Correspondence

Fructose as a significant cause of gout is unfounded and premature

Sir,
In a recent review, Suresh and Das reviewed current advances in the management of gout and highlighted fructose ingestion (in addition to alcohol and dietary purines) as an important risk factor for gout. Including fructose in the same company as alcohol and dietary purines, two well-established dietary risks for gout, was unfounded and premature for several reasons.

First, fructose intake from all sources—natural from fruits/vegetables and added from caloric sweeteners—peaked in 1999 and has been in decline for 13 years. There simply can be no positive correlation between the prevalence of gout and the declining intake of fructose among Americans.

Second, the authors cite two papers by Choi et al. in support of fructose as a risk factor for gout. One paper is a prospective cohort analysis using participants in the Health Professionals Follow-up Study, in which Choi et al. conclude that consumption of sugar sweetened soft drinks, fructose, fructose-rich fruits and fruit juices are strongly associated with an increased risk of gout. In the other, Choi et al. analyzed data from National Health and Nutrition Examination Survey (NHANES) III (1988–1994) to conclude that sugar-sweetened soft drink intake is associated with a higher level of uric acid and frequency of hyperuricemia, that orange juice intake may also be associated with a higher level of serum uric acid and that these data support strategies to reduce fructose consumption in the dietary recommendations for individuals with hyperuricemia and gout. However, both studies are severely confounded by coincidental glucose: since all dietary sources of fructose contain equal amounts of glucose, the role of glucose as a risk factor for gout cannot be excluded and the evidence for a unique fructose effect is significantly weakened. It must be acknowledged that by design, neither study is suitable to establish causation between fructose and gout.

Third, Suresh and Das display a wide tolerance in data rigor when it comes to recommending treatment options for gout. They cite head-to-head clinical trials comparing different non-steroidal anti-inflammatory drugs (NSAIDs) and human clinical trials with cytokines that establish solid causal relationships between drug intervention and relief of symptoms, but cite no such human intervention studies for fructose. The authors cite no comparable clinical data that would support a unique causal relationship between dietary fructose reduction and relief of symptoms in humans.

And, finally, two recent studies argue against fructose as a risk for gout. The systematic review and meta-analysis of controlled feeding trials by Wang et al. did not support a uric acid increasing effect of isocaloric fructose intake in non-diabetic and diabetic participants. And, an analysis of sugar intakes and health parameters from 25,506 subjects in the NHANES 1999–2006 databases by Sun et al. revealed that fructose consumption was not positively associated with indicators of metabolic syndrome, uric acid and Body Mass Index (BMI) over a wide range of intakes.

Thus, including fructose on the same list with alcohol and dietary purines as dietary risks for gout without substantive clinical proof was unfounded and premature.

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The author is a consultant and advisor to the food and beverage industry and government agencies in the area of nutritive sweeteners. Clients include research institutes, food industry councils, trade organizations and individual companies. Clients have an on-going interest in nutritive sweetener research, development, production, applications, safety and nutrition.

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References


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