beverages

MDPI

Review

Childhood Obesity and Its Physiological Association with Sugar-Sweetened, Free-Sugar Juice, and Artificially Sweetened Beverages

Meryem El Ouardi †, Guillem Garcia-Llorens *,† and Victoria Valls-Belles *

Predepartmental Unit of Medicine, Physiology Section, Faculty of Health Sciences, Universitat Jaume I, 12071 Castelló de la Plana, Spain; elouardi@uji.es

* Correspondence: gllorens@uji.es (G.G.-L.); vallsv@uji.es (V.V.-B.)

† These authors contributed equally to this work.

Abstract

Childhood obesity represents a growing global public health crisis, strongly driven by the widespread consumption of sugar-sweetened beverages (SSBs) and, increasingly, artificially sweetened beverages (ASBs). SSB intake drives excessive calorie consumption, reduces satiety, and disrupts hormones, leading to metabolic dysfunction such as insulin resistance and type 2 diabetes. Despite some regional declines, global consumption of SSBs remains high, with persistent socioeconomic disparities. Concurrently, ASBs, marketed as healthier alternatives, pose emerging metabolic and behavioral risks, such as gut microbiota disruption and altered appetite regulation, raising concerns about their long-term safety. Both beverage types displace nutritionally balanced food options in children's diets and foster enduring preferences for sweetness, exacerbating poor dietary quality. Public health interventions targeting SSB reduction have demonstrated modest success; however, rising ASB use complicates prevention strategies. Effective mitigation of childhood obesity requires comprehensive approaches that emphasize reducing all sweetened beverage consumption, promoting water and whole-food hydration, and addressing the behavioral and environmental factors underlying unhealthy beverage choices to improve lifelong health outcomes.

Keywords: sugar-sweetened beverages (SSBs); artificially sweetened beverages (ASBs); childhood obesity; type 2 diabetes; public health; physiological disruption

Academic Editor: Edgar Chambers IV

Received: 9 July 2025
Revised: 9 September 2025
Accepted: 11 September 2025
Published: 19 September 2025

Citation: El Ouardi, M.; Garcia-Llorens, G.; Valls-Belles, V. Childhood Obesity and Its Physiological Association with Sugar-Sweetened, Free-Sugar Juice, and Artificially Sweetened Beverages. *Beverages* **2025**, *11*, 137. https://doi.org/10.3390/beverages11050137

1. Introduction

Childhood obesity has become a global public health challenge, with increasing prevalence over the past few decades (WHO, 2020). According to the World Health Organization (WHO), over 39 million children under the age of five were overweight or obese in 2022, underscoring the urgency of addressing this issue. The WHO has identified sugar-sweetened beverages (SSBs) as a major contributor to this epidemic due to their high caloric content and lack of essential nutrients [1]. High consumption of SSBs has been linked to several adverse health outcomes, including type 2 diabetes, hypertension, and cardiovascular diseases, all of which share obesity as a common risk factor [2,3].

The etiology of childhood obesity is complex, encompassing genetic, behavioral, and, environmental factors [4]. Among these, dietary patterns, specifically the intake of SSBs and artificially sweetened beverages (ASBs), are regarded as significant contributors to excessive weight gain in children [5,6]. Research indicates that SSB consumption is associated

with decreased satiety and incomplete energy compensation, resulting in increased total caloric intake [7]. In addition to their high caloric content, sugar-sweetened and artificially sweetened beverages contribute to nutritional imbalance in children by displacing healthier dietary options. This is associated with a reduced intake of essential nutrients such as calcium, vitamin D, and fiber, which are critical for healthy growth and development [8]. Although marketed as healthier, artificially sweetened beverages are linked to adiposity and poor dietary patterns, suggesting they may disrupt appetite regulation and fail to prevent obesity [9]. Additional evidence shows that both types of beverages are linked to long-term metabolic risks, including insulin resistance, type 2 diabetes, hypertension, and metabolic syndrome (Figure 1). Substituting water for these drinks offers measurable improvements in weight and metabolic profiles [10].

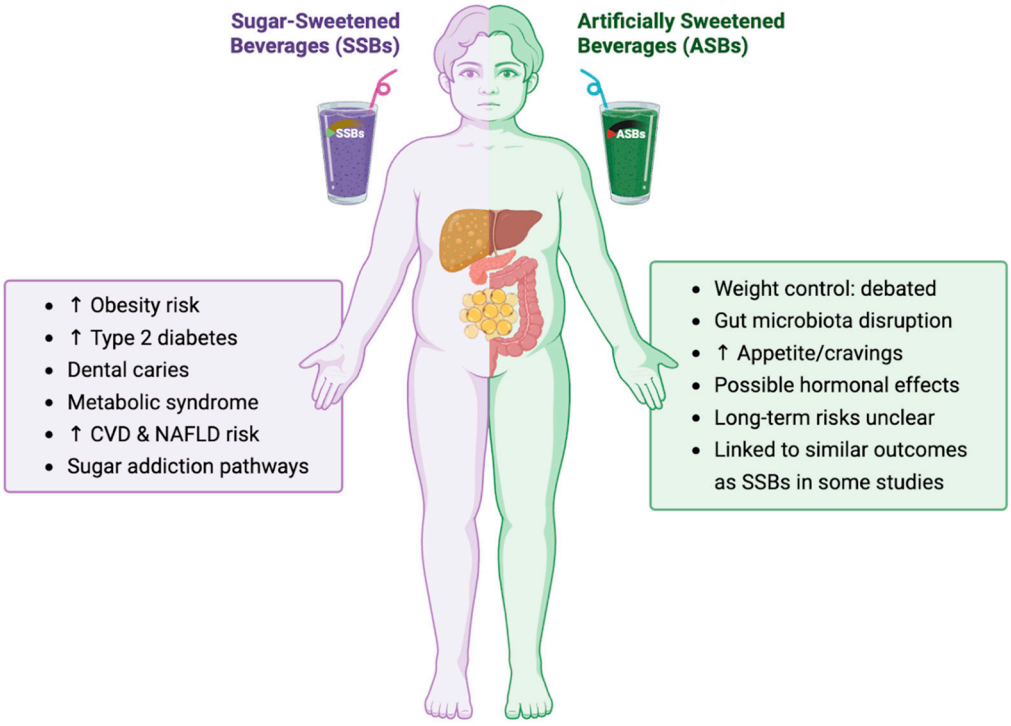

Figure 1. Health impacts associated with sugar-sweetened beverages (SSBs) and artificially sweetened beverages (ASBs). CVD (cardiovascular disease) and NAFLD (nonalcoholic fatty liver disease).

Furthermore, studies have shown that early exposure to SSBs is associated with higher body mass index (BMI) z-scores in children as young as 2 to 5 years old, indicating that the negative impact of these beverages begins early in life. Public health interventions, such as reducing access to SSBs and promoting healthier beverage choices, have shown moderate success in decreasing consumption and curbing unhealthy weight gain among children [11].

These findings emphasize the critical importance of minimizing sweetened beverage intake in children's diets, not only to preserve overall nutritional quality but also to reduce the risk of excessive calorie consumption, poor dietary habits, and the development of obesity-related health issues such as insulin resistance, type 2 diabetes, and metabolic syndrome. Early dietary interventions that limit sugary drink consumption can contribute significantly to healthier growth trajectories and long-term well-being.

2. Methods

This article is a narrative review. To improve transparency, we briefly summarize our approach. We searched PubMed, Web of Science, and Scopus for English-language publi-

cations between 2000 and January 2025 using combinations of terms such as "childhood obesity," "sugar-sweetened beverages," "artificially sweetened beverages," "fruit juice," "free sugar," and "metabolic risk." Reference lists of relevant reviews and meta-analyses were also screened.

Priority was given to higher levels of evidence. Randomized controlled trials (RCTs) were emphasized as the strongest source for causal inference. Prospective cohort studies were included to capture long-term associations, but their findings were interpreted with greater caution due to potential confounding and reverse causality. Cross-sectional studies were considered less robust and were used primarily for hypothesis generation. Mechanistic and animal studies were incorporated selectively to clarify potential biological pathways but were not weighted equally with human data.

