

Calorie Control Council Response to Purnell et al

“Brain functional magnetic resonance imaging response to glucose and fructose infusions in humans”

Purnell JQ, Klopfenstein BA, Stevens AA, et al. Brain functional magnetic resonance imaging response to glucose and fructose infusions in humans. *Diabetes, Obesity and Metabolism*. 2011;13:229–234.

Purnell et al. [1] compared brain functional magnetic resonance imaging (fMRI) response to glucose and fructose infusions, and suggested differences offer support for a neurological basis for fructose-induced weight gain and higher fat consumption. While the authors' report of yet another bio-physiological difference between glucose and fructose makes for interesting reading, the experimental model is so far removed from real world diets as to be of little use in predicting human outcomes.

With rare exception, humans consume fructose with equivalent glucose, whether from natural sources like fruits/vegetables/nuts or from added sugars like sucrose/high fructose corn syrup/honey/fruit juice concentrates [2]. Testing fructose in the absence of glucose, as the authors have done, misses entirely the concurrent metabolic effect of glucose and ultimately exaggerates the effect of fructose. For example, Coss-bu et al. [3] recently reported that glycogen synthesis in the presence of fructose is significantly altered when glucose is given simultaneously. Glucose also makes a poor control since it is rarely consumed in the absence of fructose.

Purnell et al. report no significant effect of fructose or glucose vs. saline in the hypothalamus (Figure 4). It is entirely reasonable to expect no significant effect in the cortex either, were the authors to test the two sugars together vs. saline—the positive glucose effect would be effectively nullified by the negative effect of fructose. The unfortunate consequence of testing sugars in isolation is a loss of the big picture – the whole diet effect.

References

1. Purnell JQ, Klopfenstein BA, Stevens AA, *et al*. Brain fMRI response to glucose and fructose infusions in humans. *Diabetes Obes Metab*. 2011; 13: 229–234.
2. White JS. Misconceptions about high-fructose corn syrup: is it uniquely responsible for obesity, reactive dicarbonyl compounds, and advanced glycation endproducts? *J Nutr*. 2009; 139: 1219S-1227S
3. Coss-Bu JA, Sunehag AL, Haymond MW. Contribution of galactose and fructose to glucose homeostasis. *Metabolism*. 2009; 58: 1050-1058