Is High-Fructose Corn Syrup the New Trans Fat?

Researchers link excessive fructose intake with obesity, cardiovascular disease, and renal disease epidemics.

Some evidence suggests that high consumption of fructose plays a role in the epidemics of obesity, hypertension, diabetes, and kidney disease. In the U.S., these epidemics have been paralleled by a rise in sugar consumption. High intake of free fructose (found in high-fructose corn syrup, but not in sucrose) is associated with hypertension and hyperlipidemia in animals and with dyslipidemia and insulin resistance in adults. Further, small particle size of low-density lipoprotein (LDL) is associated with obesity, the metabolic syndrome, and central adiposity in older children and adults. Investigators in Switzerland examined the relation between dietary fructose consumption and obesity, distribution of body fat, plasma lipids, and LDL particle size in a convenience sample of 74 children (age range, 6–14 years) recruited from schools and pediatric clinics; 43 children were overweight (mean BMI, 23.4), and 31 were normal weight (mean BMI, 15.9).

Dietary intake was measured by two 24-hour dietary recalls and a 1-day weighed food record. LDL particle size, triglycerides, and serum cholesterol were measured after a 12-hour fast. Overweight children had significantly higher plasma triglyceride levels, lower high-density lipoprotein (HDL) levels, and smaller LDL particle size than normal-weight children. LDL particle size was associated with overall adiposity and central adiposity. The only dietary factors that correlated significantly with LDL particle size were total fructose intake and grams of fructose per 1000 kcal consumed; higher fructose consumption was inversely associated with LDL particle size, independent of adiposity. Fructose intake did not correlate with any other lipid variable.

Comment: Disentangling the effects of excessive dietary fructose from those of excessive dietary fat is difficult. The authors of this study and of an accompanying editorial and review provide plausible mechanisms for the deleterious effects of fructose consumption. They posit that fructose may not induce satiety to the same extent as glucose, perhaps because fructose does not stimulate release of insulin or leptin or inhibit secretion of hormones known to affect satiety. Moreover, fructose is the only sugar that raises serum uric acid concentrations; high uric acid levels are known to have negative cardiovascular effects. In fact, lowering uric acid levels reduces blood pressure in adolescents with new-onset hypertension. Once inside liver cells, fructose can enter pathways to form glycerol, the essential component of triglycerides.

Despite efforts to lower fat intake during the past 2 decades, obesity levels have continued to rise. At the same time, fructose intake has increased. In a recent meta-analysis, consumption of soft drinks (which contain high-fructose corn syrup) was associated with increased caloric intake and body weight. Beverages sweetened with high-fructose corn syrup provide little nutritional value and displace essential nutrients from children’s diets. Perhaps fructose deserves the same kind of scrutiny as trans fat and the same type of interventions aimed at reducing intake.

— Alain Joffe, MD, MPH, FAAP

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