Effects of Sugar-Sweetened Beverages on Children

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Sugar-sweetened beverages (SSBs) are typically defined as beverages containing added caloric sweeteners such as sucrose, high-fructose corn syrup (HFCS), or fruit-juice concentrates, which include but are not limited to soft drinks, fruit drinks, sports drinks, energy and vitamin water drinks, sweetened iced tea, and lemonade, among others.

Importantly, the increased consumption of SSBs by people of all ages and ethnic groups has been implicated in the increased incidence of obesity and metabolic syndrome, a cluster of conditions associated with insulin resistance, including hypertension, dyslipidemia, central adiposity, impaired glucose metabolism, non-alcoholic fatty liver disease, and polycystic ovary syndrome.

In the US, between 1977 and 2002, energy intake from soft drinks and fruit drinks increased by approximately 230% and approximately 170%, respectively.1 Currently, sugar in liquid form accounts for roughly 35% of the total added-sugar intake in the US.2 Particularly relevant to the pediatric population is that consumption of SSBs often displaces consumption of healthier beverages such as water and/or nutrient-dense beverages such as milk.

In the US alone, health care costs attributable to obesity are currently estimated to be nearly $150 billion. The modulation of obesity and cardiometabolic disease risk factors is, thus, of paramount importance from a public health standpoint; reducing the consumption of SSBs is a great place to start.

SSB-ASSOCIATED ENERGY INTAKE TRENDS

The past 3 decades have evidenced a parallel rise in obesity and SSB consumption (see Figure 1, page 28).1 The percent of total daily calories from SSBs has increased more than twofold in this same time period (from an estimated 3.9% in the late 1970s to around 9.2% in the early 2000s).3

Despite a decline in the consumption of added sugars during the past few years, mean intakes continue to exceed...
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Food advertisers specifically target younger people, given that they have the means and opportunity to establish their own consumer behavior patterns. For example, in 2006, food marketers spent roughly $1.05 billion marketing to children and adolescents, almost half of which was spent to promote carbonated beverages. SSBs are also heavily promoted in the media; in 2000 alone, the soft drink industry spent more than $700 billion in advertisements, an 84% increase since 1986.

LIQUID CALORIES VS. TOTAL CALORIES

Calories from liquids seem to be particularly egregious in terms of promoting excessive weight gain. People do not appear to self-regulate energy consumed in liquid form vs. energy consumed from solid foods, especially when consumed with meals. Moreover, dietary compensation for beverages appears to be weaker than for solid foods of comparable nutrient content, leading to a greater risk of positive energy balance. Liquid forms of energy also appear to have a lower satiety effect than do isocaloric solid forms.

Higher consumption of calories in liquid form also is associated with higher total energy consumption. For example, adolescents drinking 8 oz or more of non-diet soda per day consume more total energy than those drinking non-SSBs. Children who consume the largest amounts of SSBs ingest about 330 kcal/day more than those who consume the least. Recent data suggest that total energy intake among adolescents would be reduced by approximately 235 kcal/day if all SSBs were replaced with water.

ADIPOSITY AND METABOLIC DISTURBANCES

SSB intake is specifically correlated with both adiposity and metabolic disturbances. While a few studies have not demonstrated an association, most report a positive association between SSB intake and increased body weight, increased fat mass, dyslipidemia, increased blood pressure, and other metabolic parameters associated with insulin resistance and metabolic syndrome.

INTERVENTION TRIALS SHOW THAT DECREASING SSB CONSUMPTION REDUCES BOTH ADIPOSITY AND BMI.

