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Dietary Sugar and Body Weight: Have We Reached a Crisis in the Epidemic of Obesity and Diabetes? Health Be Damned! Pour on the Sugar

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Sugar-sweetened drinks have been associated with several health problems. In the point narrative as presented below, we provide our opinion and review of the data to date that we need to reconsider consumption of dietary sugar based on the growing concern of obesity and type 2 diabetes. In the counterpoint narrative following our contribution, Drs. Kahn and Sievenpiper provide a defense and suggest that dietary sugar is not the culprit. Data from the National Health and Nutrition Examination Survey and U.S. Department of Agriculture dietary surveys along with commercial Homescan data on household purchases were used to understand changes in sugar and fructose consumption. Meta-analyses and randomized clinical trials were used to evaluate outcomes of beverage and fructose intake. About 75% of all foods and beverages contain added sugar in a large array of forms. Consumption of soft drinks has increased fivefold since 1950. Meta-analyses suggest that consumption of sugar-sweetened beverages (SSBs) is related to the risk of diabetes, the metabolic syndrome, and cardiovascular disease. Drinking two 16-ounce SSBs per day for 6 months induced features of the metabolic syndrome and fatty liver. Randomized controlled trials in children and adults lasting 6 months to 2 years have shown that lowering the intake of soft drinks reduced weight gain. Recent studies suggest a gene-SSB potential relationship. Consumption of calorie-sweetened beverages has continued to increase and plays a role in the epidemic of obesity, the metabolic syndrome, and fatty liver disease. Reducing intake of soft drinks is associated with less weight gain.

This point-counterpoint is about the health hazards of sugar, high-fructose corn syrup (HFCS), and fructose. That there could even be this debate reflects the fact that sugar, HFCS, and particularly fructose are sweet and that there is an innate human desire for sweetness. If sugar, HFCS, and fructose were not sweet, there would be no debate because their consumption would be low. Sweet taste is present in newborn babies and increases in intensity through childhood. It may be that the “craving” for sweets can even be enhanced by early exposure to intense sweeteners. Certainly the ability of human beings to consume all of the sugar that has been produced would suggest this. In 1776—at the time of the American Revolution—Americans consumed about 4 lbs of sugar per person each year. By 1850, this had risen to 20 lbs, and by 1994, to 120 lbs. The food industry has used sugar as a major sweetener for delivery for increasing amounts of beverages and food over the past half-century (1). The result has been that the consumption of

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sugar-sweetened beverages (SSBs) rose by a startling 38.5 gallons per person between 1950 and 2000 (10.8 gallons per person in 1950 to 49.3 gallons per person in 2000) (2). We have seen small declines since then; however, the industry continues to find new ways to increase liquid sugar consumption by constantly adding new products, be they in fruit juice, energy drinks, vitamin waters, protein waters, sports drinks, and hundreds of new options. Thus the question—do current levels of sugar consumption pose a serious health risk to Americans?—seems in crying need of clarity. Our proposition is that sugar and related caloric sweeteners in the amounts now consumed pose a substantial risk and that the public needs to be better informed about these risks as they select the food they eat.

The decade of the 1980s began quietly enough for obesity research and for the sugar industry. The prevalence of obesity, though rising slowly, was still only 14% (3). Sugar had received a relatively clean bill of health from the National Academy of Sciences in the Diet and Health Report—its only health problem according to this authoritative book was its role in dental caries (4). The metabolic syndrome was yet to be clearly defined (5), we did not know that consumption of calorically sweetened beverages was not adequately compensated by a reduction in food intake, and the concept of non-alcoholic fatty liver disease was only beginning to emerge (6). It was the calm before the storm. What a difference 25 years can make! As we look back, we conclude that the increasing consumption of sugar, the appearance of HFCS, and the fructose that they both contain have dramatically increased the health risks. It is our opinion that the current levels of sugar/fructose consumption contribute to the ill health of many Americans (7,8). We also do not want to give the glucose component of sugar an easy pass and create a new generation of glucose beverages as glucose also has critical cardiometabolic effects (9,10), but focus mainly on the fructose component.