Because definitions of sugar-sweetened beverages (SSBs) vary across studies, particular attention was paid to whether 100% fruit juice and juice-based drinks were included or excluded. In line with the World Health Organization, we consider fruit juice a source of "free sugars," and we highlight inconsistencies in its classification across the literature.

Articles were excluded if they were non-peer-reviewed, focused solely on adults without relevance to pediatric populations, or were duplicates. Evidence was synthesized to highlight consistencies, uncertainties, and sources of heterogeneity across study designs. Finally, throughout this review, we use "children" to refer to individuals aged 2–11 years and "adolescents" for those aged 12–19 years, consistent with WHO/CDC definitions.

3. Global Trends on SSBs and ASBs Intake

The consumption of sugar-sweetened beverages and artificially sweetened beverages continues to be prevalent among children and adolescents worldwide, posing significant nutritional and public health concerns. Despite some regional decreases, particularly in high-income countries, global intake remains high. A large-scale systematic review, spanning 43 studies across 15 countries, found that SSB intake among children and adolescents remained relatively stable over three decades in most regions, with the United States showing the most significant decline only after the early 2000s [12]. Even with observed declines, SSBs remain the leading source of added sugars in the diets of U.S. children and adolescents. Recent data show that sweetened beverages provide 12.6–14.3% of daily calories in children and adolescents, exceeding the <10% recommended by dietary guidelines [13]. These trends have improved slightly over the past two decades, but disparities persist by race, income, and age. For instance, over one-third of U.S. adolescents still consume excessive added sugars, primarily from beverages, with older teens (12–19 years) and children/adolescents showing the highest prevalence [14]. Additionally, while soda consumption has declined somewhat, other sweetened beverage categories, like sports drinks, flavored milks, and energy drinks, have either remained stable or increased, especially among children/adolescents from low-income households and certain ethnic minorities [15]. In the UK, similar disparities were observed over time, with adolescents from lower socioeconomic backgrounds maintaining higher SSB consumption despite national-level reductions [16]. Daily consumption of sugar-sweetened beverages among European adolescents has significantly declined between 2002 and 2018 across all 21 surveyed countries [17]. Despite this overall decline, socioeconomic disparities persist, adolescents from lower-income families continue to report higher levels of daily consumption, especially in Eastern Europe, where rates ranged from 5.1% to 28.1% in 2018 [17]. Beyond national trends, behavioral and environmental factors also strongly shape consumption. A large Brazilian cohort study found that adolescents with habits like high screen time, snacking on ultra-processed foods, and frequent purchases from school cafeterias were significantly more likely to consume SSBs daily [18]. Similarly, adolescents reporting loneliness and emo-

tional distress tend to crave SSBs more frequently, suggesting a psychological component to consumption behavior [19].

In the United States, approximately 13.3% of adolescents consume artificially sweetened beverages on a given day, according to nationally representative dietary data from the National Health and Nutrition Examination Survey (NHANES), with stable rates in recent years and higher intake observed among older adolescents and those with higher household income or obesity-related conditions [20].

3.1. Juices as Free Sugars

In line with WHO and SACN definitions [21,22], sugars naturally present in fruit juices are classified as free sugars, because the juicing process removes cellular structure and fiber, making their metabolic effects similar to added sugars. A standard 240 mL serving of apple or grape juice contains 24–28 g of free sugars, comparable to or exceeding the sugar content of cola. In contrast, whole fruit provides the same natural sugars packaged with fiber and micronutrients, slowing absorption, improving satiety, and reducing glycemic load. Epidemiological evidence shows that frequent fruit juice consumption in children is linked to higher BMI z-scores and increased risk of dental caries, whereas whole fruit intake is consistently protective. Public health guidance should therefore clearly distinguish between whole fruit (beneficial) and fruit juice (free sugars), grouping juice alongside sugar-sweetened beverages for obesity prevention.

3.2. SSBs and Childhood Obesity

Sugar-sweetened beverages, including sodas, fruit drinks with added sugars, and sports drinks, are a major contributor of empty calories in children's diets and a well-established factor in childhood obesity [23]. Numerous systematic reviews and meta-analyses confirm a strong, dose-dependent association between SSB intake and increased body mass index in children and adolescents across diverse populations [1,12,24]. A landmark randomized controlled trial demonstrated that children consuming SSBs experienced significantly greater weight gain and fat accumulation compared to those given sugar-free alternatives [25]. Another longitudinal study reported that each additional daily intake of 237 mL of SSB was associated with a mean increase of 0.050 units in BMI z-score during growth [26].

Globally, rising SSB consumption has paralleled the increase in pediatric obesity, particularly in low- and middle-income countries [27]. While public health campaigns have contributed to modest reductions in SSB intake in some high-income settings, SSBs remain a primary source of added sugars in children's diets [23]. Observational studies further suggest that early, habitual SSB intake can shape long-term dietary patterns and elevate the risk of obesity-related metabolic complications well into adulthood [19,28]. These consistent findings position SSB reduction as a top priority for global efforts to combat pediatric obesity.

Although the overall body of evidence strongly supports a link between SSB consumption and obesity, a review of systematic literature reviews found some heterogeneity: most analyses reported a positive association, but a minority did not [23]. This inconsistency highlights variability in study designs and populations but does not undermine the general conclusion that reducing SSBs is critical for obesity prevention. These discrepancies likely reflect methodological differences across reviews, such as reliance on short-term randomized trials versus longitudinal cohort studies, heterogeneity in SSB definitions, and limitations of dietary assessment tools. Differences in energy intake adjustment and the potential influence of funding sources may also have contributed to the divergent conclusions.

While sugar-sweetened beverages are defined by the presence of added sugars, it is important to recognize that many fruit juices and juice-based drinks contain equal or even higher amounts of free sugar than standard soft drinks. Common juices such as apple, white grape, and pear juice are particularly high in naturally occurring fructose and glucose, which metabolically behave similarly to added sugars due to the lack of fiber and high glycemic load [29,30]. Despite being marketed as "100% juice," many commercial products are sweetened primarily with these high-fructose juices to enhance sweetness without the appearance of added sugars, creating a misleading health halo effect [31]. For example, 240 mL of apple juice typically contains 24–28 g of sugar, comparable to or exceeding that found in cola, and offers minimal fiber or satiety value [25].

Several studies and expert bodies, including the WHO and UK Scientific Advisory Committee on Nutrition (SACN), now classify fruit juice sugars as "free sugars", recognizing their similar contribution to metabolic risk [32,33]. While orange juice may offer some micronutrients such as vitamin C or folate, apple and grape juices contain little beyond sugar, making their health benefits marginal compared to whole fruits. Importantly, epidemiological evidence links frequent fruit juice consumption, particularly in young children, to excess calorie intake, weight gain, and dental caries [25,32].

Therefore, in the context of childhood obesity, it is essential to expand the definition of sugar-containing beverages to include both traditional SSBs and high-sugar fruit juices, especially those consumed in large volumes or presented as "natural" alternatives. Public health messaging should reflect this broader classification to better guide parents, caregivers, and policymakers.

3.3. ASBs: A Risky Alternative?

Artificially sweetened beverages contain non-nutritive sweeteners such as aspartame and sucralose and are often marketed as healthier alternatives to sugar-sweetened beverages. They have gained popularity, especially among adolescents aiming to manage weight. Some studies suggest that substituting SSBs with ASBs may reduce caloric intake and assist with weight management in the short term [33–35]. Overall, randomized controlled trials consistently indicate that ASBs have neutral to modestly beneficial effects on body weight compared with SSBs, supporting their short-term utility in reducing calorie intake. However, their long-term metabolic effects remain controversial.

Evidence suggests that ASBs may influence appetite regulation and reinforce a preference for sweet flavors, which could contribute to greater energy intake from other dietary sources [36,37]. Emerging research has also associated ASB consumption with alterations in gut microbiota, which may in turn affect metabolic health and obesity risk [38]. In addition, several cohort studies have reported associations between long-term ASB intake and higher incidence of metabolic syndrome and type 2 diabetes in children and adolescents [34,39]. A systematic review similarly identified a correlation between artificially sweetened soda consumption and obesity, raising questions about their proposed benefits [40].