USE OF FRUCTOSE IN SSBs

In the US, SSBs are sweetened primarily with HFCS, which has resulted in an estimated 30% increase in the amount of fructose consumed in the US since 1970. Americans older than 2 years of age are estimated to consume 130 kcal/day or more from HFCS. Sugar intake from SSBs alone, which currently represent the largest single caloric food source in the US, now approaches or exceeds 15% of the daily caloric intake in several population groups, including adolescents. According to the Corn Refiners Association, HFCS is defined as either 42% or 55% fructose, with the remaining percentage as glucose. However, a recent study suggested that the mean fructose content in some popular off-the-shelf HFCS-containing SSBs in Los Angeles was 59%, with a few products containing HFCS composed of as much as 65% fructose. Given the metabolic dichotomies of fructose vs. glucose, this increase in the percentage of fructose in HFCS-containing beverages may not be insignificant.

METABOLIC DETERMINANTS OF FRUCTOSE

Although fructose (fruit sugar), glucose (starch), and galactose (milk sugar) are isomers with the same chemical formula (C6H12O6), the metabolism of fructose is markedly different from the other two. Although fructose is isocaloric with glucose and galactose, it is not isometabolic. Unlike glucose, in which the liver only metabolizes roughly 20%
of an ingested bolus, fructose is a highly lipogenic nutrient. After its absorption from the gut into the portal vein, it is almost completely metabolized in the liver. In contrast to glucose, the initial steps of fructose metabolism are insulin-independent (Figure 2, see page 29).

Whereas hepatic glucose metabolism is regulated by cellular energy status and the negative feedback control by adenosine diphosphate (ADP) and citrate on the initial steps of glycolysis, hepatic fructose metabolism is not subjected to feedback inhibition; it bypasses the phosphofructokinase regulatory step in glycolysis, providing a rapid influx of hepatic energy substrate that must be metabolized.30 In a glycogen-depleted state (eg, vigorous exercise), much of an ingested fructose bolus can be isomerized from fructose-1,6-bisphosphate back to glucose-6-phosphate for repletion of glycogen.

However, in the glycogen-replete state, virtually all of the ingested fructose is rapidly converted to hepatic triose phosphates, causing immediate increases in pyruvate and lactate production, extra substrate for mitochondrial metabolism, and a shift in balance from hepatic beta-oxidation to synthesis and esterification of fatty acids. This results in de novo lipogenesis and increased intrahepatic triglyceride (TG) and very low density lipoprotein (VLDL) production.30 The increased postprandial circulating VLDL leads to TG deposition in adipose tissue (causing obesity), and also fat deposition in ectopic tissues such as skeletal muscle (causing intramyocellular lipid accumulation) and liver (causing hepatic steatosis), eventually resulting in insulin resistance and dyslipidemia.31 In controlled isocaloric feeding studies, fructose consumption preferentially induces visceral adiposity, which drives cytokine production, and predisposes to the development of type 2 diabetes mellitus (T2DM) and other cardiometabolic disorders.23

Fructose consumption associated with SSBs also causes loss of inorganic phosphate and diminished adenosine triphosphate (ATP) synthesis in the liver, removing the inhibition by ATP of the enzymes involved in purine nucleotide degradation. This promotes uric acid formation,30 which is excreted from the liver. High serum uric acid concentrations inhibit endothelial nitric oxide synthase, potentially contributing to fructose-associated blood pressure changes and cardiovascular disease.32

Biophysical differences between fructose and glucose also potentiate fructose’s adverse effects.

Lastly, fructose reduces the secretion of insulin and leptin (preventing central transduction of the satiety signal), and attenuates the postprandial suppression of ghrelin (promoting continued hunger) — endocrine changes that may promote weight gain by maintaining a sensation of “hunger” in the hypothalamus.30 Fructose also appears to inhibit dopamine signaling within the nucleus accumbens, the brain’s “pleasure center.”33

ADDITIONAL METABOLIC DETRIMENTS

Other aspects of SSBs may also contribute to pathology. The glucose component of sucrose and HFCS may have its own deleterious effects. Even though SSBs have a relatively low glycemic index (Coca Cola, for example, has a glycemic index of 53, whereas pure glucose has a glycemic index of 100), rises in blood glucose and insulin levels still occur after the ingestion of SSBs due to the glucose content. In turn, high glycemic load diets have been shown to stimulate appetite and induce weight gain. They also have been associated with the development of insulin resistance, dyslipidemia, and glucose intolerance, and have been shown to increase levels of inflammatory biomarkers such as C-reactive protein, a known risk marker for cardiometabolic disease.34 Moreover, the caramel coloring used in many SSBs — especially colas — may further
increase insulin resistance and inflammation because of the high amounts of advanced glycation end-products that result from the caramelization process.