“Some factor of diet and/or lifestyle must be driving weight upward, because human biology and our underlying genetic code cannot change in such a

short time” (11). There is clear evidence that dietary factors are driving weight up, but genetic variety also plays a key role. Some genes have a major effect on obesity (12), while others contribute only a small amount individually but collectively provide the background for individual responses to diet (13–15). A gene-environment interaction was shown recently for intake of soft drinks and weight gain (15). We might thus state the relation of diet and genes this way: Genetic variability loads the gun; diet/environment pulls the trigger.

Our research interest in this problem was stimulated by a quotation from Yudkin: “If then there is reason to be concerned about a dietary cause of a widespread disease [obesity], one should look for some constituent of man’s diet that has been introduced recently or has increased considerably, recently” (16). Even earlier Yudkin warned us of the dangers of sugar (17), but his words fell on deaf ears. We began to explore the issue of the sweetening of our global diet separately—Bray in an article to the International Congress on Obesity in Sao Paulo, Brazil, in 2002 based on preliminary work by a postdoctoral fellow; Drs. Paeratakul and Popkin with a focus on large global sugar increases and also U.S. consumption shifts in consumption (18–22). The Paeratakul-Bray work showed that HFCS consumption rose along with the increasing prevalence of obesity during the latter half of the 20th century. Shortly after, in 2004, we published an article entitled “Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity,” which described the state of affairs at that time (7). Although correlations do not prove causation, and total sugar consumption has not increased in the last few years (23) and has actually decreased in recent years in the U.S. (24,25), sugar consumption is still at very high levels. Our initial article highlighted the potential importance of fructose in HFCS and SSBs and posited the hypothesis that it could represent a potential pathway for some of the detrimental effects of sugar (26–31).

Sucrose is one of the few components of our diet for which no upper limits are suggested in either our dietary guidelines or by the U.S. Food and Drug Administration (32). However, two European front-of-the-package labeling

systems—the U.K. Department of Health requires this with its traffic light system and the Denmark-Netherlands Choices International system—do use excessive added sugar as a component (33,34). We believe that, along with other macronutrients, total sugar intake should have an upper recommended level of, say, 10% (34,35). In addition, the public should be warned about the direct pernicious effects of sugar on the development of the metabolic syndrome and fatty liver disease (36–38). Even in 1999, the food industry understood its role in creating the obesity epidemic, but failed to take clear action (1).

HEALTH RISKS ASSOCIATED WITH SUGAR OR HFCS INTAKE

In a meta-analysis of adults with ad libitum diets, reduced intake of dietary sugars was associated with a decrease by 0.80 kg in body weight. Conversely, increased sugar intake was associated with an increase of 0.75 kg. As expected, the isoenergetic exchange of starch for sugar had no effect. Thus, changing sugar intake affects body weight unless there are corresponding changes in other nutrients. Analysis of SSBs showed a weight gain of 1.55 kg over 1 year among groups with the highest intake compared with those with lowest intake. These authors conclude that among free-living people intake of free sugars or SSBs modifies energy intake and thus body weight (35).

In another meta-analysis, intake of sugar and animal products in >75 countries was directly associated with gross domestic product and urbanization rates. In a multivariate regression model, sugar consumption ($P = 0.03$), physical inactivity ($P = 0.003$), and cereal consumption ($P < 0.001$) were significant predictors of obesity. These authors conclude that high sugar consumption and sedentary lifestyle are associated with increased risk of obesity (39).

In another survey of 75 countries, soft drink intake increased globally from 9.5 gallons per capita per year in 1997 to 11.4 gallons in 2010. A 1% rise in soft drink consumption was associated with an additional 48 overweight adults per 100 people, 2.3 obese adults per 100, and 0.3 adults with diabetes per 100. These findings remained robust in

low- and middle-income countries, again showing the detrimental effects of higher sugar intake on the risk of becoming overweight or obese (40).

Fructose comes from many sources; however, it first came to fame as a component of HFCS. Fructose from any sugar is just as bad, whether delivered in beverages or solid food. Numerous research studies have shown that overall calories have increased in our diet over the past 40 years, particularly from beverages (20,41–43). More recently there have been declines, but, overall, caloric intake remains excessive and declines in beverage intake are small (24,25,44). A recent meta-analysis showed that adding fructose to the diet in controlled studies produced weight gain unless calories were reduced somewhere else in the diet—i.e., a hypercaloric diet produced by adding fructose produced weight gain (45); however, fructose intake is mostly HFCS and even fruit juice concentrate comes from sugar or HFCS in beverages (43) (Fig. 1).

