

Stephan BC, Wells JC, Brayne C, Albanese E, Siervo M. Increased fructose intake as a risk factor for dementia. *J Gerontol A Biol Sci Med Sci*. 2010.

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In a recent paper, Stephan et al.¹ speculated that high fructose intake is a risk factor for dementia and that increasing consumption of fructose in the U.S. population could lead to greater dementia risk. Their premise is weakened, however, by outdated references to fructose intake and functional properties, and by a reliance on supporting evidence gathered under extreme experimental conditions unrelated to typical human fructose exposure.

First, the cited secular trend in sweetener intake is outdated. The most recent U.S. Department of Agriculture loss adjusted availability data reveal that per capita intake of high fructose corn syrup (HFCS) and all caloric sweeteners — that is, all added sources of fructose — has been in decline since 1999.² In fact, sweetener intake was the same in 2008 (most recent data) as in 1992, nearly 20 years ago.

Second, energy contribution from added sugars—and fructose—over the past 40 years (since the introduction of HFCS) relative to other dietary nutrients is far lower than the authors believe. While total energy intake increased 515 kcal/d (24%) between 1970 and 2008,³ energy from added sugars (including honey, sucrose, high fructose corn syrup and fruit juice concentrates) increased only 58 kcal/d; and energy from fructose accounted for no more than half this amount. For perspective, energy from flour/cereal products and added fats increased by 185 and 235 kcal/d, respectively. It is highly unlikely, therefore, that increased consumption of sugary drinks is “mostly...accountable” for the increase in energy intake over the past four decades or that “increasing consumption of fructose...could lead to greater dementia risk,” as the authors suggest.

Third, the authors overstate the comparative sweetness of HFCS (“powerful sweetening effects, which are likely to induce more addictive consumption behaviors”) in building their case for fructose as a risk factor. While fructose is sweeter than sucrose, glucose is less sweet. HFCS-55 (55% fructose, 45% glucose) was specifically formulated with the same sweetness as sucrose to make it a suitable replacement in carbonated beverages and other applications.⁴ And it should be noted that Benton⁵ recently disproved the notion of Lenoir et al. promoted by the authors that sweeteners are addictive.

And fourth, much of the evidence cited in support of a positive association for fructose in dementia and other diseases is based on experimentation using highly exaggerated test diets. In comparison to recent 25th, 50th and 95th percentile human fructose intakes estimates of 4%, 9% and <18% of total energy,⁶ respectively, studies cited by the authors tested fructose levels in humans (author reference 24) and animals (references 3, 30, 31, 36) at 25% and 28-60% of energy, respectively; tested diets simultaneously high in both fat and fructose (references 34, 35); or allowed animals uncontrolled and unmonitored access to fructose (author references 2, 37). These experiments do not model the human fructose experience.

While it may be fashionable to promote fructose as a unique risk factor for various diseases, as Stephan et al. have done for dementia, there is little persuasive evidence in humans at typical intake levels. The authors’ description of fructose as “a potential time bomb” for dementia is hyperbole that was not substantiated by the recently released 2010 Dietary Guidelines for Americans,⁷ in which fructose significantly was not singled out from other dietary carbohydrates for special consideration or concern.

References

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