

“Watch what you put in that sippy cup, experts warn.” *ScienceDaily.com*. May 22, 2007.

Richard Johnson, MD. (University of Florida College of Medicine) and Peter Havel, Ph.D. (University of California, Davis) were the scientific resources quoted in the article

Johnson statements:

- Fructose could trigger obesity in humans

Dr. Johnson appears unconvinced himself that fructose is responsible for obesity, causing him to qualify the statement with the word “could.” Presumably, he is referring to the hypothesis of Bray et al (1) stating that fructose (specifically high fructose corn syrup (HFCS)) is a *unique* cause of obesity. This hypothesis was developed based on a statistical correlation between increased fructose consumption (via HFCS) and increased rates of obesity during the 30 year period from 1970 to 2000.

It should be noted that 1) there is no proof that fructose is any more obesogenic at typical dietary levels than any other caloric food ingredient; and 2) dietary fructose levels attributable to HFCS have actually declined since 2000 (2), while obesity rates have continued to climb(3).

- Studies have linked excessive fruit juice and soft drinks with increased risk for obesity, diabetes and high blood pressure

Fruit juice contains a mixture of several simple sugars (glucose, fructose, and sucrose) and sorbitol, all of which are caloric; soft drinks contain both glucose and fructose, the two most common monosaccharides in the human diet. To imply that the fructose component is responsible for obesity, diabetes and high blood pressure is clearly a stretch. A far more reasonable explanation is that excessive caloric intake from caloric sweeteners in beverages and/or other sources of calories, imposes a significant caloric surplus that may result in obesity, diabetes and high blood pressure.

Importantly, a recent study by Sun and Empie compared frequent caloric beverage consumers with infrequent consumers using data from the USDA CSFII 1989-1991 and 1994-1998, and NHANES III 1988-1994 and combined 1999-2002 databases (4). They discovered that there was no difference in prevalence of obesity between the two groups.

- Fructose may not turn off satiety response, so you will continue to eat; Fructose, unlike glucose, does not signal the body to produce insulin, the hormone that turns sugar into energy and lets the brain know it's time to stop eating; Fructose causes resistance to insulin and blocks the “do not eat” order from reaching the brain

It has been a common practice among researchers for 50 years to exaggerate metabolic responses by feeding physiologically excessive levels of fructose (up to 35% of calories) to their test subjects in the absence of other carbohydrates. This

response, however sensational, has little applicability to what people really eat since fructose typically makes up about 8-9% of total calories and is *always* taken with other carbohydrates, most often glucose. Riby demonstrated that glucose has a moderating effect on fructose, attenuating the exaggerated metabolic response to one that is somewhat of an average between the two sugars (5).

Furthermore, a number of prospective studies using the Framingham Nurses and Women's Health studies also show a lack of correlation between fructose intake and weight gains (4, 6)

- Parents should limit fruit juice to 6 ounces per day. Children who drink more than 12 ounces of juice each day were heavier than those who drank less

It should surprise no one that there is a positive relationship between the amount of caloric food or beverage consumed and body weight. Taking things one step further, however, is the question of whether fructose beverage calories are somehow ignored by the body. There appear to be conflicting data on the subject.

Earlier studies comparing pure fructose (again at high levels) with pure glucose concluded that fructose calories were not as well compensated for as glucose calories, and that beverage calories were not as well compensated for as solid calories. Recent studies comparing "real world" sweetened beverages found that sugar, HFCS (half glucose and half fructose), aspartame and 1% milk all had similar effects on feelings of fullness (7, 8).

- Fructose and HFCS also cause the body to produce more uric acid, which caused insulin resistance [and obesity?]
There is no scientific data to suggest that normal levels of fructose in a typical diet cause increased production of uric acid.
- Sucrose is half fructose and could also activate pathways that increase the risk for obesity and insulin resistance
There is no scientific data to suggest that normal levels of sucrose increase the risk for obesity and insulin resistance, beyond the calories inherent in the sweetener

Havel statements:

- Because fructose doesn't stimulate insulin, consuming too much over a long period of time could trigger weight gain
The purported association between fructose and weight gain is not appropriate, since HFCS has largely supplanted sucrose on a 1-for-1 basis, resulting in no net gain of fructose in the diet relative to other nutrients (9).
- If you consume fructose, it's more like you're consuming fat
This is a current research area of Dr. Havel's. Again, however, there are differences of opinion among scientists as to the effect of fructose on triglyceride production. Excessive levels of dietary fructose in the absence of any dietary

glucose produce predictably high triglyceride levels (10). It is worth reiterating that such exaggerated exposures to fructose are highly artificial, highly prejudicial and produce predictable aberrant triglyceride levels.

Zukley and co-workers found no significant difference in triglyceride production when sucrose and HFCS were compared (11). This experiment tested “real world” sweetener compositions of fructose and glucose common in typical human diets.

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