In the United Kingdom and broader European context, longitudinal data from Germany show that approximately 23% of adolescents consume sweetened beverages daily, including ASBs. This intake often coincides with other unhealthy lifestyle behaviors such as smoking, high screen time, and low fruit and vegetable consumption, suggesting that ASBs are part of a broader pattern of poor dietary habits [41]. Notably, adolescents tend not to replace SSBs with ASBs, but consume both concurrently, potentially compounding cardiometabolic risks.

Recent reviews support these concerns. Although ASBs may contribute to short-term caloric reduction, they have also been linked to insulin resistance, altered gut microbiota, and increased cravings for sweet foods [42]. Similarly, it has been reported that high

ASB consumption is associated with elevated risks of cardiovascular events and all-cause mortality [42]. The difficulty of forming evidence-based intake guidelines for non-sugar sweeteners has been highlighted, due to conflicting findings and the absence of long-term randomized controlled trials. In mice, long-term consumption of ASBs, containing acesulfame potassium (Ace-K), aspartame, sucralose, saccharin, or steviol glycosides, did not induce adverse metabolic effects, whether on a regular or high-fat diet. However, the steviol glycoside improves insulin sensitivity and alters the intestinal microbiota [43].

Although ASBs are commonly grouped based on their shared effects, each has a unique chemical structure that influences its metabolism and differentially modulates mechanisms mediated by the sweet taste receptor, gut hormones, brain activation, the microbiota, and appetitive sensations, necessitating further research to elucidate the specific impact of each ASB on appetite, food intake, and body mass index [44].

Overall, the growing body of evidence suggests that ASBs may not provide meaningful metabolic or dietary advantages and may instead serve as markers of unhealthy behavioral patterns in children/adolescents. Public health strategies should focus on reducing overall sweetened beverage consumption, regardless of whether they are sugar or artificially sweetened, and address the behavioral and environmental factors driving their widespread intake.

4. Comparison of SSBs vs. ASBs

From a nutritional standpoint, sugar-sweetened beverages and artificially sweetened beverages differ substantially in their composition and metabolic effects. SSBs, including sodas, sweetened teas, and fruit drinks, are typically sweetened with sucrose or high-fructose corn syrup (HFCS), both of which provide substantial energy without accompanying micronutrients or fiber. A key concern identified in laboratory-based analyses is the high and variable fructose content of many commercial SSBs [19]. Several beverages, particularly those sweetened with HFCS, were found to contain fructose concentrations exceeding the expected 55% of total sugar, with some as high as 65%. This has important metabolic implications, as fructose is primarily metabolized in the liver, where excessive intake can drive hepatic lipogenesis, dyslipidemia, insulin resistance, and elevated uric acid levels, risk factors for obesity, fatty liver disease, and cardiometabolic complications [45]. However, it is important to recognize that similar metabolic concerns extend to beverages containing high levels of free (naturally occurring) sugars, such as fruit juices and juice-based drinks. While often marketed as "natural" or "healthier," juices like apple, grape, and pear juice frequently contain equal or even greater total sugar content than conventional sodas. These sugars, primarily free fructose and glucose, act biochemically similar to added sugars when consumed in liquid form and in the absence of fiber. Leading health authorities, including the WHO and SACN, have thus categorized sugars from fruit juice as "free sugars," emphasizing that their impact on metabolism is comparable to that of added sugars. Nevertheless, studies vary in whether 100% fruit juice is classified as a sugar-sweetened beverage (SSB), which complicates direct comparisons across the literature and contributes to heterogeneity in findings [46]. Yet, public discourse often overlooks these beverages, creating a misleading dichotomy between "natural" and "added" sugar sources.

In contrast, ASBs contain non-nutritive sweeteners such as aspartame, sucralose, or stevia, formulated to deliver sweetness without significant caloric or carbohydrate content. Although often perceived as healthier alternatives, ASBs may still exert metabolic effects. Emerging evidence suggests that certain non-nutritive sweeteners may influence gut microbiota composition, glucose metabolism, and reward signaling in the brain, potentially altering hunger and satiety regulation. Furthermore, replacing SSBs with ASBs does not necessarily constitute a nutritionally superior choice, as both categories lack essential

nutrients and may reinforce habitual preference for sweet-tasting foods, regardless of calorie content [19].

Indeed, Table 1 provides a comparative overview of common SSBs and ASBs, underscoring differences in caloric load, sugar composition, including the fructose percentage, and types of sweeteners used. These findings highlight the need for more nuanced dietary guidance that goes beyond sugar content alone, focusing instead on overall diet quality and the broader metabolic consequences of habitual sweetened beverage intake.

Regarding appetite regulation, a randomized, double-blind, crossover fMRI study in 75 fasted adults demonstrated that sucralose significantly increased medial hypothalamic blood flow compared to sucrose and water, an effect associated with greater subjective hunger. Sucralose also enhanced functional connectivity between the hypothalamus and brain regions involved in motivation and somatosensory processing, suggesting that non-caloric sweeteners may influence key mechanisms of central appetite regulation [47].

Table 1. Nutritional content of selected sugar-sweetened beverages (SSBs) and artificially sweetened beverages (ASBs). Values are shown per standard 12 oz (355 mL) serving, with equivalent per-100 mL values provided for international comparability. Data are based on nutrient composition data from USDA FoodData Central and manufacturer SmartLabel™ disclosures, supplemented by FDA serving-size standards, ensuring values are traceable to primary sources. Example products are brand-agnostic (e.g., "cola-type soda," "fruit punch-type drink") to avoid commercial bias [48–50].

Beverage Type	Example	Sweetener(s)	Energy (kcal) [2]	Total Sugars (g) [2]	Added Sugars (g) [2,3]	Total Carbohydrates (g) [2]
SSB	Cola-type soda	High-fructose corn syrup	140/39	39/11	39/11	39/11
SSB	Fruit punch–type drink	Sucrose, HFCS [1]	150/42	35/10	35/10	38/11
SSB	Orange juice (100% juice)	Fructose/glucose	160/45	36/10	0/0	37/10
ASB	Diet cola	Aspartame	0/0	0/0	0/0	0/0
ASB	Zero-sugar lemonade	Sucralose, Acesulfame-K	5/1	0/0	0/0	1/<0.5

[1] High-Fructose Corn Syrup. [2] Energy and nutrient values are per 12 oz (355 mL) serving, with per-100 mL equivalents for comparability. [3] Added sugars are those incorporated during processing/preparation (e.g., sucrose, HFCS); free sugars follow the WHO/SACN definition, including sugars naturally present in fruit juice.

Although much of the literature supports a positive association between sugar-sweetened beverage (SSB) consumption and adverse health outcomes in children, several recent systematic reviews and prospective cohort studies have reported null or inconsistent findings, particularly when confounding factors such as total dietary intake, physical activity, and reverse causality are considered. For example, a 2024 systematic review and meta-analysis by Espinosa et al. found that randomized controlled trials (RCTs) showed modest reductions in BMI when children consumed non-nutritive sweeteners (NNS) instead of sugar, whereas prospective cohort studies did not demonstrate statistically significant associations between NNS intake and BMI in children/adolescents populations [51]. In the case of 100% fruit juice, multiple cohort studies similarly reported no consistent association with weight gain in preschoolers [52,53], while a recent umbrella review concluded that fruit juice consumption was associated with a very small increase in BMI z-score among children ($\beta \approx 0.03$ per 240 mL/day). In contrast, RCTs in adults reported no significant effects [54]. Although these differences were detectable in large samples, the observed changes in children are of limited clinical relevance at the individual level.

The included studies showed moderate methodological rigor, with considerable heterogeneity and potential residual confounding, limiting the strength of the conclusions. Observational cohorts linking artificially sweetened beverage (ASB) intake to obesity, type 2 diabetes, or cardiometabolic risk should therefore be interpreted with caution, as these associations may reflect confounding (e.g., diet quality, pre-existing conditions) or reverse causality (e.g., weight gain prompting a switch from SSBs to ASBs). These findings suggest that the context in which these beverages are consumed, such as total energy intake, dietary pattern, and physical activity, may moderate their health impact, underscoring the

importance of a nuanced and comprehensive approach in evaluating the role of both SSBs and ASBs in pediatric nutrition.

