

Weak Association between Sweeteners or Sweetened Beverages and Diabetes

Dear Editor,

The recent article by Montonen et al. (1) fails to demonstrate that a higher intake of fructose, glucose, and sweetened beverages (but not sucrose) may increase type 2 diabetes risk because 1) the authors do not acknowledge the fundamental compositional and metabolic similarities between monosaccharide (fructose + glucose) and disaccharide sucrose, and 2) their conclusion regarding diabetes risk of sweetened beverages vs. sucrose is inconsistent with the dominant use of sucrose in caloric beverages outside the United States and in Finland.

First, it is fundamentally important to recognize that fructose and glucose are present in approximately the same proportions (1:1) in all major nutritive sweeteners: sucrose, high-fructose corn syrup (HFCS), honey, and fruit juice concentrates. True, sucrose is a disaccharide with a covalent bond joining fructose and glucose whereas the other sweeteners contain the two as monosaccharides; however, the covalent bond of sucrose is rapidly hydrolyzed in the lumen of the small intestine prior to absorption (2,3). With this in mind, it is difficult to understand how the authors could postulate a differential effect for monosaccharides (fructose + glucose) vs. disaccharides [fructose + glucose (sucrose)], when fructose and glucose from both sources reach the portal blood as monosaccharides immediately after absorption. That is, both sources present the same monosaccharide sugars in the same ratios in the same concentrations within the same time frame to the same tissues.

Second, it is simply not plausible for the authors to conclude that the risk of type 2 diabetes is increased by higher intake of caloric beverages but not higher intake of sucrose. Although most caloric beverages in the United States use HFCS, this is not the case elsewhere: we live in an overwhelmingly sucrose-sweetened world. Fereday et al. (4) estimated 2007 HFCS production worldwide at 8% of the total (sucrose + HFCS) and HFCS production in the European Union (of which Finland is a member) at just 3% of the total. It is thus reasonable to conclude that the majority of caloric beverages produced worldwide use sucrose, including those produced in Finland, where the data analyzed by Montonen et al. [17,18,23–25 in (1)] were generated. Clearly, a sucrose-sweetened caloric beverage cannot pose an increased risk for type 2 diabetes without sucrose posing the same risk. Furthermore, published correlations with caloric soft drink consumption are tenuous at best: recent reviews found inconsistent or no correlation with overweight, obesity, or BMI (5–8). Because all are aggravating conditions for diabetes, there is strong reason to expect there would likewise be little correlation between caloric soft drink consumption and type 2 diabetes.

It is not surprising the authors found it “impossible to differentiate...potential associations (of fructose and glucose) with risk of diabetes due to the high correlation between their intakes.” Forshee et al. (5) recently estimated the fructose:glucose ratio in the whole diet at 0.78, essentially unchanged since the introduc-

tion of HFCS 35 y ago. Glucose is more abundant than fructose because it is available from such diverse dietary sources as fruits and vegetables; cereal grains; ingredient starches, maltodextrins, corn (glucose) syrups, liquid and dry glucose; and the common caloric sweeteners: sucrose, HFCS, honey, and fruit juice concentrates. The exceeding unlikelihood of consuming a meal, or even a snack, containing one sugar without the other clearly calls into question the dietary relevance of experiments that depend on high concentrations of fructose in glucose-free diets to create metabolic upsets [22,31,37–39,45,46,73,117,118 in (9)].

The hypothesis that fructose, HFCS, and caloric beverages play a unique role in obesity and type 2 diabetes beyond their inherent energy contributions has generated tremendous attention from scientists and the media, but no credible scientific support. The failure of Montonen et al. to demonstrate that higher intake of fructose, glucose, and sweetened beverages, but not sucrose, may increase type 2 diabetes risk offers nothing new in support of this hypothesis.

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