In the 1960s and 1970s, sugar from beverages represented only a third of our total added sugar intake, but this rose to two-thirds of our sugar intake by the year 2000 and has subsequently been declining to about 40% of total added sugar intake (46,47). Nevertheless, an array of meta-analyses have shown a powerful relationship between sugar consumption in beverages and obesity, diabetes, the metabolic syndrome, and cardiovascular disease (36,48–52). It is quite possible that intake of SSBs represents a major reason for the rapid increase in the metabolic syndrome (5), which was first detected in the 1980s and now affects about a fourth of U.S. adults.

HEALTH RISKS ASSOCIATED WITH FRUCTOSE

Fructose is essentially 50% of both sugar and HFCS. While the glucose from all sucrose has important effects on obesity and elicits many other adverse health responses (10), it appears that fructose, when consumed at high levels, has critical adverse effects. The data that have accumulated in the last 10 years from both meta-analyses of epidemiological studies and from randomized clinical trials, along with the experimental studies on the effect of fructose, would suggest that our earlier article on HFCS and

beverage intake (7) might have been better titled as: “Consumption of calorie-sweetened beverages and the fructose they contain may play a role in the epidemic of obesity, the metabolic syndrome, and fatty liver disease.” We will briefly examine each of these issues. In addition, we provide some insights into the continued use of HFCS and other newer caloric sweeteners containing fructose—fruit juice concentrate.

Fructose is a sweet-tasting sugar that is found naturally in fruits and some vegetables and has been part of the human diet for eons in modest amounts (53). It has the highest sweetness taste of all natural components of sugar. Its dramatic increase in the past 30 years led to concern and extensive research (7). It is not the only caloric sweetener found in our food supply. As noted, about 75% of all U.S. foods and beverages contain added sugars (43), and only in SSBs, yogurt, and a few other food categories do we find that HFCS is the dominant sweetener. Nevertheless, the large increase in added sugar has led to a major increase in total fructose intake, an increase that has occurred since about 1980 (46). While many health problems are linked with this increase in fructose intake, fatty liver disease is one which increase is noted in both the U.S. and Europe and certainly is linked with excessive fructose intake (54).

THE NEW NATURAL SUGAR–FRUIT JUICE AND FRUIT JUICE CONCENTRATE

For a long time, the beverage industry tried to state that cane sugar and beet sugar were natural, and HFCS was the unnatural unhealthy sugar. At the same time, there has been steadily increasing promotion and sales globally of fruit juice and fruit juice concentrate that consumers consider natural and yet ignores the fact that these are often just another source of concentrated sugar. In the U.S. food supply, 7% of all foods and beverages have fruit juice concentrate used as an added sugar (43). Furthermore, if we counted fruit juices (defined to be 100% fruit juice), most of which are made by combining fruit juice concentrate, water, and flavorings, we would find that over 98% of the fruit juice sold in bottles or containers are based on fruit juice concentrate.

In Western Europe, including the U.K., and the U.S., the per capita intake of fruit juice is approximately 50–60 kcal/day (42,55,56). However only 20–35% of children and adults consume fruit juice, thus among fruit juice consumers actual intake per consumer is over 100 kcal/day across these countries. Universally, adults consider fruit juice to be a “healthy” beverage, and most studies of dietary intake patterns associated with an otherwise healthy diet among children find fruit juice as the beverage parents provide their children (57).

In most countries, some public health authorities are attempting to limit fruit juice intake to approximate 4 ounces (\approx 118 mL) per day maximum (58). Furthermore, the only literature examining long-term effects of fruit juice consumption finds either no effect or adverse effects on both weight and risk of diabetes (59–62).