Taken together, discrepancies across studies can largely be explained by heterogeneity in study design (short-term RCTs versus multi-year cohort studies), population characteristics (age, baseline adiposity, socio-behavioral context), and by differences in how studies account for key lifestyle and dietary factors such as total energy intake, physical activity, and weight-control behaviors.

5. Innate and Modifiable Nature of Sweet Preference

Human preference for sweetness is innate, emerging from birth and shaped by both biological and environmental factors. From an evolutionary perspective, an affinity for sweet tastes likely served an adaptive function by guiding infants toward energy-rich, safe foods and away from bitter or toxic substances. Colostrum and breast milk are naturally sweet, due to the presence of lactose and various oligosaccharides, which contribute to early flavor learning and hedonic acceptance of sweet tastes [55].

While repeated exposure to sweet-tasting beverages, whether sugar-sweetened or artificially sweetened, can reinforce or maintain sweet preferences, these exposures do not create the preference. Numerous studies in developmental nutrition have demonstrated that children, even those who have never consumed SSBs or artificially sweetened beverages, still exhibit a strong liking for sweetness, suggesting that sweet preference is biologically driven, and subsequently modulated, but not determined, by dietary exposure [56]. Beyond these biological foundations, recent longitudinal and experimental evidence shows that short-term changes in what children eat or drink do not reset their built-in liking for sweetness. In the OPALINE birth cohort, it was demonstrated that infants' liking for sweet taste was already detectable at three months of age and remained stable through the first year of life, even as exposure to sweet tastes increased markedly during the introduction of complementary foods. These findings suggest that early exposure to dietary sweetness does not recalibrate innate sweet preferences during infancy [19]. Consistent with these findings, a randomized controlled trial in preschoolers revealed that a two-week period of daily consumption of either a sucrose-sweetened beverage or an iso-palatable sour drink did not alter the sucrose concentration preferred at a two-month follow-up, confirming that brief dietary interventions fail to modify intrinsic preference for sweet flavors [56]. Recognizing this distinction is important for developing realistic public health strategies. Rather than attempting to eliminate sweetness from children's diets altogether, efforts should aim to shift the source and frequency of sweet exposures, emphasizing nutrient-rich, naturally sweet foods over high-calorie, low-nutrient beverages [57].

Sweet preference is a robust feature of childhood, but it is not merely a behavioral habit, it rests on physiological mechanisms that are being progressively elucidated. In many settings, sweetened beverages constitute a non-negligible source of free sugars for children/adolescents [23], with physiological consequences: they bypass normal gastric-volume satiety, leading to incomplete energy compensation at subsequent meals [58]. Beneath this behavioral phenotype lies a coordinated gut–brain–liver–microbiome network: intestinal sweet-taste chemosensing and incretin release, hepatic hepatokines that signal fructose load, cortico-striatal reward adaptations that shape cue-driven intake, fructose-to-uric-acid biochemistry fueling de novo lipogenesis, developmental changes (including GLUT5 maturation and epigenetic programming) that create windows of susceptibility, hypothalamic gliosis that blunts leptin/insulin signaling, and microbiota-mediated effects on metabolism and cognition.

5.1. Hepatokine FGF21: A Fructose Stress Sensor

Fructose is catabolized in hepatocytes by ketohexokinase (fructokinase) without feedback inhibition, rapidly depleting ATP and increasing AMP. The ensuing bioenergetic stress activates ChREBP and induces hepatic transcription of fibroblast growth factor 21 (FGF21). In humans, oral fructose or sucrose acutely elevates circulating FGF21 within hours, and the magnitude of this response tracks habitual sweet intake and preference [59]. These dynamics suggest that repeated, modest daily exposures, such as sugar-sweetened beverages or fructose-rich juices, may sustain FGF21 signaling over time, particularly in adolescents with greater adiposity [59,60]. In animal models, chronic fructose exposure drives sustained FGF21 release and reinforces sweet-seeking behavior through reward pathways. Clinically, elevated circulating FGF21 concentrations are consistently associated with higher BMI z-scores, and in adolescents the anticipation of soft-drink intake is accompanied by heightened ventral-striatal activation [60,61]. Thus, chronically higher FGF21 acts as an endocrine early-warning signal that predicts deterioration of glycemic control and incident type 2 diabetes, but current evidence supports a predictive rather than causal role [62].

5.2. Intestinal Sweet-Taste Receptors (T1R2/T1R3) and Incretin–SGLT1 Coupling

Enteroendocrine cells express the T1R2/T1R3 sweet-taste receptor and the G-protein α-gustducin. This chemosensory pathway detects luminal sweetness and stimulates glucagon-like peptide-1 (GLP-1) secretion [63]. In parallel, gut T1R3/α-gustducin signaling upregulates SGLT1 (Na^+–glucose cotransporter 1) in enterocytes, increasing apical glucose transport capacity and thereby coupling sugar detection to absorptive readiness [64]. In humans, sucralose alone does not reliably stimulate GLP-1 or GIP, nor does it slow gastric emptying, whereas caloric sugars do [65]. When sweetness is decoupled from calories, central appetite signals can diverge from peripheral glycaemia. In a large randomized study, consumption of a sucralose-sweetened beverage led to a greater increase in medial-hypothalamic blood flow and subjective hunger compared to a sucrose-sweetened beverage, across all BMI categories [47]. Together, these findings support a "coupling/decoupling" model in which caloric sugars deliver a coherent gut–incretin–brain signal, while some non-nutritive sweeteners provide sweetness without calories, altering neuroendocrine responses, helping reconcile mixed results for artificially sweetened beverages and clarifying why high-fructose juices (fully coupled calories) can metabolically resemble SSBs.

5.3. Cortico-Striatal Reward Adaptations

Repeated sucrose exposure reduces μ-opioid and dopamine D2/3 receptor availability across striatal, accumbal, and related reward regions in animal models, as demonstrated by longitudinal PET studies. This is consistent with addiction-like neuroadaptations that bias behavior toward seeking stronger sweet cues [51]. At the transcriptional level, repeated natural rewards cause the unusually stable activity-dependent transcription factor delta FosB to accumulate in the nucleus accumbens. Overexpression of delta FosB increases sucrose intake in rodents, supporting a causal role in sweet seeking [66]. Not all sweeteners have identical neurobiological signatures: for example, stevia exposure modifies delta FosB immunoreactivity in reward circuits, and environmental enrichment can attenuate this effect, highlighting the interplay between palatability, learning context, and plasticity [67]. In summary, reward-circuit plasticity may explain why simply switching beverages does not reduce the neurobiological drive for sweetness.

5.4. Fructose, Uric Acid, and Hepatic De Novo Lipogenesis

Excess AMP, generated during fructose metabolism via ketohexokinase, is catabolized via the purine pathway to uric acid by xanthine oxidase. Intracellular uric acid acts as

a signaling metabolite that further upregulates ketohexokinase and promotes lipogenic pathways, thereby increasing de novo lipogenesis and hepatic triglyceride accumulation. Thus, high fructose exposure directly drives hepatic fat production and contributes to fatty liver disease [68]. Clinical studies show that when adolescents with fatty liver disease reduce their intake of free sugars, including those from 100% fruit juice, the amount of fat produced by the liver and stored in it goes down [69]. In short, understanding this link between fructose, uric acid, and fat production helps explain why even so-called "healthy" juices can have similar metabolic effects as sugar-sweetened drinks. This supports including them in the same category as other free-sugar beverages when giving guidance for children.

5.5. GLUT5 Maturation and Epigenetic Programming Across Early Life

Fructose absorption depends primarily on GLUT5 (SLC2A5), whose expression is low in early life and increases with postnatal maturation and luminal fructose exposure. Dietary fructose further up-regulates GLUT5 through transcriptional and post-transcriptional mechanisms, potentially creating a feed-forward loop of heightened absorptive capacity during growth spurts that may be epigenetically reinforced [70]. Pediatric biopsy data demonstrate higher duodenal GLUT5 protein expression in children with obesity compared to normal-weight peers, supporting the concept of developmental intensification of fructose handling in at-risk children [71]. However, this up-regulation is not inevitable and appears to vary across individuals, highlighting the importance of inter-individual variability in transporter expression. Parallel to transporter maturation, prenatal exposures may also influence offspring phenotype: in a large birth cohort, maternal consumption of ASBs during pregnancy was associated with higher infant BMI z-score and greater odds of overweight at 1 year, suggesting an in utero epigenetic susceptibility to sweet taste or exposure, though mechanisms remain incompletely defined [72]. Overall, developmental windows from late gestation through early childhood can magnify, but not universally determine, the metabolic impact of sweetened beverage intake.

