

# Calorie Control Council Response to Jürgens *et al*

## “Consuming fructose-sweetened beverages increases body adiposity in mice”

Jürgens H, Haass W, Castaneda TR, Schurmann A, Koebnick C, Dombrowski F, Otto B, Nawrocki AR, Scherer PE, et al. *Obes Res.* 2005 Jul;13:1146-56.

### Background

This study is a collaboration between research labs in Potsdam-Rehbruecke, Germany; University of Cincinnati; Otto-von-Guericke University in Magdeburg, Germany; Albert Einstein College of Medicine in the Bronx, NY; Charite University of Medicine, Berlin; and University of California at Davis.

### Hypothesis

The authors hypothesized that fructose might promote obesity.

### Justifications

- The marked increase in the prevalence of obesity in the US has been attributed to increased fructose consumption.
- Consumption of soft drinks has increased markedly in the past two to three decades; they are now the most popular refreshments among much of the world’s population.
- The fructose content of beverages sweetened with sugars ranges from 7% to 15% by weight (1).

### Experimental Design

If and how fructose might promote obesity was tested by measuring body composition, energy intake, energy expenditure, substrate oxidation and several endocrine parameters related to energy homeostasis in mice consuming fructose.

### Author Conclusions

- Exposure to fructose water increased adiposity (fat accumulation); consumption of soft drinks or diet soft drinks did not.
- Total energy intake was unaltered, because mice proportionately decreased caloric intake from chow.
- There was an insignificant trend (why mention it, then?) toward reduced energy expenditure and increased respiratory quotient in the fructose group.
- Fructose produced a hepatic lipid accumulation with a characteristic pericentral (surrounding the core) pattern.
- Data are compatible with the conclusion that a high intake of fructose selectively enhances adipogenesis (fat production), possibly through a shift of substrate use to lipogenesis.

### Critique

- These researchers make the same error many experimentalists have made: they confuse pure fructose with HFCS. HFCS used in US caloric soft drinks is primarily 55% fructose — not the 100% fructose used in this study. HFCS also importantly contains glucose in about the same proportions as in both the sucrose-sweetened soft drink in Jürgen’s study

and in table sugar. By contrast, the pure fructose used in the study contained no glucose.

- The absence of glucose makes pure fructose fundamentally different from HFCS, because glucose has been shown to have a tempering effect on adiposity. Once the combination of glucose and fructose found in HFCS and sucrose are absorbed into the blood stream — and regardless of whether they come from soft drinks sweetened with HFCS or with sucrose — they are indistinguishable from one another. They are metabolized according to pathways designed to utilize them effectively and efficiently.

- The authors cite William Dills, a Maillard/advanced glycation endproducts (AGE) researcher, as their resource for claiming “...the fructose content of beverages sweetened with sugars ranges from 7% to 15% by weight.” There was no such reference in Dill’s cited paper; Jürgen’s high-end figure appears to be a gross exaggeration.

The overwhelming majority of nutritively/calorically-sweetened beverages are carbonated sodas, which are 10-11% dry solids; half of this, whether from sucrose or HFCS, is fructose. Thus, fructose in carbonated beverages is approximately 5.5% by weight. A beverage that is 15% fructose by weight would be 30% by weight sucrose or HFCS (when glucose is added back) — and have three times the viscosity of a carbonated soft drink.

*This means that Jürgen’s rationale for using such inflated fructose levels is completely unjustified.*

- This study is at odds with a University of Barcelona study (2), in which rats given a liquid supplement of fructose or glucose (~ 20% of calories) showed no weight gain over the course of the experiment and actually reduced the amount of solid food consumed in comparison with the water-only rats. Jürgen attempts an explanation of how the fructose-fed rats can take in the same number of calories as the caloric and diet soft drink rats, but gain weight while the others did not — a thermodynamic impossibility — but it remains unconvincing.
- The current study and the Barcelona studies both showed good compensation between liquid and solid calories.
- It is curious that the authors chose to feed fructose and sucrose to mice at different concentrations: fructose at 15% and sucrose at 10% solids. This creates a two-dimensional problem — fructose in solution is ~ 20% sweeter than sucrose, and it will be sweeter yet when presented to rats at 1.5-times the concentration of sucrose. There is a very real possibility that the adiposity effects attributed to fructose are not due to the fructose *per se*, but rather to a simple over-consumption of calories due to a simple preference for increased sweetness or higher viscosity.

## References

1. Dills WL, Jr. Protein fructosylation: fructose and the Maillard reaction. *Am J Clin Nutr.* 1993 Nov;58:779S-87S.
2. Roglans N, Vila L, Farre M, Alegret M, Sanchez RM, Vazquez-Carrera M, Laguna JC. Impairment of hepatic Stat-3 activation and reduction of PPARalpha activity in fructose-fed rats. *Hepatology.* 2007 Mar;45:778-88.