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An Exploration of the Role of Sugar-Sweetened Beverage in Promoting Obesity and Health Disparities

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Abstract

Purpose of Review The mechanistic role of sugar-sweetened beverage (SSB) in the etiology of obesity is undetermined. We address whether, compared to other foods, does consumption of SSB (1) automatically lead to failure to compensate for the energy it contains? (2) fail to elicit homeostatic hormone responses? (3) promote hedonic eating through activation of the brain's reward pathways? We followed the evidence to address: (4) Would restriction of targeted marketing of SSB and other unhealthy foods to vulnerable populations decrease their prevalence of obesity?

Recent Findings The data are lacking to demonstrate that SSB consumption promotes body weight gain compared with isocaloric consumption of other beverages or foods and that this is linked to its failure to elicit adequate homeostatic hormone responses. However, more recent data have linked body weight gain to reward activation in the brain to palatable food cues and suggest that sweet tastes and SSB consumption heightens the reward response to food cues. Studies investigating the specificity of these responses have not been conducted. Nevertheless, the current data provide a biological basis to the body of evidence demonstrating that the targeted marketing (real life palatable food cues) of SSB and other unhealthy foods to vulnerable populations, including children and people of color and low socioeconomic status, is increasing their risk for obesity.

Summary While the mechanisms for the association between SSB consumption and body weight gain cannot be identified, current scientific evidence strongly suggests that proactive environmental measures to reduce exposure to palatable food cues in the form of targeting marketing will decrease the risk of obesity in vulnerable populations.

Keywords Obesity · Weight gain · Sugar-sweetened beverages · Health disparities · Targeted marketing · Palatable food cues

Introduction

The increasing prevalence of obesity has paralleled the rising intake of sugar-sweetened beverage (SSB) [1–3]. Over the past decades, SSB consumption has emerged as a contributing risk factor for metabolic diseases such as obesity, type 2 diabetes, and cardiovascular disease [4]. The Nutrition and Chronic Diseases Expert Group has concluded that evidence from prospective studies suggests a body mass index (BMI)–independent effect of SSB on incidence of type 2 diabetes and cardiovascular disease and an additional effect on adiposity [5]. Among US youth and adults, the prevalence of obesity was as high as 20.6% and 42.4%, respectively, between 2015 and 2018 [6, 7]. Already a major public health crisis, overweight or obesity are projected to affect nearly 90% of the US population by 2030 [8]. It is estimated that one-half of adults and about two-thirds of youth consume at least one SSB on a given day [9, 10], with one 12-oz can of soda alone containing more than half a day's worth of the recommended daily added sugar limit to reduce risk for obesity and other metabolic diseases [11].

While obesity has been increasing among the entire US population for decades, its prevalence and related health burdens are the greatest among racial and ethnic minorities and low-income populations [12]. Consistent with historical data, non-Hispanic Black and Hispanic American youth and adults

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are more likely to be obese than non-Hispanic White Americans [6, 7]. A recent study using nationally representative data from NHANES showed that levels of SSB consumption were also highest among these groups [13]. Understanding the etiology between SSB consumption and obesity may help address this health disparity.

The positive associations between consuming SSB and body weight gain are supported by dietary intervention studies in which subjects exhibited increased body weight when they consumed SSB with their usual ad libitum diet [14]. There are those who suggest this relationship is not unique to SSB and is simply the consequence of consuming any source of palatable energy in excess [15]. Yet, for more than 20 years, it has been generally accepted that there is a causal relationship between SSB and body weight gain that is mediated by failure of the homeostatic system to recognize the energy in SSB or beverages in general [16–18]. This results in inadequate satiation, failure to decrease consumption of other foods to compensate for the SSB energy, and weight gain. However, more recently, it is postulated that the rise of obesity is associated with an increase in food intake that is driven by activation of the hedonic system, rather than the failure of the homeostatic system [19–21]. Specifically, our hyper-palatable food environment promotes excess energy intake by activating the brain reward centers while superseding homeostatic satiety signals [22•]. This hyper-palatable food environment is inescapable, and in areas with the highest minority and low-income populations, it is paired with more limited options for healthy food choices [23, 24]. SSB may have a unique role in activating brain reward centers compared with other foods, or its role in the obesity crisis may simply involve it being one of the most available, popular, and promoted components of our hyper-palatable food environment.

In this review, we will consider both possibilities, SSB consumption elicits homeostatic failure or hedonic activation, by reviewing the evidence specific to the following questions. Compared to other foods: (1) Does consumption of SSB automatically lead to a failure to compensate for the energy it contains? (2) Does consumption of SSB fail to elicit appropriate homeostatic hormone responses? (3) Does SSB intake promote hedonic eating through activation of the brain's reward pathways? To conclude, we will follow the evidence to a final question: (4) Would restriction of targeted marketing of SSB and other unhealthy foods to children and other vulnerable populations decrease their prevalence of obesity?

Compared to Other Foods, Does Consumption of SSB Automatically Lead to a Failure to Compensate for the Energy It Contains?