5.6. Hypothalamic Gliosis and Appetite Signaling

Children commonly show a strong sweet preference, and the hypothalamus is the brain center that converts leptin and insulin signals into "eat" or "stop" messages. Within the hypothalamus, the mediobasal region (MBH) helps set day-to-day appetite thresholds. In pediatric MRI studies, a higher T2 signal in the MBH, interpreted as compatible with gliosis, has been observed in children with obesity, and the magnitude of this signal predicts later BMI z-score gain in at-risk children [73]. However, this T2 signal should be regarded as an associative marker of risk rather than proof of causation. Early-life studies indicate that microglia actively wire hypothalamic circuits that govern glucose control and shape orexigenic AgRP innervation during development. When these glial processes are perturbed in critical windows, the resulting networks can shift toward stronger "eat" signaling and weaker satiety, with effects that can persist into adulthood [74,75]. This brain-centered mechanism helps explain why late lifestyle advice often underperforms, and it underscores the value of prevention in childhood, particularly by limiting sweetened beverages during brain maturation.

6. Physiological Alterations Associated with SSBs and ASBs

Sugar-sweetened beverages contribute to weight gain in children through multiple physiological pathways. First, SSBs are energy-dense yet provide minimal satiety, meaning they add significant calories to the diet without reducing subsequent food intake. This poor caloric compensation is associated with positive energy balance, which is a direct contributor to weight gain. SSB intake did not significantly suppress hunger or increase

satiety in minority adolescents, supporting the idea that these beverages fail to trigger appropriate appetite-regulating responses [76]. Similarly, it has been found that energy-dense snacks and sugar-containing beverages were perceived to have similar expected satiation, yet actual consumption of SSBs led to lower fullness, further underscoring their limited impact on satiety signaling [77].

6.1. Gut Microbiota

Moreover, frequent consumption of SSBs has been shown to affect the gut microbiota, reducing microbial diversity and promoting dysbiosis, which is linked to inflammation and altered energy metabolism. It has been found that higher intake of added sugars and sugar-sweetened beverages was associated with a distinct gut microbiota composition, characterized by lower microbial diversity and shifts toward pro-inflammatory bacterial profiles. Similarly, it has been reported that SSB intake was correlated with gut microbiota alterations and changes in circulating metabolites linked to higher diabetes risk [35]. Despite concerns about the impact of non-caloric artificial sweeteners on glucose metabolism and gut microbiota, a double-blind study in humans and mice found that short-term consumption of pure saccharin at the maximum acceptable intake level did not alter glucose tolerance and absorption, or gut microbiota composition [78].

6.2. Inflammatory Effects

In parallel, another study demonstrated that SSB consumption was positively associated with elevated inflammatory markers, such as C-reactive protein (CRP) and white blood cell counts, particularly among individuals with abdominal obesity and prediabetes [20]. Their findings underscore the role of chronic inflammation as a potential mediator between high SSB intake and the progression of metabolic disorders. Collectively, these findings suggest that habitual consumption of SSBs may have long-term metabolic consequences mediated through both gut microbial pathways and systemic inflammation.

With regard to adipose tissue, a study demonstrated that repeated consumption of a commercially available diet soda sweetened with sucralose and Acesulfame-K, administered three times daily over an eight-week period, induces significant molecular alterations within this tissue, affecting immune and inflammatory signaling pathways such as NF-κB, interleukins, and interferons [79].

6.3. Hormonal Disruption and Obesity

Secondly, SSBs have a high glycemic load, rapidly increasing blood glucose and insulin levels. Thus, frequent SSB intake has been linked to hormonal imbalances that influence energy regulation and fat accumulation [80]. Repeated spikes in insulin not only promote fat storage (lipogenesis) but also contribute to the development of insulin resistance over time. Fructose, a dominant sugar in many SSBs, has been shown to induce insulin resistance and impair leptin signaling, a hormone responsible for appetite suppression [81]. Leptin resistance, in turn, promotes increased food intake and reduced energy expenditure, exacerbating obesity [19]. Supporting this, a rodent model study demonstrated that consumption of glucose led to increased expression of several hypothalamic satiety peptides, including cholecystokinin (CCK), whereas fructose consumption suppressed CCK and significantly raised circulating triglycerides [82]. This disruption in hypothalamic signaling suggests that fructose may impair central appetite regulation, increasing the risk of overeating. Additionally, sucrose and high-fructose corn syrup produced mixed effects on satiety-related genes, indicating varied metabolic consequences depending on sugar type. Similarly, some artificial sweeteners may paradoxically stimulate insulin secretion even in the absence of glucose, leading to hypoglycemia and increased hunger [39]. Consequently, habitual intake of SSBs and ASBs is associated with repeated elevations in insulin levels and suppression

of glucagon release, thereby impairing the homeostatic balance of this key metabolic axis. Beyond their lipogenic potential, evidence suggests that a physiological rise in circulating glucagon concentrations can increase hepatic mitochondrial oxidation rates by 50–75% [83]. Animal studies have reported metabolic disruptions linked to ASB consumption, yet further clinical research is needed to determine their significance in pediatric populations [84,85].

Taken together, these mechanisms explain why high consumption of sugar-sweetened beverages is strongly associated with increased adiposity in children and support public health efforts aimed at reducing their intake.

6.4. Other Pathologies

Sugar-sweetened beverage consumption has also been associated with the onset of asthma by age two, prior to the emergence of obesity-related signs in children. A prospective cohort study involving 1140 participants demonstrated that intake of SSBs was directly correlated with increased childhood asthma indicators, including physician diagnosis, elevated eosinophil levels, emergency visits, and rhinitis [54].

A study by the Interact Consortium 2013 [86] demonstrated that a daily increase of 350 mL in SSBs consumption was associated with a 22% higher risk of developing type 2 diabetes, while the association with ASBs was no longer statistically significant after adjusting for total energy intake and body mass index. However, another prospective cohort study found that consumption of $\geq$2 servings/day of both SSBs and ASBs was associated with a 41% and 11% higher risk of developing type 2 diabetes, respectively [87]. Although both studies included adjustment for BMI and total energy intake, divergent results are more likely driven by differences in exposure measurement and classification (single baseline measure or repeated cumulative assessments; increment models or high-intake categories), alongside variation in follow-up length and in the sociodemographic and metabolic profiles of the populations studied.

The consumption of SSBs and ASBs is also associated with increased total mortality. A cohort study reported that, compared to consuming less than one serving per month, individuals consuming $\geq$2 servings/day of SSBs had a 21% higher risk of total mortality, with significant associations observed for cardiovascular disease mortality (31%) and cancer mortality (16%) [88]. For ASBs, increased total mortality was observed only at the highest intake levels, and this association was limited to cardiovascular disease mortality, with no significant association found for cancer mortality.

Moreover, nonalcoholic fatty liver disease (NAFLD), a condition characterized by excessive fat accumulation in the liver unrelated to alcohol use, has been increasingly linked to the consumption of sugar-sweetened beverages [89]. These drinks, high in fructose and other added sugars, promote hepatic fat synthesis through lipogenesis and insulin resistance, key mechanisms in the development of NAFLD. Regular intake of SSBs has been associated with a higher prevalence and severity of this liver condition, especially in children and adolescents. Although artificially sweetened beverages do not contain sugar, some studies suggest they may still influence metabolic processes and liver health indirectly, possibly through alterations in gut microbiota or insulin sensitivity. While ASBs are often marketed as safer alternatives, their long-term impact on liver function remains unclear, highlighting the need for cautious consumption of both beverage types [90]. In pediatric populations, longitudinal evidence is scarce; there is a lack of long-term prospective studies on NAFLD [91], and the evidence linking ASB consumption to NAFLD remains limited [92], underscoring the need for well-designed cohorts with standardized liver endpoints.

