

Calorie Control Council Response to Johnson et al
"The effect of fructose on renal biology and disease"

Johnson RJ, Sanchez-Lozada LG, Nakagawa T. The effect of fructose on renal biology and disease. *J Am Soc Nephrol*. Dec 2010;21(12):2036-2039

In a 2010 paper, Johnson et al¹ reviewed the effect of fructose on renal biology and disease, and concluded "excessive fructose intake should be considered an environmental toxin with major health implications." Their conclusion overstates the effect of fructose on renal disease on two counts.

First, the mean intake statistics quoted by the authors (74 g/day) are outdated and exaggerate current fructose consumption. Marriott et al.² recently estimated mean (50th percentile) fructose intake at 9.1% of energy (49 g/day); the authors' value is closer to Marriott's estimate for the 90th percentile. And USDA-ERS data reveal that availability of the prominent fructose-containing sweeteners (sucrose and high fructose corn syrup) has been in decline since 1999.³ Thus, the authors' implication that fructose consumption is a growing problem is simply not correct; quite the opposite is true.

And second, the bulk of the experimental data cited in support of the authors' conclusion comes from exaggerated model systems not at all consistent with how fructose is consumed. Dietary fructose is derived from natural sources like fruits/vegetables/nuts and added sugars like sucrose/high fructose corn syrup/honey/fruit juice concentrates. But, fructose is accompanied in these sources by equivalent amounts of glucose.⁴ Many of the cited studies fed subjects fructose or glucose in the absence of the other sugar (author references 4, 6, 9, 12, 14, 16, 19-23), not at all consistent with human patterns of exposure. And as noted above, 50th and 95th percentile intakes of fructose as percent of energy were most recently estimated at 9.5% and <18%, respectively. Experimental data cited by the authors exposed human and animal subjects to fructose at 25-40% and 60+% of energy, respectively (author references 6, 9, 12, 14, 16, 19, 22, 23). Humans simply do not consume fructose in such extreme amounts or in isolation in the diet. Data gathered under such exaggerated conditions surely have low predictive value for assessing human health implications.

In summary, the case presented by Johnson et al. in favor of a role for fructose in renal disease is unconvincing, relying as it does on exaggerated statistics and experimental models that overstate the fructose-renal effect. The authors' conclusion may be accurate as worded, but it reflects exposures so remote from the real-world human experience that it is more inflammatory than useful in shaping public health policy.

References

1. Perez-Pozo SE, Schold J, Nakagawa T, Sanchez-Lozada LG, Johnson RJ, Lillo JL. Excessive fructose intake induces the features of metabolic syndrome in healthy adult men: role of uric acid in the hypertensive response. *Int J Obes (Lond)* 2010; 34(3): 454-61.
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(6): 1228S-1235S.