It has been proposed many times that sustained SSB consumption may promote body weight gain because energy-containing beverages elicit inadequate dietary compensation [16–18, 25]. Dietary energy compensation is the adjustment of energy intake elicited by the previous ingestion of energy in the form of a beverage, snack, or meal [26]. In the context of SSB intake, a failure to compensate is the failure to adjust for the SSB energy consumed as prior snacks or with prior meals, resulting in energy intake that exceeds daily requirement. Sustained failure to compensate fully for the energy in SSB, without significant adjustments to physical activity, ultimately leads to weight gain. Support for this mechanism requires evidence that compared to other foods sustained consumption of SSB leads to weight gain and fails to elicit the appropriate responses of satiety and appetite hormones.

Epidemiological studies demonstrate a positive association between consuming SSB and body weight gain [18, 27, 28, 29••]. A meta-analysis of dietary intervention studies showed that increased sugar intake was associated with a weight increase (0.75 kg, 95% CI 0.30 to 1.19; $P = 0.001$) in adults consuming ad libitum diets [14]. This meta-analysis also showed that reduced intake of dietary sugars was associated with a decrease in body weight (0.80 kg, 95% confidence interval (CI) 0.39 to 1.21; $P < 0.001$). Of the 10 studies that showed increased body weight, nearly all provided the sugar supplementation as SSB, either solely or predominantly. Additionally, our group has conducted an NIH-funded investigation that showed that subjects gained weight in a dose-dependent manner when provided 0, 10, 17.5, or 25% of energy requirement (Ereq) as SSB to consume with their free choice ad libitum diets [30]. However, there is an important limitation of this study and of nearly all of the ad libitum studies included in the meta-analysis [14]. The limitation is that the dietary interventions were not isocaloric. Specifically, the majority of these studies compared body weight change during consumption of SSB versus consumption of water or noncaloric sweetener. Therefore, it is not possible to know if differences in weight gain were specific to the provision of SSB, or simply to the provision of kcals that were not provided to the control group.

Only one study in the meta-analysis compared high- versus low-sugar interventions that were isocaloric. In a 14-day crossover study, Raben et al. provided subjects (20 normal-weight healthy women: 9 previously obese, 11 never obese) with restricted ad libitum (up to 3584–4300 kcals/day, but consumption was restricted to only the provided study diet) high SSB/sucrose (23% Ereq sucrose, with > 50% of sucrose energy as SSB and syrup) or high complex carbohydrate (2% Ereq sucrose) diets. Both diets contained similar proportions of total carbohydrate, protein, and fat [31]. Subjects consumed more energy during consumption of the SSB/sucrose diet than the complex carbohydrate diet, resulting in a significant weight difference of 0.9 kg between arms. Since the interventions were isocaloric, it could be concluded that the weight difference was mediated by the SSB/sucrose. However, as the principal investigator of this study, Dr. Arne Astrup, noted

[32], these results could also possibly be explained by the complex carbohydrate diet containing 80% more fiber/kcal than the SSB/sucrose diet. They could also be explained by a palatability difference between the 2 diets, as the subjects reported liking the SSB/sucrose diet more than the high complex carbohydrate diet [31]. The findings could also be of limited relevance to the general population as the increases in energy intake and body weight on the SSB/sucrose diet were driven by the women who were previously obese. Other findings from the study illustrate the complexity and challenges involved in conducting clinical studies assessing energy intake and body weight change. The study included a 3rd arm in which subjects consumed a high-fat diet. Energy intake during the high-fat diet was nearly identical to that during the high-SSB/sucrose diet, and significantly higher than during the complex carbohydrate diet. Yet, body weight was 0.5 kg higher after the sucrose diet than the high-fat diet. Interpretation of these findings is further complicated by the observation that the subjects exhibited the highest increase in energy expenditure during the SSB/sucrose diet. Dr. Astrup concluded that additional trials strictly controlling macronutrient ratios and fiber content are necessary [32].

To date, there are no published studies testing the effects of consuming SSB on body weight that utilized dietary protocols in which the experimental group and the control group were provided standardized isocaloric diets in which the only variable was the sugar in the SSB. This would require a blinded intervention during which each group receives sweetened beverages and ad libitum meals formulated to ensure identical calories, macronutrients, fiber, and saturated fat (i.e., calories, carbohydrate, protein, fat, saturated fat, fiber in meals plus SSB = calories, carbohydrate, protein, fat, saturated fat, fiber in meals plus aspartame-SB), and also formulated to be comparably palatable.

Question 1 could also be answered in the affirmative by clinical dietary intervention studies demonstrating that sustained consumption of SSB promotes more weight gain than the isocaloric sugar in solid food. Surprisingly, however, this comparison has been attempted only once [25]. In a crossover study, 15 subjects consumed 450 kcal/day of jelly beans or SSB for 4 weeks each. While consuming SSB subjects gained 0.5 kg body weight, which was significant compared with baseline, but was not significantly higher than the body weight gained during jelly bean consumption (0.3 kg). While these results do not provide a definitive answer, they suggest the potential for a differential effect of SSB vs. sugar in solid food on body weight gain that could possibly be detected in longer, high-powered studies. At this time, however, unconfounded data are lacking that support the hypothesis that, compared to other foods, SSB promotes body weight gain due to failure to decrease consumption of other foods to compensate for the SSB energy.