Sugar-sweetened beverages (SSBs) are a major contributor to dental caries due to their high sugar content and acidity. The sugars in these drinks serve as a substrate for oral bacteria, which produce acids that demineralize enamel and promote cavities.

Frequent consumption, particularly between meals or in children, significantly increases risk. Globally, dental caries affects a majority of school-aged children and nearly all adults, according to the most recent WHO estimates [93]. Frequent intake of acidic beverages, including SSBs, carbonated sodas, artificially sweetened beverages (ASBs), and even natural fruit juices, is a primary risk factor for dental erosion, a chemical process that irreversibly dissolves tooth enamel independent of bacterial acids [94]. While ASBs are sugar-free, their acidity can still erode enamel and contribute to tooth wear [95]. Public health guidelines recommend limiting intake of both SSBs and ASBs, maintaining good dental hygiene, and minimizing exposure to acidic drinks to reduce the risk of dental caries and erosion [93,94].

7. Public Health Implications

The widespread consumption of sugar-sweetened beverages and artificially sweetened beverages among children presents a major public health concern, particularly as childhood obesity rates continue to rise globally. Robust evidence links high SSB intake to increased risk of obesity, insulin resistance, type 2 diabetes, inflammation, and metabolic dysfunction. This has prompted public health policies targeting SSB reduction through taxation, marketing restrictions, improved labeling, and bans in school settings. Fiscal interventions such as SSB taxation have been shown to substantially reduce purchases and intake. A systematic review and meta-analysis including 17 evaluations across multiple countries indicated that a 10% excise tax is associated with an average 10% decline in SSB purchases and intake, with sensitivity analyses suggesting reductions of up to 13.3% [96].

A well-documented case is the beverage excise tax implemented in Philadelphia in 2017. The city levied a 1.5-cent-per-ounce excise tax on sugar-sweetened beverages, equivalent to approximately 5 cents per 12–16 oz drink. Following implementation, sales of taxed beverages within the city declined significantly, while modest increases were observed in bordering areas, yielding a net reduction in overall [97]. This standardized per-ounce framing ensures consistency across studies and better reflects the fiscal mechanism underpinning observed behavioral changes.

The interventions have shown some success in lowering SSB consumption, yet the simultaneous rise in ASB intake introduces new complexities. Though ASBs may contribute to short-term reductions in caloric intake, growing concerns about their effects on appetite regulation, insulin sensitivity, and gut microbiota highlight the uncertainty of their long-term safety. The ambiguity surrounding ASBs complicates public health messaging, particularly when they are marketed as “healthier” alternatives.

In addition to the physiological burden, childhood obesity carries significant psychosocial consequences, including low self-esteem, social stigma, and impaired academic and social development [98,99]. Economically, obesity-related conditions such as type 2 diabetes and cardiovascular disease strain healthcare systems worldwide, driving up medical costs and reducing quality of life [100,101].

Evidence from several studies indicates that implementing specific school food environment policies has been shown to improve targeted dietary behaviors, thereby contributing to enhanced childhood dietary habits and overall child health. Across regions, regulation of the sale of foods and beverages within the school setting resulted in a reduction in SSB intake by 0.18 servings per day without compensatory increases in total calories, indicating real behavioral change within schools [102]. In the UK, introducing national School Food Standards reduced the amount of free sugar in secondary-school lunches by an average of 2.78 g, demonstrating that statutory nutrient standards can measurably lower sugar exposure during the school day [103]. In the USA, installing cafeteria water dispensers increased convenient access to water and was linked to small reductions in

BMI z-score (0.022 in girls and 0.025 in boys) and a lower risk of overweight, illustrating a simple, scalable substitution strategy [104].

Thus, public health strategies must move beyond substitution of SSBs with ASBs and instead promote a broader cultural shift toward water and whole-food-based hydration. Educational initiatives targeting children, parents, and schools play a critical role in reshaping dietary norms and reducing dependency on all sweetened beverages. Ultimately, tackling the environmental and behavioral drivers of excessive SSB and ASB consumption is key to reversing the childhood obesity epidemic and building lifelong healthy habits.

8. Conclusions

Childhood obesity remains a critical global health challenge, influenced by a complex interplay of dietary, behavioral, and socioeconomic factors. Among these, sugar-sweetened beverages, including sodas, fruit drinks, and juices with high free sugar content, are consistently associated with increased caloric intake, weight gain, and metabolic disturbances. However, a growing body of research highlights that not all sweetened beverages exert uniform effects, and that context matters. Artificially sweetened beverages, often considered healthier alternatives, present their own concerns. Although they do not contain calories, emerging evidence suggests possible effects on appetite regulation, gut microbiota, and long-term dietary preferences. At the same time, some studies report null or inconclusive findings, especially when confounding factors such as reverse causality, total energy intake, and physical activity are accounted for. Importantly, the preference for sweet taste is biologically driven, present from birth through exposure to lactose-rich breast milk and colostrum and is not created solely by consuming sweetened beverages. Nevertheless, frequent and early exposure to sweet beverages, whether caloric or non-caloric, can reinforce these preferences and shape long-term eating habits. Both SSBs and ASBs may displace nutrient-dense drinks like milk or water, contributing to poor dietary quality. From a public health standpoint, strategies to reduce sweetened beverage consumption have shown promise, particularly in high-income settings. Yet, global consumption remains high, with the growing ASB intake among adolescents and persistent disparities across income and education levels. Moving forward, interventions must go beyond SSB reduction alone, addressing all sources of sweet-tasting beverages, including fruit juices with high free sugar and artificially sweetened drinks.

Future efforts should emphasize the promotion of water and whole-food hydration options, supported by behavioral, environmental, and policy-based strategies that begin early in life. Reducing all forms of sweetened beverage intake, not just those with added sugars, is essential to mitigating the physical, psychological, and economic burden of pediatric obesity and fostering healthier generations worldwide.

Author Contributions: Conceptualization, G.G.-L., M.E.O. and V.V.-B.; writing—original draft preparation, G.G.-L. and M.E.O.; writing—review and editing, G.G.-L., M.E.O. and V.V.-B.; supervision, V.V.-B. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

ASBs	Artificially Sweetened Beverages
BMI	Body Mass Index
CCK	Cholecystokinin
CRP	C-Reactive Protein
CVD	Cardiovascular Disease
GIP	Glucose-Dependent Insulinotropic Polypeptide
GLP-1	Glucagon-Like Peptide-1
HFCS	High-Fructose Corn Syrup
MBH	Mediobasal Hypothalamus
NAFLD	Nonalcoholic Fatty Liver Disease
NHANES	National Health and Nutrition Examination Survey
SGLT1	Sodium-glucose cotransporter 1
SSBs	Sugar-Sweetened Beverages
T1R2	Taste Receptor Type 1 Member 2
T1R3	Taste Receptor Type 1 Member 3
WHO	World Health Organization

