructose-containing sweeteners, sucrose and high fructose corn syrup—increased at nearly the identical rate (23%) as total calories (24%). Indeed, most of the calorie-dense nutrient categories (added fats, flour/cereal, added sugars) increased in parallel with total calories. Clearly, caloric excess and *not* fructose is the primary cause of obesity, diabetes, hypertension and kidney disease: we eat more of *everything* today than we did 35 years ago.

Vos (4) estimated the mean consumption of fructose at 10.2% of total calories. Animal studies cited by the authors (references 4, 12, 15 in reference 2) utilize diets in which fructose constitutes up to 60+% of calories—six times the typical human exposure. While they defend the feeding of such excess on the basis of necessity when studying rodents, it is clearly inappropriate to extrapolate data gathered under such extreme diets to humans. The human studies cited (references 5-7, 11, 14 in reference 2) are unconvincing because they test atypically high fructose levels (up to three times normal human exposure), compare pure sugars in a highly artificial diet, rely on unreliable dietary recall tests or utilize insufficient numbers of subjects. The comparison of fructose vs. glucose control is inappropriate, since added sugars are neither pure fructose nor pure glucose; rather, they are a combination of both at the fructose:glucose ratio of 0.79 (5).

Finally, there is no evidence that specifically limiting fructose in the diet—in essence, restricting the use of all fructose-glucose nutritive sweeteners including sucrose, high fructose corn syrup (HFCS), honey, crystalline fructose and fruit juice concentrates—would be superior to restricting calorie intake generally from *all* nutrient groups. Recent reviews by Grundy (6) and Cornier et al (7) emphasize lifestyle modification, weight loss via reduction of *total* calories and increased physical activity as the most effective keys to controlling metabolic syndrome. For perspective, neither review singled out added sugars or fructose as unique risk factors for metabolic syndrome. Riccardi and Rivellese (8) proposed a diet for treatment of the metabolic syndrome which limits intake of saturated fat, permits moderate amounts of monounsaturated fat and allows use of high fiber/low glycemic index (GI) foods

without specific limitations. While the latter allowance is obviously aimed at encouraging the consumption of high fiber carbohydrates, it should be acknowledged that among the commonly used nutritive sweeteners, fructose (GI = 20) has the lowest glycemic index, while glucose (GI = 100) has the highest (9).

Clearly, there is no evidence to warrant the blanket discouragement given to Livesey and Taylor, who have argued reasonably for a re-evaluation of specific benefits of fructose at low-to-moderate intake levels. Is it not curious that with all the research interest in the metabolic effects of fructose, dose-response experiments at typical intake levels and typical dietary fructose:glucose ratios have been so assiduously avoided? It is time, as the authors suggest, to conduct these experiments and finally lay to rest the notion that fructose is a significant cause of the metabolic syndrome and contributing conditions.

Literature Cited

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