Compared to Other Foods, Does Consumption of SSB Fail to Elicit Appropriate Homeostatic Hormone Responses?

Control of energy intake through appetite regulation is managed by homeostatic hormone responses. These responses involve coordinated changes in circulating hormone levels to either suppress energy intake (glucagon-like peptide-1 (GLP-1), gastric inhibitory polypeptide (GIP), leptin, peptide YY (PYY), pancreatic polypeptide, etc.) or promote energy intake (ghrelin) [33]. Upon being released, these hormones can act on neurons in the brain or stimulate ascending vagal pathways that lead from the gut to the brainstem [34].

Satiety hormone responses are increased and ghrelin responses are decreased by test meals that contain excess energy [35, 36]. There is evidence from a limited number of acute studies that demonstrate satiety hormone responses are lowered when the excess energy is supplied as SSB. Two crossover studies conducted in adults [37] and children [38] demonstrated that consuming SSB before/with an ad libitum test meal lowered postprandial GLP-1, GIP, or PYY compared with consuming the same test meal with milk. Compared with beverages supplemented with fruit fiber or beta-glycan, SSB consumption with a standardized breakfast lowered pancreatic polypeptide and failed to suppress ghrelin [39]. A crossover study in young women showed post-meal ghrelin concentrations were less suppressed when fructose- compared with glucose-SB were consumed [40].

Raben and Astrup have assessed the effects of sustained SSB/sucrose consumption on GLP-1 and GIP in two dietary studies. The first was the crossover study previously described [31], in which normal-weight women (9 previously obese, 11 never obese) consumed ad libitum diets, each for 2 weeks, that were high fat (46% of energy fat, 41% carbohydrate, 2% sucrose), high complex carbohydrate (28% of energy fat, 58% carbohydrate, 3% sucrose), or high SSB/sucrose (28% of energy fat, 59% carbohydrate, 23% sucrose). GLP-1 and GIP meal responses were higher after subjects consumed the high-fat diet compared with the high SSB/sucrose diet or high complex carbohydrate diet [41]. These responses however were not correlated to energy intake [31]. As previously stated, 14-day energy intake was comparable when the women consumed the high-fat diet and high SSB/sucrose diet, and was significantly lower when they consumed the high complex carbohydrate diet. There were no differences between the effects of the high SSB/sucrose diet and the high complex carbohydrate diet on the satiety hormones. However, when consuming the high SSB/sucrose diet, the women consumed 21% more energy during the feeding trial that generated the GLP-1 and GIP results than they did while consuming the high complex carbohydrate diet. In the second study, 24 healthy overweight women were randomized to consume drinks and foods sweetened with either sucrose or noncaloric sweeteners for

10 weeks [42]. The women consuming SSB/sucrose-sweetened foods had higher pre- and post-breakfast GLP-1 concentrations than the women consuming beverages and foods sweetened with noncaloric sweeteners [42]. Again, the high sugar group consumed more energy (+ 32%) than the noncaloric sweetener group during the feeding trials that tested GLP-1 response. As already stated, satiety hormone responses are increased by test meals that contain excess energy [35, 36]. It is possible the results from both these studies reflect the differences in energy intake more than the specific effects of SSB/sucrose on GLP-1 and GIP.

The strongest evidence that consumption of SSB fails to elicit homeostatic hormone responses relates specifically to the effect of fructose consumption on circulating leptin. Leptin production by adipocytes is regulated by insulin-mediated glucose metabolism [43], and ingestion of fructose does not result in increases of circulating glucose or insulin. Accordingly, 24-h plasma leptin concentrations were decreased in subjects consuming fructose-SB with meals compared with glucose- or sucrose-SB in 24-h crossover studies [40, 44] and in dietary intervention studies lasting 2 or 10 weeks [45, 46]. However, in the intervention studies, the changes in circulating leptin were not related to the changes in body weight or ad libitum energy intake [45–47]. Furthermore, since the sugar added to SSBs and other sweetened food are usually HFCS or sucrose, the effects of these two sugars on circulating leptin may be more relevant to the prevalence of obesity than the effects of fructose. Interestingly, in both our 24-h crossover study [48] and 2-week dietary intervention study [46] comparing the effects of fructose, glucose, HFCS, and sucrose on 24-h leptin levels, the effects of HFCS were more comparable to fructose and the effects of sucrose were more comparable to glucose. It is worth noting in a 10-week intervention study, subjects consuming 30% Ereq as HFCS-sweetened milk gained more than twice the weight (~ 2.3 kg, $P < 0.01$ vs. baseline, $P < 0.05$ vs. 8% Ereq HFCS-sweetened milk group) than the subjects consuming 30% Ereq as sucrose-sweetened milk (~ 1.1 kg, $P < 0.05$ vs. baseline) [49]. The same trend was observed in our 2-week study where subjects consuming 25% Ereq as HFCS-SB gained 0.8 kg ($P < 0.01$ vs. baseline), while subjects consuming 25% Ereq as sucrose-SB gained 0.4 kg ($P > 0.05$ vs. baseline) [46].