References

1. Malik, V.S.; Pan, A.; Willett, W.C.; Hu, F.B. Sugar-Sweetened Beverages and Weight Gain in Children and Adults: A Systematic Review and Meta-Analysis. *Am. J. Clin. Nutr.* **2013**, *98*, 1084–1102. [CrossRef]
2. Hu, F.B. Resolved: There Is Sufficient Scientific Evidence That Decreasing Sugar-sweetened Beverage Consumption Will Reduce the Prevalence of Obesity and Obesity-related Diseases. *Obes. Rev.* **2013**, *14*, 606–619. [CrossRef]
3. Johnson, R.K.; Appel, L.J.; Brands, M.; Howard, B.V.; Lefevre, M.; Lustig, R.H.; Sacks, F.; Steffen, L.M.; Wylie-Rosett, J. Dietary Sugars Intake and Cardiovascular Health: A Scientific Statement from the American Heart Association. *Circulation* **2009**, *120*, 1011–1020. [CrossRef]
4. Lobstein, T.; Jackson-Leach, R.; Moodie, M.L.; Hall, K.D.; Gortmaker, S.L.; Swinburn, B.A.; James, W.P.T.; Wang, Y.; McPherson, K. Child and Adolescent Obesity: Part of a Bigger Picture. *Lancet* **2015**, *385*, 2510–2520. [CrossRef]
5. Ludwig, D.S.; Peterson, K.E.; Gortmaker, S.L. Relation between Consumption of Sugar-Sweetened Drinks and Childhood Obesity: A Prospective, Observational Analysis. *Lancet* **2001**, *357*, 505–508. [CrossRef] [PubMed]
6. Te Morenga, L.; Mallard, S.; Mann, J. Dietary Sugars and Body Weight: Systematic Review and Meta-Analyses of Randomised Controlled Trials and Cohort Studies. *BMJ* **2012**, *346*, e7492. [CrossRef] [PubMed]
7. Calcaterra, V.; Cena, H.; Magenes, V.C.; Vincenti, A.; Comola, G.; Beretta, A.; Di Napoli, I.; Zuccotti, G. Sugar-Sweetened Beverages and Metabolic Risk in Children and Adolescents with Obesity: A Narrative Review. *Nutrients* **2023**, *15*, 702. [CrossRef]
8. Leung, C.W.; DiMatteo, S.G.; Gosliner, W.A.; Ritchie, L.D. Sugar-Sweetened Beverage and Water Intake in Relation to Diet Quality in U.S. Children. *Am. J. Prev. Med.* **2018**, *54*, 394–402. [CrossRef] [PubMed]
9. Laverty, A.A.; Magee, L.; Monteiro, C.A.; Saxena, S.; Millett, C. Sugar and Artificially Sweetened Beverage Consumption and Adiposity Changes: National Longitudinal Study. *Int. J. Behav. Nutr. Phys. Act.* **2015**, *12*, 137. [CrossRef]
10. Zhu, Y.; Olsen, S.F.; Mendola, P.; Halldorsson, T.I.; Rawal, S.; Hinkle, S.N.; Yeung, E.H.; Chavarro, J.E.; Grunnet, L.G.; Granström, C.; et al. Maternal Consumption of Artificially Sweetened Beverages during Pregnancy, and Offspring Growth through 7 Years of Age: A Prospective Cohort Study. *Int. J. Epidemiol.* **2017**, *46*, 1499–1508. [CrossRef]
11. Scharf, R.J.; DeBoer, M.D. Sugar-Sweetened Beverages and Children's Health. *Annu. Rev. Public Health* **2016**, *37*, 273–293. [CrossRef]
12. Della Corte, K.; Fife, J.; Gardner, A.; Murphy, B.L.; Kleis, L.; Della Corte, D.; Schwingshackl, L.; LeCheminant, J.D.; Buyken, A.E. World Trends in Sugar-Sweetened Beverage and Dietary Sugar Intakes in Children and Adolescents: A Systematic Review. *Nutr. Rev.* **2021**, *79*, 274–288. [CrossRef]
13. Ricciuto, L.; Fulgoni, V.L.; Gaine, P.C.; Scott, M.O.; DiFrancesco, L. Trends in Added Sugars Intake and Sources Among US Children, Adolescents, and Teens Using NHANES 2001–2018. *J. Nutr.* **2022**, *152*, 568–578. [CrossRef]
14. Park, S.; Zhao, L.; Lee, S.H.; Hamner, H.C.; Moore, L.V.; Galuska, D.A.; Blanck, H.M. Children and Adolescents in the United States with Usual High Added Sugars Intake: Characteristics, Eating Occasions, and Top Sources, 2015–2018. *Nutrients* **2023**, *15*, 274. [CrossRef]

15. Ricciuto, L.; Fulgoni, V.L.; Gaine, P.C.; Scott, M.O.; DiFrancesco, L. Intakes of Added Sugars, with a Focus on Beverages and the Associations with Micronutrient Adequacy in US Children, Adolescents, and Teens (NHANES 2003–2018). *Nutrients* **2023**, *15*, 3285. [CrossRef] [PubMed]
16. Morgan, K.; Lowthian, E.; Hawkins, J.; Hallingberg, B.; Alhumud, M.; Roberts, C.; Murphy, S.; Moore, G. Sugar-Sweetened Beverage Consumption from 1998–2017: Findings from the Health Behaviour in School-Aged Children/School Health Research Network in Wales. *PLoS ONE* **2021**, *16*, e0248847. [CrossRef]
17. Chatelan, A.; Lebacq, T.; Rouche, M.; Kelly, C.; Fismen, A.-S.; Kalman, M.; Dzielska, A.; Castetbon, K. Long-Term Trends in the Consumption of Sugary and Diet Soft Drinks among Adolescents: A Cross-National Survey in 21 European Countries. *Eur. J. Nutr.* **2022**, *61*, 2799–2813. [CrossRef]
18. Rocha, L.L.; Pessoa, M.C.; Gratão, L.H.A.; Carmo, A.S.D.; Cunha, C.D.F.; Oliveira, T.R.P.R.D.; Mendes, L.L. Health Behavior Patterns of Sugar-Sweetened Beverage Consumption among Brazilian Adolescents in a Nationally Representative School-Based Study. *PLoS ONE* **2021**, *16*, e0245203. [CrossRef]
19. Ventura, A.K.; Mennella, J.A. Innate and Learned Preferences for Sweet Taste during Childhood. *Curr. Opin. Clin. Nutr. Metab. Care* **2011**, *14*, 379–384. [CrossRef] [PubMed]
20. Lin, W.-T.; Kao, Y.-H.; Li, M.S.; Luo, T.; Lin, H.-Y.; Lee, C.-H.; Seal, D.W.; Hu, C.; Chen, L.-S.; Tseng, T.-S. Sugar-Sweetened Beverages Intake, Abdominal Obesity, and Inflammation among US Adults without and with Prediabetes—An NHANES Study. *Int. J. Environ. Res. Public. Health* **2022**, *20*, 681. [CrossRef] [PubMed]
21. World Health Organization. *Guideline: Sugars Intake for Adults and Children*; World Health Organization: Geneva, Switzerland, 2015; ISBN 978-92-4-154902-8.
22. Scientific Advisory Committee on Nutrition (SACN). *Carbohydrates and Health*; The Stationery Office: London, UK, 2015; ISBN 978-0-11-708284-7.
23. Keller, A.; Bucher Della Torre, S. Sugar-Sweetened Beverages and Obesity among Children and Adolescents: A Review of Systematic Literature Reviews. *Child. Obes.* **2015**, *11*, 338–346. [CrossRef]
24. Ruanpeng, D.; Thongprayoon, C.; Cheungpasitporn, W.; Harindhanavudhi, T. Sugar and Artificially Sweetened Beverages Linked to Obesity: A Systematic Review and Meta-Analysis. *QJM Int. J. Med.* **2017**, *110*, 513–520. [CrossRef]
25. de Ruyter, J.C.; Olthof, M.R.; Seidell, J.C.; Katan, M.B. A Trial of Sugar-Free or Sugar-Sweetened Beverages and Body Weight in Children. *N. Engl. J. Med.* **2012**, *367*, 1397–1406. [CrossRef]
26. Marshall, T.A.; Curtis, A.M.; Cavanaugh, J.E.; Warren, J.J.; Levy, S.M. Child and Adolescent Sugar-Sweetened Beverage Intakes Are Longitudinally Associated with Higher Body Mass Index z Scores in a Birth Cohort Followed 17 Years. *J. Acad. Nutr. Diet.* **2019**, *119*, 425–434. [CrossRef]
27. Bray, G.A.; Popkin, B.M. Dietary Sugar and Body Weight: Have We Reached a Crisis in the Epidemic of Obesity and Diabetes?: Health Be Damned! Pour on the Sugar. *Diabetes Care* **2014**, *37*, 950–956. [CrossRef]
28. Bleich, S.N.; Vercammen, K.A. The Negative Impact of Sugar-Sweetened Beverages on Children's Health: An Update of the Literature. *BMC Obes.* **2018**, *5*, 6. [CrossRef]
29. Vos, M.B.; Kaar, J.L.; Welsh, J.A.; Van Horn, L.V.; Feig, D.I.; Anderson, C.A.M.; Patel, M.J.; Cruz Munos, J.; Krebs, N.F.; Xanthakos, S.A.; et al. Added Sugars and Cardiovascular Disease Risk in Children: A Scientific Statement from the American Heart Association. *Circulation* **2017**, *135*, e1017–e1034. [CrossRef]
30. Pereira, M.A.; Fulgoni, V.L. Consumption of 100% Fruit Juice and Risk of Obesity and Metabolic Syndrome: Findings from the National Health and Nutrition Examination Survey 1999–2004. *J. Am. Coll. Nutr.* **2010**, *29*, 625–629. [CrossRef] [PubMed]
31. Fleming-Milici, F.; Phaneuf, L.; Harris, J.L. Marketing of Sugar-Sweetened Children's Drinks and Parents' Misperceptions about Benefits for Young Children. *Matern. Child. Nutr.* **2022**, *18*, e13338. [CrossRef] [PubMed]
32. Marshall, T.A.; Levy, S.M.; Broffitt, B.; Warren, J.J.; Eichenberger-Gilmore, J.M.; Burns, T.L.; Stumbo, P.J. Dental Caries and Beverage Consumption in Young Children. *Pediatrics* **2003**, *112*, e184–e191. [CrossRef] [PubMed]
33. Miller, P.E.; Perez, V. Low-Calorie Sweeteners and Body Weight and Composition: A Meta-Analysis of Randomized Controlled Trials and Prospective Cohort Studies. *Am. J. Clin. Nutr.* **2014**, *100*, 765–777. [CrossRef]
34. Nettleton, J.A.; Lutsey, P.L.; Wang, Y.; Lima, J.A.; Michos, E.D.; Jacobs, D.R. Diet Soda Intake and Risk of Incident Metabolic Syndrome and Type 2 Diabetes in the Multi-Ethnic Study of Atherosclerosis (MESA). *Diabetes Care* **2009**, *32*, 688–694. [CrossRef]
35. Zhang, G.-H.; Chen, M.-L.; Liu, S.-S.; Zhan, Y.-H.; Quan, Y.; Qin, Y.-M.; Deng, S.-P. Effects of Mother's Dietary Exposure to Acesulfame-K in Pregnancy or Lactation on the Adult Offspring's Sweet Preference. *Chem. Senses* **2011**, *36*, 763–770. [CrossRef] [PubMed]
36. Fowler, S.P.; Williams, K.; Resendez, R.G.; Hunt, K.J.; Hazuda, H.P.; Stern, M.P. Fueling the Obesity Epidemic? Artificially Sweetened Beverage Use and Long-Term Weight Gain. *Obesity* **2008**, *16*, 1894–1900. [CrossRef] [PubMed]
37. Pepino, M.Y.; Tiemann, C.D.; Patterson, B.W.; Wice, B.M.; Klein, S. Sucralose Affects Glycemic and Hormonal Responses to an Oral Glucose Load. *Diabetes Care* **2013**, *36*, 2530–2535. [CrossRef]