MECHANISMS FOR PRODUCING UNDESIRABLE SIDE EFFECTS FROM SUGAR, HFCS, AND FRUCTOSE

The relation of SSBs to obesity can be attributed to two different effects. The first is the increased caloric intake. As noted above, adding fructose to the diet without subtracting other sources of energy produces weight gain (45,63). In addition, several meta-analyses have shown this relationship. Second, beverages do not suppress the intake of other food calories to an appropriate degree to prevent weight gain. Thus, beverage calories can be viewed as “add-on” calories, enhancing the risk of obesity. The pathbreaking work by Rolls and colleagues (64–66) and Mattes and colleagues (67–70) has led to dozens of replications highlighting this relationship. Several short-term clinical trials have provided insights into the metabolic consequences of ingesting fructose in calorie-sweetened beverages. In an early clinical study comparing the effect of glucose, fructose, and sucrose on plasma triglycerides, Cohen and Schall (63) found that both fructose in the amount found in sucrose and sucrose increased triglycerides following a meal, but that glucose did not, leading them to conclude that the effects on lipids were due to the fructose either alone or as part of sucrose (table sugar), and not glucose. Another 10-week study comparing beverages providing 25% of

calories from fructose with a beverage providing 25% of calories from glucose showed that fructose increased triglycerides, particularly at night (71). This study also showed that fat synthesis (de novo lipogenesis) was increased in those consuming fructose-containing drinks. Most important, visceral fat increased in a fat depot, which has the strongest association with cardiovascular risks with only 10 weeks of drinking a fructose beverage compared with the glucose beverage (26,72). A third study compared daily intake of 1 L per day—approximately two 16 ounces of cola, diet cola, milk, or water. The sugar-sweetened cola consumed for 6 months increased liver fat, visceral fat, muscle fat, and triglycerides compared with the others (73). Thus, as little as 6 months of consuming two 16-ounce servings of a sugar-sweetened cola beverage per day will increase the risk of fatty liver and the metabolic syndrome, something not seen with the aspartame-sweetened beverage, milk, or water. A schematic representation of the health consequences is shown in Fig. 2 (9). This figure pulls together the findings from the studies described above into a single model (26,69,73–75). The high levels of consumption of SSBs and other sugary beverages (23,42,46,76,77) is viewed as the driver for the increase in energy and

fructose, which play a part in the development of obesity and the metabolic consequences depicted here (78). The caffeine present in these beverages may serve as a positive feedback signal due to its ability to stimulate the central nervous system. Interestingly, the U.S. Food and Drug Administration is currently reviewing caffeine use, a drug which some consider mildly addictive. Even 3 weeks of SSB ingestion was sufficient to alter lipid metabolism by decreasing LDL and increasing HDL, which is a marker of increased cardiovascular disease risk (9,79). This and a study by Aeberli et al. (74) provide insights into the unique role of fructose in initiating liver dysfunction and possibly leading to nonalcoholic fatty liver disease and the metabolic syndrome, which have become increasingly prevalent (54,77).

From a public health perspective, it is concerning that drinking two SSBs per day for 6 months can induce features of the metabolic syndrome and fatty liver. These studies certainly need to be repeated, but if replicated, the public should be warned about the hazards of drinking SSBs in much the same way as the U.S. Food and Drug Administration warns people about risk of taking medications.

A recent series of randomized controlled trials in children and adults lasting 6 months to 2 years have shown that weight gain is slowed by replacing SSBs with alternative beverages. The two most noteworthy were done by a group from Boston (80) and a group from Amsterdam (81). After 1 year, the control group in the Boston study gained significantly less weight than the group receiving the SSBs. The Amsterdam study went further and provided either 250 mL of low-caloric sweetened beverage or a sugar-containing beverage providing 104 kcal to 641 youth over an 18-month period. The BMI, weight, skinfold-thickness, and fat mass increased significantly less in the low-caloric beverage group. A third 6-month adult study with three arms—low-calorie beverage, water, and normal beverage intake—found significantly greater likelihood of a 5% weight loss among the first two groups compared with the normal beverage group, but all lost weight as this was part of an active weight-loss regimen (82).