The very few studies that have investigated the effects of sustained consumption of SSB on homeostatic hormones do not provide the evidence needed to determine whether consumption of SSB fails to elicit appropriate homeostatic hormone responses compared to other foods. The evidence gap would best be filled by dietary intervention studies (e.g., isocaloric SSB versus milk) conducted in energy-balanced conditions in order to ensure that the pre- and post-assessments of homeostatic hormones are not confounded by within- and between-group differences in energy intake or body weight gain/loss [50, 51]. We also lack data demonstrating that the increases in body weight induced by SSB are linked to decreases in satiety hormones and/or increases in the appetite hormone ghrelin. Filling this gap will be challenging, because as shown by the Raben ad libitum feeding studies [41, 42], the changes in energy intake and body weight may confound the effects of SSB on homeostatic hormones. The optimal study may require 2 phases: phase 1—energy-balanced dietary intervention to assess the unconfounded effects of SSB vs. control on homeostatic hormones; phase 2—ad libitum dietary intervention to relate the changes in phase 1 homeostatic hormones to the phase 2 changes in energy intake and body weight. Additional dietary intervention studies comparing the effects of sustained consumption of sucrose and HFCS on body weight, leptin, and the other homeostatic hormones are also warranted.

Answering question 2 is also hindered by the possibility that the actions of the satiety hormones may not be reflected by their blood concentrations. For example, GLP-1 messages are received by the brain via the circulation, but this is not the only pathway [52]. GLP-1 also signals the brain though activation of the afferent neural pathways initiating in the intestine, which may be the major pathway [52]. Thus, a dietary intervention that does not affect circulating GLP-1 could, nonetheless, cause a brain and behavioral response due to GLP-1 activation of the afferent neural pathway.

Compared to Other Foods, Does SSB Intake Promote Hedonic Eating Through Activation of the Brain's Reward Pathways?

Hedonic eating, eating for pleasure in the absence of hunger or need, is driven by reward-related brain signals that can override the homeostatic signals and contribute to overeating [21]. Activation of the brain's reward system can be assessed by functional magnetic resonance imaging (fMRI). Importantly, studies utilizing fMRI, have shown that it is not greater brain reward activation to the tastes of palatable food that predicts future weight gain, but rather greater reward activation to the sight or images of palatable foods (including advertisements) and visual cues (e.g., logos, Golden Arches™) that signal impending tastes of palatable foods [53]. Both animal [54, 55] and human [56] experiments indicate that, with pairings of visual food cues that cause anticipation of palatable food tastes and the actual palatable food tastes, dopamine signaling increases in response to repetitive visual food cues (i.e., picture of chocolate milkshake), but decreases in response to repetitive food tastes (i.e., taste of chocolate milkshake). The elevated brain reward responses to palatable food cues are associated with BMI [57]. These findings are not surprising when viewed from a survival perspective. For early humans, robust reward activation to food cues, which signaled the potential availability of food, optimized survival by leading to

food procurement. However, in today's environment of inescapable food cues and highly available palatable foods, this survival mechanism is superfluous and having the opposite effect. It is undermining optimal survival by prompting eating in the absence of hunger and need, which contributes to excessive weight gain and metabolic diseases.

Cohort studies show that in addition to being positively associated with BMI [57], brain reward responses to food cues are positively associated with abdominal obesity [58], insulin resistance [59, 60], desire to eat sweet and savory foods [58], and inversely associated with physical activity [59, 61]. In prospective studies, elevated reward responses to television advertisements depicting palatable foods that are high in fats and sugars [62] or to food cues [63] predicted weight gain in adolescent girls and boys [62–64], young women [65], and adults [66] over periods ranging from 6 months to 3 years. Compared with healthy weight adults, adults with obesity do not exhibit decreased reward responses to food cues after meal consumption [57]. Likewise, compared with healthy weight children, children with obesity failed to exhibit reduced reward response to palatable food cues following meal consumption, even though both groups exhibited appropriate circulating satiety hormone responses to the meal [67•]. Additionally, children who failed to exhibit reduced reward response to palatable food cues following meal consumption exhibited the least body weight loss during a family-based behavioral treatment for obesity [68••]. Interventions that reduced reward region responses to food cues through food response training have promoted weight loss [69, 70].

