Dear Editor,

In a recent article, Stephan and colleagues (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 Their premise is weakened, however, by outdated references to fructose intake and functional properties and by a reliance on supporting evidence gathered under extreme experimental conditions unrelated to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First, the cited secular trend in sweetener intake is outdated.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. Although total energy intake increased 515 kcal/day (24%) between 1970 and 2008 (3), energy from added sugars (including honey, sucrose, HFCS, and fruit juice concentrates) increased only 58 kcal/day 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/day, respectively. It is highly unlikely, therefore, that increased consumption of sugary drinks is “mostly . . . accountable” for the increase in energy intake over the past few 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. Although fructose is sweeter than sucrose, glucose is less sweet. HFCS-55 (55% fructose and 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 Benton (5) recently disproved the notion of Lenoir and colleagues promoted by the authors (reference 11) 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 with recent 25th, 50th, and 95th percentile human fructose intake 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, and 36) at 25% and 28%–60% of energy, respectively. Fructose is rarely consumed alone but rather with equicaloric amounts of glucose either in fruits/vegetables or added sugars (eg, sucrose and HFCS). To obtain this much energy from added sugars alone would obligate 50%–100%+ of the typical daily energy intake—a highly unlikely scenario. Other evidence cited by the authors tested diets simultaneously high in both fat and fructose (40% and 20% of daily energy, respectively (author references 34 and 35) vs Institute of Medicine daily energy recommendations of 20%–35% and 12.5% (7)) or allowed animals uncontrolled and unmonitored access to fructose (author references 2 and 37). Furthermore, Coss-bu and colleagues demonstrated that fructose consumed with glucose is metabolized very differently than fructose alone (8), calling into question the usefulness of fructose alone studies in predicting human risk. These experiments do not model the human fructose experience, which commonly includes moderate fructose with comparable glucose.

Although it may be fashionable to promote fructose as a unique risk factor for various diseases, as Stephan and colleagues 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 (9) in which fructose significantly was not singled out from other dietary carbohydrates for special consideration or concern.

PROPOSED LINK BETWEEN FRUCTOSE INTAKE AND DEMENTIA RISK IS NOT PERSUASIVE

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CONFLICT OF INTEREST

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REFERENCES