One key question that Aeberli et al. (74) begin to address is whether the detrimental effects of fructose are simply the result of a linear dose-response to our increasing dietary intake of fructose, or whether there is a threshold below which fructose is without harm. The

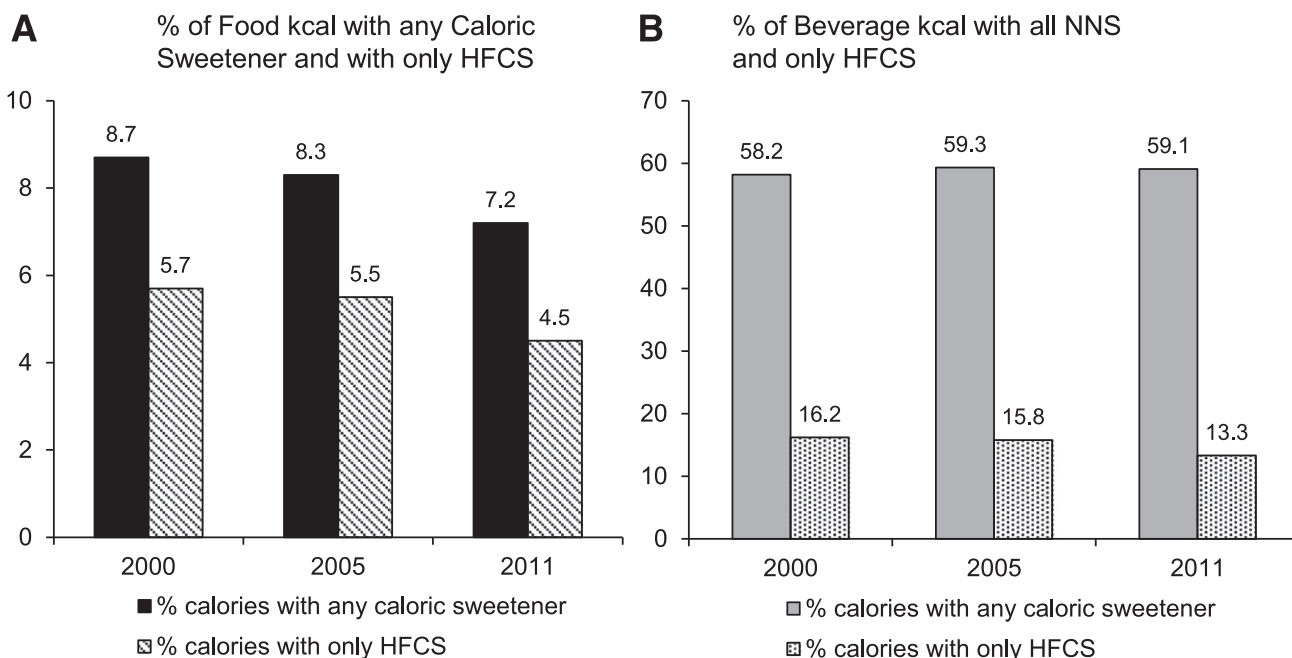


Figure 1—Proportion of total calories in foods and beverages purchased in the U.S. food supply sweetened with any caloric sweetener and with HFCS, 2000–2011. NNS, non-nutritive sweetener. Source: Homescan data linked with Nutrition Facts panel data for 2000, 2005, and 2011, weighted to be nationally representative (43).

current data suggest that it is a “linear” response and that the reason we are now detecting the pathophysiological consequences of fructose is that its dietary load has continued to increase, largely as a consequence of increased soft drink and fruit drink consumption. This is particularly important as many studies have shown that there is a group of adolescents and young adults that consume large amounts of SSBs both in the U.S. and across many other countries whose gene-beverage interaction may make the outcomes worse (45,55,56,76,83–85). In fact, it appears that a major push toward marketing

SSBs and other sugary beverages exists in low- and middle-income countries (86).

CONCLUSIONS AND RECOMMENDATIONS

These recommendations are based on two principal facts: The current surge in BMI and obesity began about 1975 and that calorically sweetened beverages, and possibly other sugar-containing foods, play a role in the development of obesity.

Recommendations for the individual:

1. Chose water, unsweetened coffee, or tea in place of calorically sweetened beverages.

2. Chose and eat fruit rather than drink fruit juice or fruit drinks.
3. If you drink calorically sweetened beverages, reduce your levels to the average 6 ounces per day for adults (>19 years) and 7 ounces for kids aged 2–18 years, which was the intake in 1977–1978.