In contrast to the brain reward response to palatable food cues, the repetitive tasting of palatable foods leads to decreased brain reward responses. There is evidence to suggest that this decrease is associated with body weight gain. Yokum and Stice assessed brain responses to the taste of high- versus low-fat milkshakes in healthy weight adolescents at baseline and 2–3 years later [71]. For analyses, the group was divided into those whose BMIs increased since baseline by $\geq 10\%$ ($n = 36$) and those whose BMIs changed by $\leq 2\%$ ($n = 31$). At baseline, the increased-BMI group displayed higher reward and taste processing responses to the high fat milkshake tastes than the BMI-stable group. The same tests 2–3 years later showed decreased reward and taste processing responses in the increased-BMI group and increased responses in the BMI-stable group. The authors interpret these results as suggesting that initial hyper-responsivity in taste processing regions to high fat foods increases risk of overeating a high-fat diet and that repetitive overeating of high-calorie foods produces a reduction in reward region responsivity to tastes of such foods [71]. The authors suggest that the results provide support for prior evidence suggesting that low sensitivity of reward circuitry to palatable food tastes increases risk for overeating and that this overeating may further attenuate responsivity of reward circuitry in a feedforward process [72].

Some studies have shown that the increases in the brain reward region responses to food cues are paralleled with decreased responses in the prefrontal regions of the brain that are involved with inhibitory and executive decision [73, 74••, 75]. These decreased inhibitory responses are likely associated with less motivation to devalue the pleasure of eating highly palatable food, even when faced with the prospect of unwanted weight gain. In contrast, patients who underwent laparoscopic sleeve gastrectomy surgery exhibited decreases in body weight and in reported food cravings 1 and 6 months post-surgery that were associated with the increased connectivity to the prefrontal regions in the brain in response to food cues [76]. Another recent study showed that success in a 3-month weight loss program was predicted by activity in the prefrontal cortex in response to food cues [77].

Thus, attenuated brain reward responses to repetitive palatable food tastes, heightened brain reward responses to palatable food cues, and lowered brain inhibitory responses to palatable food cues are linked to overeating and body weight gain. Stice and Burger recently summarized the evidence supporting these links and noted the other brain regions (those involved in visual processing/attention, gustatory processing, motor response, and somatosensory processing) and other factors (genetics, immediate reward bias) that may be involved in neural vulnerability to obesity [78]. Studies utilizing fMRI have greatly advanced our knowledge in this area. However, almost no prospective or sustained dietary intervention studies have been conducted that inform us about how the general food categories (beverage vs. solid, healthy versus refined/palatable) and the specific macronutrients, their subtypes, and their combinations differ in their effects on the specific brain responses that appear to be associated with weight gain. In exception, Yokum and Stice have reported that the decreased brain reward and taste processing brain responses, exhibited 2–3 years later by the adolescents who increased BMI during that time, were specific to the tastes of the high-fat versus low-fat milkshake, and were not apparent when contrasting the high-sugar versus low-sugar milkshake [71].

There is evidence that suggests that sweetness is involved in elevating responses in brain reward regions. Specifically, an initial taste of a high-sugar food increases reward region (i.e., caudate) response to anticipated intake of more of the high-sugar food compared with a gymnemic acid lozenge that blocks sweet taste receptors [79]. Interventions that reduced reward region responses to food cues through blockage of sweet taste receptor have promoted weight loss or reduced intake of high-sugar foods [70, 80••]. Results from acute ingestion fMRI studies suggest that SSB [81] or fructose-SB [82] differentially recruit reward and gustatory regions in the brain compared with fat [81], glucose [82], or noncaloric sweeteners [83–85]. Observational results showed that

habitual added sugar consumption was positively associated with brain reward region responses to food cues in healthy young adults [86••]. Another observational study showed that adolescents who habitually consumed Coke® exhibited greater reward activation and less inhibitory response to the Coke® logo than adolescents who were non-Coke® consumers [87].

In the only dietary intervention study to assess brain responses before and after 3 weeks of sustained consumption of SSB, all the responses that have been shown to be predictive of body weight gain were observed [74••]. Reward responses to the taste of the SSB were decreased after the intervention. Viewing of food cues consisting of the logo of the consumed SSB increased responses in brain regions associated with reward and decreased responses in regions associated with inhibition. Interestingly, when the subjects viewed a food cue consisting of the logo of a similar SSB, which they had not consumed during the 3-week intervention, responses were similar to that of the logo of the SSB they had consumed. Importantly, however, this study did not include a control group that consumed a calorically equivalent control food or beverage [74••].

Therefore, our third question "compared to other foods, does SSB intake promote hedonic eating through activation of the brain's reward pathways?" cannot be answered. Studies that can identify a unique role of SSB in promoting brain responses linked to weight gain have not been conducted. Indeed, as discussed above, the only prospective study to compare the effects of 2 different macronutrients on brain responses associated with weight gain demonstrated a unique role of fat compared with sugar [71]. Dietary intervention studies testing the hypothesis that sustained consumption of SSB, or any other food/macronutrient, affects reward responses in the brain to palatable food tastes and cues compared with different beverage or food/macronutrient are clearly needed. Two priority comparisons that are needed to address basic gaps in evidence are palatable unhealthy/junk food versus healthy food and high sugar versus high fat. Sugar-specific comparisons that would illuminate the roles of food form, saccharide type, and sweetness with/without energy include SSB versus other beverage (milk), SSB versus high-sugar solid food (jelly beans, taffy, and hard candy), fructose versus glucose versus HFCS versus sucrose, and sugar vs. nonnutritive sweeteners. The various combinations of macronutrients present in unhealthy high fat foods also need to be investigated: high fat with protein (fried chicken nuggets, sausage), high fat with complex carbohydrate (french fries, potato chips), and high fat with sugar (donuts, chocolate). And finally, long-term dietary intervention trials that pair diet-induced changes in reward responses to palatable food cues and tastes with changes in eating behavior and body weight are also needed. However, despite the unanswered questions about the role of SSB in promoting hedonic eating, the fMRI studies appear to have revealed another very important environmental contributor to the obesity epidemic: palatable food cues.