**ADIPOSY AND METABOLIC DISTURBANCES**

In children and adolescents, an effect size of around 0.08 unit change in BMI per 12 oz of SSB consumption (soda in particular) has been reported,\(^3^5\) as has a 0.90-percentile increase in BMI percentile for age per SSB serving equivalent.\(^2^4\) Each additional SSB consumed per day is also associated with an increased estimate of insulin resistance, an increase in systolic blood pressure, an increase in waist circumference, and a decrease in high-density lipoprotein (HDL) cholesterol concentrations.\(^2^4\)

The consumption of SSBs has been implicated in at least 20% of the net weight gained in the US population between 1977 and 1978 and 1999-2001.\(^2\) The odds of a pediatric patient becoming obese — and therefore at risk for developing metabolic syndrome — is increased by approximately 60% for each additional SSB serving per day.\(^3^6\) Thus, the observation that the average intake of SSBs in US children and adolescents is now estimated to be more than double the amount consumed in the 1970s has tremendous public health implications.\(^3\)

**OBESITY AND CARDIOMETABOLIC DISEASE PREVENTION**

Although SSBs are not the sole cause of the obesity epidemic, they have several characteristics that make them promising targets for obesity prevention efforts. First, SSBs are a clearly defined category and are unlike other “junk foods” that may contain some positive nutrients such as protein, fiber, or micronutrients. Second, SSBs contribute more calories to the diet than any other single type of food or beverage.\(^5\) Third, the evidence supporting an association between SSB intake and excess weight is stronger than for any other single foodstuff.\(^1^6\) Fourth, SSBs provide no nutritional benefit and are thus a source of only “empty” calories. Although efforts to market “functional” beverages such as vitamin water and sports drinks abound, the fructose and caloric content of these beverages place them with other SSBs in terms of prevention. Reducing SSB intake could have a measurable impact on body weight and cardiometabolic health without having any negative dietary consequences for the consumer — a “win-win” situation.\(^1^6\)

For example, a relatively simple intervention — reducing SSB consumption by roughly 25% (provided there is no metabolic compensation for SSB calories) — has been estimated to lead to a net decrease of around 37 kcal/day in adults and around 43 kcal/day in children, with a postulated reduction of roughly 3.8 lb/year in adults and 4.5 lb/year in children.\(^1^3\) This intervention would decrease the prevalence of overweight and obesity in adults from 67% to 62% and 33% to 30%, respectively; in children and adolescents, it would decrease the prevalence of overweight and obesity from 32% to 27% and 17% to 14%, respectively.\(^1^3\) While restriction studies are wanting, given that SSBs promote hypertension, hyperuricemia, gout, non-alcoholic fatty liver disease, visceral adiposity, atherogenic dyslipidemia, inflammation, insulin resistance, and the development of T2DM, a reduction in their consumption is common sense.

**PUBLIC HEALTH INITIATIVES**

Thus far, the beverage industry has lobbied against all measures aimed at limiting SSB consumption. As such, policy
changes, both at the individual and public policy levels, such as negative incentives and restriction of access, will be needed. One proposed policy—an excise tax of 1 cent/oz for SSBs—has been researched, and has the potential to both dramatically decrease SSB consumption (and thus improve overall health), as well as increase national and state revenue.  

**CONCLUSION**

Since the prevalence of obesity and related cardiometabolic diseases is escalating around the globe, identifying modifiable risk factors is essential to the abatement of their continued rise. The consumption of SSBs is one such important risk factor. A reduction in SSB consumption in place of healthier alternatives can only lead to improved overall health and reduced medical expenditures.

**REFERENCES**