Recommendations for society:

1. Reduce average intake of sugar to the levels seen in 1977–1978.
2. Make healthy alternatives comparable in cost to the items they are expected to replace.

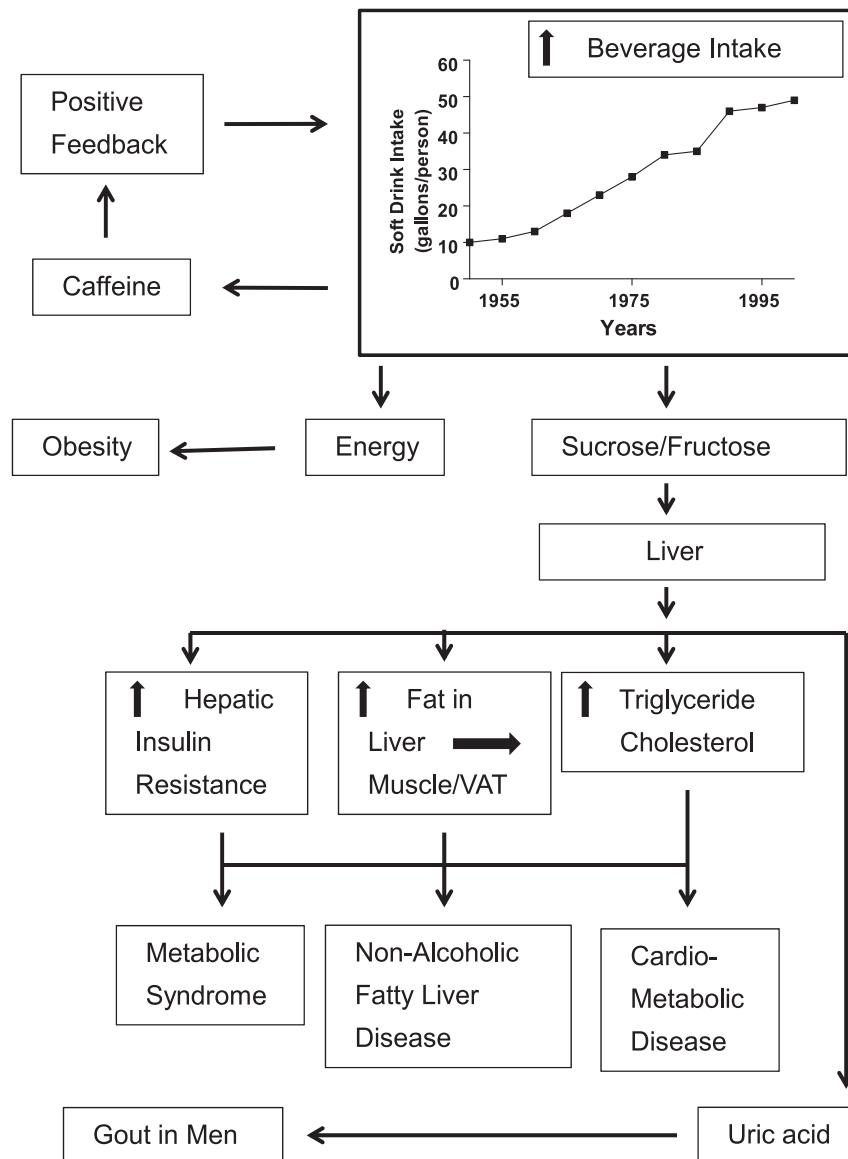


Figure 2—Model showing some potential consequences of increasing fructose and energy intake from sugar or HFCS in beverages. VAT, visceral adipose tissue.

3. Treat caffeine as the “mildly addictive” drug that it is and limit its use as it may drive the intake of caffeinated beverages.

Recommendations for government:

1. Provide greater subsidies for vegetable and fruit crops.
2. Provide added financial incentives for government-funded food programs to increase fruit and vegetable consumption (WIC, SNAP, School Feeding).
3. Provide incentives for stores in low-income areas to carry fresh produce.
4. Add guidance about beverages and sugar intake to Dietary Guidelines for Americans.

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