Would Restriction of Targeted Marketing of SSB and Other Unhealthy Foods to Children and Other Vulnerable Populations Decrease Their Prevalence of Obesity?

If elevated brain reward response to palatable food cues does promote hedonic eating, the escalation of the obesity epidemic is not surprising. Palatable food cues, in the form of direct and indirect advertising, logos, or strategic product placement at food and nonfood stores and entertainment venues, have become increasingly ever present in our lives. However, these marketing schemes are disproportionately aimed at our vulnerable populations, which include children.

While recent evidence suggests that consumption of unhealthy foods (i.e., junk food) has decreased by 10% in US children and adolescents from 2003 to 2016, the data shows that their diets remained dominated by less-healthy foods [88]. Importantly, unhealthy foods include not only SSB and other high-sugar and high-sugar/high-fat foods, but also high-fat foods that contain little or no sugar (e.g., fried chicken nuggets, sausage, bacon, French fries, potato and corn chips). The many recent studies in children that have investigated the effects of exposure to advertisements on total energy intake and/or the intake of the advertised product, or palatable food in general, provide strong evidence that advertising plays an influential role in their food intake. A 2005 study representing 10 countries showed a positive association between the number of TV advertisement/h promoting unhealthy foods and the proportion of children who were overweight [89]. In 2013, a review of the international evidence established that SSB, presweetened cereal, candy, snacks, and fast foods were the foods most often marketed to children and advertising of these foods appeared more often during children's programs than adult programs [90]. Exposure to these advertisements was linked to an increase in snacking, greater energy consumption, and lowered consumption of healthy foods [90]. Meta-analyses of the direct experimental data support these findings [91, 92••]. More recent publications provide confirmatory results [93, 94, 95•, 96] and express concerns about a variety of issues, including indirect advertising in children's movies [97•], direct advertising on YouTube [98] and Facebook [99], indirect YouTube advertising by kid influencers [100••], and youth sports sponsorship by the SSB industry [101•].

Children and adolescents have a heightened sweet taste preference [102] and prefer a sugar concentration nearly double that of a typical soda [103]. Evidence has shown that the stronger the sweet preference, the higher the intake of SSB in children and adults [104•] and that SSB intake during infancy is a predictor of future SSB intake [105]. Sweet taste preference in children is strengthened by early and frequent

exposure to sweetness [106–108]. Consistent exposure to sweet taste during infancy and early childhood development can increase the tendency to be a picky eater [109, 110] and also negatively affect children's future intake of fruits and vegetables [111, 112], and their willingness to try new foods [113, 114]. These data suggest that targeted marketing of SSB and other sweetened foods to children not only takes advantage of their innate sweet preference, but also increases the chances that the preference will persist through adulthood and that the child will become an adult consumer of SSB and other sweetened foods at the expense of healthier foods.

An fMRI investigation in children and adults showed that children had stronger activation in brain regions involved in reward while viewing unhealthy foods than adults [115]. This clearly, along with their heightened sweet taste preference [102, 103], makes children the most vulnerable targets for marketers of SSB, other sweetened foods, and unhealthy foods. This vulnerability and its effects on children's weight status have been studied for nearly 40 years [116]. This research eventually led to a set of recommendations on the marketing of food and beverages to children established by the World Health Organization in 2010 [117]. In response and in "support" of these recommendations, the International Food and Beverage Alliance Global Commitment for Responsible Marketing to Children younger than 12 years, founded by CEOs of leading food and beverage companies like Coca-Cola© and PepsiCo©, was initiated [118]. The outlined commitments that essentially prohibited the advertising of unhealthy food to children were laudable, but industry adherence to the commitments was voluntary. In 2013, a systematic review found a marked discrepancy in the evidence from scientific, peer-reviewed papers that showed high and unchanged levels of advertising directed to children compared with industry-sponsored reports that indicated high adherence to the commitments [119]. More recently, television recordings from 22 countries indicated four times more advertisements for unhealthy foods/beverages (2.4 h/day) than for healthy foods/beverages (0.6 h/day) [120••]. Compared with other times, the per hour frequency of unhealthy food/beverage advertisements was higher during the periods (5 h total) designated as the peak viewing time for children ($P < 0.001$). During children peak viewing times, the number of unhealthy food and beverage advertisements were higher in countries with industry self-regulatory programs for responsible advertising compared with countries with no policies [120••].

