

LETTER TO THE EDITOR

Fructose as cause of metabolic syndrome is poorly supported

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In a recent paper, Perez-Pozo *et al.*¹ concluded that high doses of fructose raise blood pressure and cause features of metabolic syndrome, and that fructose may therefore have a role in the current epidemics of obesity and diabetes. Their conclusion is poorly supported and should not be used to inform the public debate on fructose for three reasons.

First, the study is weakened through lack of a rigorous double-blind experimental design. That investigators and staff knew throughout the study which participants were receiving allopurinol makes it impossible to rule out the prospect of subjective bias from experimental subjects, analysts and study principals.

Second, the experimental design lacked a proper control. The authors administered fructose \pm allopurinol to human subjects to test the hypothesis that excessive fructose intake can induce the features of metabolic syndrome and that the induction is reversible. Their failure to provide an appropriate non-fructose comparison group invalidates all conclusions about fructose.

Third, the amount of dietary + supplementary fructose in experimental diets (55 g + 200 g = 34% of the total energy) is nearly quadruple the mean (9.1% of the total energy) and double the 95th percentile (<18% of the total energy) human fructose intakes recently established by Marriott *et al.*² Such macronutrient excess is atypical of the human fructose experience—characterized instead by moderate fructose intake in the presence of equivalent amounts of glucose—and further diminishes the clinical relevance of this study.

Thus, the conclusion by Perez-Pozo *et al.* that excessive fructose consumption could have a causal role in the metabolic syndrome is inadequately supported by the current work. The weak experimental design fails to demonstrate a unique role for fructose vis-à-vis other nutritive sugars, lacks a suitable control, is not double-blinded and relies on exaggerated fructose exposures. It would be inappropriate to use this study to inform the public debate on fructose.

Conflict of interest

The author is a consultant to the food and beverage industry in the area of nutritive (caloric) sweeteners. He has advised research institutes, food industry councils, trade organizations, national health associations, governmental agencies and individual companies.

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- 2 Marriott BP, Cole N, Lee E. National estimates of dietary fructose intake increased from 1977 to 2004 in the United States. *J Nutr* 2009; **139**: 1228S–1235S.