Fructose:
Friend or Foe?

Fructose is a monosaccharide, also known as fruit sugar. It is the predominant sugar in most fruits leading many nutrition professionals to assume that since it's in fruit, it must be healthy. Are they correct? Let's investigate further as some believe that fructose is not a problematic sugar because unlike glucose, fructose does not require insulin to enter cells, thereby resulting in a minimal effect on the stimulation of insulin secretion. For this reason fructose has gained popularity as being the best sweetener for Diabetics.

How Does Fructose Get Absorbed?

Unlike glucose, fructose enters glycolysis or gluconeogenesis at the triphosphate level. Fructose is absorbed in the small intestine. It is rapidly taken up by the liver. Here, fructose is phosphorylated by ATP to fructose-1-phosphate, catalyzed by fructokinase (Hers 1952). Fructose-1-phosphate gets split into glyceraldehydes and dihydroxyacetone phosphate by hepatic aldolase B. These 2 metabolites feed into glycolysis, gluconeogenesis, glyconeogenesis, and lipogenesis.

Rapid liver assimilation of fructose has been proven to lead to elevated triglycerides, cholesterol, uric acid, and urea nitrogen. It also increases hepatic pyruvate and lactate production, decreases glucose tolerance, increases insulin resistance and causes a shift in balance from oxidation to esterification of nonesterified fatty acids resulting in elevated secretion of VLDL (Mayes 1993). Scientific evidence shows that fructose has effects on blood lipids, specifically serum LDL cholesterol. In baboons, dietary fructose has been shown to promote atherosclerosis in comparison to other carbohydrates.[1] Another study found that fructose glycosylated hemoglobin 7 times faster than glucose.[2] This may be important because glycosylation (as well as oxidation) of other proteins, including LDL & HDL particles, may increase the rate of artery clogging.[3]

Is the Food Industry High on Fructose?

Dietary fructose, high fructose corn syrup, and total sugar have all increased in the average American diet in the last 30 years. Consumption of sucrose (50% fructose) and high-fructose corn syrup has increased by 26% since 1970.

Unfortunately, even while evidence continues to mount for the hyper-cholesterolemic effect of fructose relative to other sugars, its use as a sweetener in the average American diet continues to increase. This is due largely to the displacement of sucrose (50% fructose) and corn syrup (0% fructose) with high fructose corn syrup (HFCS, which can contain up to 90% fructose) and crystalline fructose. In the 1980s soft drink manufacturers switched to HFCS because US tariffs on sugar made HFCS a less expensive sweetener. Others in the US foods industry also made this switch, and by 1985 HFCS accounted for 35% of the total amount of sweeteners by dry weight in the food supply.

How Does Consumption of Fructose Contribute to Cardiovascular Disease?

The mechanism by which dietary fructose increases LDL cholesterol is still a matter of debate. However, because fructose metabolism in the liver bypasses the rate-limiting step catalyzed by phosphofructokinase (PFK), it is more readily converted to acetate and fatty acids than glucose. In the short term, the addition of either sucrose or fructose to a fat load is known to greatly exaggerate the postprandial rise in serum triglyceride levels.[5]

Since both fructose and sucrose appear to increase insulin resistance, they may contribute to the development of Insulin Resistance Syndrome or Syndrome X and Type-2 Diabetes Mellitus, both of which increase the risk of cardiovascular disease.[6] In further research fructose feeding was also found in animal studies to induce hypertension and hypertriglyceridemia.[7] A high fructose diet was also found to cause more lens damage in severely diabetic rats.[8] Other research on high dietary fructose found it significantly increased kidney calcification in both male and female rats, particularly when dietary magnesium was low. This exhibits an interaction between dietary fructose and magnesium to adversely affect macro mineral homeostasis.[9] This would suggest further studies to see if a high fructose diet coupled with low dietary

magnesium and marginal calcium leads to bone loss. These presumably adverse metabolic effects of consuming fructose or sucrose appear to warrant a position of limiting the use of refined fructose-rich sweeteners in diabetic or obese subjects, as well as individuals withelevated blood lipids. Nevertheless, the American Dietetic Association adopted a position paper on the use of nutritive and nonnutritive sweeteners that stated, "At this time there is no evidence that current levels of fructose intake contribute to hyperlipidemia."[10] Obviously there is good evidence but none of the research referenced above was discussed in the current ADA position paper on sweeteners.

Does Fructose Contribute to Weight Gain?

Consider research from Indiana's Purdue University which tested 14 overweight individuals following a 40-minute workout. Subjects were split into two groups, and assigned to follow either a normal or low calorie diet. After six days on each diet, they were then given a meal containing either 50 grams of glucose, or a similar quantity of fructose.

Diet	Fat Oxidation after Fructose	Fat Oxidation after Glucose
Normal calorie	18 grams	29 grams
Low calorie	28 grams	29 grams

As you can see, there was no real difference in oxidation rates during the low calorie diet. However, when subjects were tested following the normal diet, fat oxidation was 38% lower after the high fructose meal.[11]

It is interesting that consumption of sucrose from 1970 to 1997 decreased from 46kg to 30kg while at the same time the consumption of fructose increased from a mere .23kg to 28kg! Yes, that was .23, not 23. In the same past 2 decades we have seen the greatest increase in the prevalence of obesity. Three human studies show negative effects of dietary fructose on weight gain. One study reported that drinking 1150 g of soda sweetened with HFCS for 3 wk resulted in significant increases in body weight compared with the same amount of soda with aspartame in male and female subjects.[12] In another study, a group of 14 middle-aged men, 11 with Type 2 Diabetes and 3 with Type 1, who consumed 50-60g of fructose for 24 wks showed an increase in body weight.[13] The third study compared weight gain from an artificial sweetener with the consumption of sucrose (50% fructose). Individuals who consumed large amounts of sucrose (28% of energy) showed an increase in energy intake, body weight, fat mass and blood pressure after the 10 wk intervention.[14]

Can Fructose Consumption Contribute to the Cause of Hypertension?

Many studies have shown that high-fructose diets induce hypertension in animals. In a matter of fact, it is interesting that to study whether medications are effective in treating hypertension, fructose-fed rats are often used as a model. One researcher showed that long-term (40 week) fructose feeding impaired vascular relaxation in the mesenteric arteries of male rats.[15] A study published in the Journal of Hypertension proved fructose feeding induced hypertension in rats fed normal diets and high salt diets and was associated with an increased expression of the angiotensin II type 1 receptor in adipose tissue.[16]

Does Fructose Contribute to Aging?

Another negative of fructose is that it can glycate with proteins. This is called the Maillard reaction. Accelerated aging and several complications of Diabetes Mellitus can be blamed on this glycation.

In an interesting study by Levi and Werman, 1998, rats were fed either fructose, glucose or sucrose for one year. Blood fructose, cholesterol, fructosamine (fructose bound to protein), glycated hemoglobin levels, and urine lipid peroxidation products were significantly higher in fructose-fed rats than in the other groups (J.Nutr 128:1442-1449, 1998). "The data suggest that long-term fructose consumption induces adverse effects on aging; further studies are required to clarify the precise role of fructose in the aging process."[4]

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