There are calls from international scientists and politicians [120••, 121] for remedial action in the form of statutory regulation, but very few countries have established policies restricting the marketing of unhealthy food and beverages to children [122••]. In the USA, there are no statutory regulations that limit the marketing of unhealthy foods to children [123]. The obstacles to such regulations include the economic power of the industry [124–126], prioritizing individual rights over public health outcomes [127], and the challenges of regulating advertising through digital media, which was recently described as the most significant paid-for media channel for unhealthy advertising to children [128••]. In 2019, the California Dental Association and the California Medical Association launched a campaign to highlight the soda industry's marketing strategies targeted at children and low-income and minority communities [129]. The campaign included the introduction of assembly bills restricting some of these marketing strategies, including SSB placement near the grocery store check-out stands and promotional pricing incentives used by the beverage industry to heavily subsidize discounts on SSB [130•]. Both assembly bills were withdrawn early in 2020 due to lack of legislative support. Hopefully, the current and future research demonstrating heightened biological connections between advertising (food cues in real life), sweet taste preference, and brain reward activation in children will help to convince lawmakers that it is imperative to protect our most vulnerable population from the targeted marketing of SSB and other unhealthy foods.

People of color and low socioeconomic status are considered vulnerable populations due to their high risk for obesity and obesity-related diseases [12, 131]. Beverage companies like Coca-Cola© and PepsiCo© selectively target their marketing towards people of color and of lower income, especially children [132]. In 2017, PepsiCo© and Coca-Cola© each spent more than $20 million in Black-targeted and Spanish-language youth TV advertising [132]. However, even in 2007, Black children and adolescents saw an average of 2 SSB advertisements/day on television compared with 1.3 SSB advertisement/day for White children and adolescents [133]. In 2017, White teenagers viewed 8 food-related television advertisements/day, while Black teens viewed 17/day [132]. PepsiCo© was the company that made the greatest contribution to this disparity [132]. This specific targeting is undoubtedly profitable as studies have demonstrated that Black children and adolescents preferred a more concentrated sweet taste compared with White children and adolescents [103], and that Black adolescents were more likely to be highly susceptible to advertisements than White adolescents [134••]. The targeted marketing is not confined to television and digital media. Low-income Black and Latinx neighborhoods were disproportionately exposed to a greater number of outdoor SSB advertisements such as billboards, store posters, and building murals compared with low-income White and high-income neighborhoods [135]. Retailers in New York neighborhoods with high enrollment of Supplement Nutrition Assistance Program (SNAP), the largest federal food assistance program in the USA, were over four times more likely to promote SSB marketing during SNAP benefit issuance days compared with the other days of the month [136•]. This appears to be an effective strategy as soda represents the number one most commonly purchased food item in SNAP

households [137]. On a per person basis, SNAP households consume 35 kcal/day more SSB than non-SNAP households [138]. Across all ages; 2–60+ years; Blacks, Mexican Americans, and nonMexican-Hispanics consume more SSB than non-Hispanics Whites [11]. Thus, selective targeting of these vulnerable populations for marketing of SSB appears to be an effective, but arguably unethical, business practice that likely impacts their health [139]. A spatial analysis establishing a model to estimate neighborhood-level rate of adult obesity across New York City found that intake and accessibility of SSB made more contributions to adult obesity prevalence than socioeconomic status [140]. A review investigating the influence of the food environment on overweight and obesity in children also found that availability to SSB had the strongest correlation and concluded that reducing availability and providing alternative beverages could greatly contribute to obesity prevention [141].

This targeted marketing towards people of color is not only practiced by beverage companies, but by restaurant and food companies promoting unhealthy foods such as fast food, candy, and high fat snacks [132]. Yum!© (TacoBell, Kentucky Fried Chicken (KFC), PizzaHut), Hershey©, McDonald's©, and Domino's© are some of the top food companies directly targeting Black and Hispanic youth consumers [132]. Recent NHANES data show during 2015–2018, non-Hispanic Black (21.5%) and Hispanic (18.5%) adolescents, age 12–19 years, consumed a significantly higher percentage of daily calories from fast food, compared with non-Hispanic White adolescents (14.8%) [142]. Fast food company McDonald's© utilized targeted marketing strategies specifically to bolster sales that had been negatively impacted during the coronavirus disease (COVID-19) pandemic. During October and November of 2020, McDonald's© launched celebrity collaborations with Black hip hop artist Travis Scott and Latin reggaeton singer J. Balvin, each promoting a specific meal and SSB combination that were professed to be their favorites. This marketing strategy increased quarterly sales and was so popular that product shortages and long ordering lines were reported in multiple locations across the USA [143, 144]. Meanwhile, there is a disproportionate rate of infection, severe illness, and death among people of color due to COVID-19 [145].

Adults in general do not show comparable susceptibility to unhealthy food advertisements as children [91]. A meta-analysis of 13 studies in children showed that acute exposure to unhealthy food advertisements increased their energy intake. The same meta-analysis included 7 acute studies in adults in which the overall effect was no increase in energy intake after viewing palatable food advertisements [91]. Whether this lack of effect occurs following sustained exposure is unknown as studies testing effects of repeated exposure to palatable food advertisements in adults are lacking. However, it was recently reported in an international population of 15,515 adults that consumption of SSB was positively associated with self-reported exposure to SSB marketing [146]. Interestingly, the type of SSB marketing that showed the strongest correlation to SSB consumption was via digital media (online/Internet ads; mobile app/video game; social media (e.g., Twitter, Facebook, and Snapchat)). An earlier study showed that the SSB consumption of both caregivers and their children was positively related to the caregivers' self-reported exposure to SSB advertisements [147].

Any negative health impact of targeted advertising in vulnerable populations is likely compounded when the food environment provides limited access to healthy foods and by healthy foods costing more than unhealthy foods [148]. About 2 million American households live more than a mile from a supermarket and do not own or have access to a car for transportation [149]. Compared with middle-income neighborhoods and White neighborhoods, low-income as well as Black and Hispanic neighborhoods are less likely to have access to large supermarkets offering high-quality affordable foods [23, 24]. A recent study examining the effect of SSB affordability on consumers' purchasing behavior and weight-related health outcomes found that SSB affordability is a major influence of purchasing behavior and is significantly associated with the prevalence of both overweight and obesity [150•].

Overall, evidence suggests that targeted marketing of SSB and other unhealthy foods to vulnerable populations, in the form of advertisements, and product availability, placement, and pricing, is taking advantage of their responsiveness to food cues, their preference for sweetness, their more limited access to healthy foods, and their need to purchase the most affordable foods. There is evidence from mathematical models that suggests that reducing advertising of unhealthy foods and beverages would lower obesity risk in children. Mytton et al. recently concluded that restricting TV advertising of food and beverage high in sugar, fat, and salt between the hours of 05:30 and 21:00 in the UK would reduce the prevalence of overweight and obesity in children by 3.6% and 4.6%, respectively [151]. Veerman et al. reported that between one in seven to one in three obese American children might not have been obese in the absence of TV advertising for unhealthy foods [152]. A model from Magnus et al. showed that restricting TV advertising of high-sugar and high-fat foods could be one of the most cost-effective interventions for preventing childhood obesity [153]. Research evidence to support these models is lacking but may eventually be obtained from Chile. In 2016 Chile implemented laws, which were finalized in 2019, that prohibits the marketing of unhealthy foods to children across all media. Additionally, the laws ban the use of a wide range of child marketing techniques, including brand characters and mascots that appeal to children; tie-ins in movie, cartoons, interactive games, applications, and contests; use of child voices and actors; and offering of premiums such as toys, accessories, or stickers [122••].

Conclusion

Questions 1–3 cannot be definitively answered and the mechanism for the association between SSB consumption and body weight gain remains unknown. Unconfounded data demonstrating that SSB consumption promotes ad libitum energy intake and body weight gain compared with an isocaloric control food or beverage are lacking. There is also a lack of data demonstrating that sustained SSB consumption fails to elicit appropriate responses of homeostatic hormones. fMRI studies suggest that heightened responses to palatable food cues in the reward regions of the brain lead to increased energy intake and to body weight gain. While there are data to suggest that SSB consumption may heighten the reward response to palatable food cues, the relative strength of this response compared with other palatable foods is unknown. SSB may be the food most able to activate reward response to palatable food cues and promote hedonic eating. On the other hand, many or all palatable foods may have this capacity, and some, i.e., high fat foods [71], may prove to have even more. SSB's association with obesity in population studies may simply reflect it being the most marketed, consumed, and homogeneous of the unhealthy food categories.

Not currently knowing the specificity of the SSB and palatable food cue response does not undermine the importance of knowing there is a biological connection between palatable food cues and hedonic eating that could be a key contributor to the obesity crises. Nor does it prevent us from acting upon this knowledge. It is difficult to avoid passive exposure to palatable food cues in a food environment carefully crafted to make "blissful" combinations of sweet, fat, and salty tastes available at any time and place [154]. The challenge of avoiding palatable food cue exposure, however, is greatly compounded by the marketing of unhealthy foods through all possible media. Advertising of SSB and other unhealthy foods takes advantage of the relationship between palatable food cue-driven reward activation and food seeking. Targeted marketing of SSB and other unhealthy foods to children and adolescents also takes advantage of their heightened susceptibility to advertisements and greater preference for sweetness. Targeted marketing of SSB and other unhealthy foods to people of color and low socioeconomic status takes advantage of their more limited options for healthy food choices and their need for product affordability, while ignoring their greater risk for obesity and obesity-related diseases. Therefore, in response to question 4, we echo others [120••, 155••] in suggesting that proactive environmental measures to decrease exposure to palatable food cues, particularly in the form of targeted marketing to our vulnerable populations, is a necessary strategy for slowing the obesity epidemic.

Compliance with Ethical Standards

Conflict of Interest The efforts of Drs. Sigala and Stanhope on this review were supported by NIH grants R01HL137716-03S1 and R01HL137716, respectively.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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