38. Suez, J.; Korem, T.; Zeevi, D.; Zilberman-Schapira, G.; Thaiss, C.A.; Maza, O.; Israeli, D.; Zmora, N.; Gilad, S.; Weinberger, A.; et al. Artificial Sweeteners Induce Glucose Intolerance by Altering the Gut Microbiota. *Nature* **2014**, *514*, 181–186. [CrossRef]
39. Brown, R.J.; De Banate, M.A.; Rother, K.I. Artificial Sweeteners: A Systematic Review of Metabolic Effects in Youth. *Int. J. Pediatr. Obes.* **2010**, *5*, 305–312. [CrossRef]
40. Zheng, M.; Rangan, A.; Olsen, N.J.; Andersen, L.B.; Wedderkopp, N.; Kristensen, P.; Grøntved, A.; Ried-Larsen, M.; Lempert, S.M.; Allman-Farinelli, M.; et al. Substituting Sugar-Sweetened Beverages with Water or Milk Is Inversely Associated with Body Fatness Development from Childhood to Adolescence. *Nutrition* **2015**, *31*, 38–44. [CrossRef]
41. Schneider, S.; Mata, J.; Kadel, P. Relations between Sweetened Beverage Consumption and Individual, Interpersonal, and Environmental Factors: A 6-Year Longitudinal Study in German Children and Adolescents. *Int. J. Public Health* **2020**, *65*, 559–570. [CrossRef] [PubMed]
42. Mullee, A.; Romaguera, D.; Pearson-Stuttard, J.; Viallon, V.; Stepien, M.; Freisling, H.; Fagherazzi, G.; Mancini, F.R.; Boutron-Ruault, M.-C.; Kühn, T.; et al. Association Between Soft Drink Consumption and Mortality in 10 European Countries. *JAMA Intern. Med.* **2019**, *179*, 1479. [CrossRef]
43. Rathaus, M.; Azem, L.; Livne, R.; Ron, S.; Ron, I.; Hadar, R.; Efroni, G.; Amir, A.; Braun, T.; Haberman, Y.; et al. Long-Term Metabolic Effects of Non-Nutritive Sweeteners. *Mol. Metab.* **2024**, *88*, 101985. [CrossRef]
44. Hunter, S.R.; Reister, E.J.; Cheon, E.; Mattes, R.D. Low Calorie Sweeteners Differ in Their Physiological Effects in Humans. *Nutrients* **2019**, *11*, 2717. [CrossRef] [PubMed]
45. Raynor, H.A.; Deierlein, A.L.; Gardner, C.D.; Giovannucci, E.; Taylor, C.A.; Hoelscher, D.M.; Anderson, C.A.M.; Booth, S.L.; Fung, T.T.; Stanford, F.C.; et al. *Low- and No-Calorie Sweetened Beverages and Growth, Body Composition, and Risk of Obesity: A Systematic Review*; USDA Nutrition Evidence Systematic Review (NESR) Systematic Reviews, Rapid Reviews, and Evidence Scans; USDA Nutrition Evidence Systematic Review: Alexandria, VA, USA, 2024.
46. Murphy, M.M.; Barrett, E.C.; Bresnahan, K.A.; Barraj, L.M. 100% Fruit Juice and Measures of Glucose Control and Insulin Sensitivity: A Systematic Review and Meta-Analysis of Randomised Controlled Trials. *J. Nutr. Sci.* **2017**, *6*, e59. [CrossRef]
47. Chakravartti, S.P.; Jann, K.; Veit, R.; Liu, H.; Yunker, A.G.; Angelo, B.; Monterosso, J.R.; Xiang, A.H.; Kullmann, S.; Page, K.A. Non-Caloric Sweetener Effects on Brain Appetite Regulation in Individuals across Varying Body Weights. *Nat. Metab.* **2025**, *7*, 574–585. [CrossRef]
48. Reference Amounts Customarily Consumed per Eating Occasion (21 CFR §101.12). US Food and Drug Administration. 2020. Available online: https://www.law.cornell.edu/cfr/text/21/101.12 (accessed on 14 July 2025).
49. U.S. Department of Agriculture, Agricultural Research Service. Beverages, Carbonated, Cola, Fast-Food Cola. FoodData Cent. 2019. Available online: https://fdc.nal.usda.gov/food-search?type=SR%20Legacy&query=cola (accessed on 14 July 2025).
50. Coca-Cola®Coke—Nutrition Facts—SmartLabelSmartLabel™. Coca-Cola®Coke. 2024. Available online: https://smartlabel.coca-colaproductfacts.com/nutrition/index.html?CocaCola-6760fluidounce&upc=049000050103 (accessed on 10 July 2025).
51. Espinosa, A.; Mendoza, K.; Laviada-Molina, H.; Rangel-Méndez, J.A.; Molina-Segui, F.; Sun, Q.; Tobias, D.K.; Willett, W.C.; Mattei, J. Effects of Nonnutritive Sweeteners on the BMI of Children and Adolescents: A Systematic Review and Meta-Analysis of Randomized Controlled Trials and Prospective Cohort Studies. *Adv. Nutr.* **2024**, *15*, 100292. [CrossRef] [PubMed]
52. Skinner, J.D.; Carruth, B.R. A Longitudinal Study of Children's Juice Intake and Growth. *J. Am. Diet. Assoc.* **2001**, *101*, 432–437. [CrossRef] [PubMed]
53. Shefferly, A.; Scharf, R.J.; DeBoer, M.D. Longitudinal Evaluation of 100% Fruit Juice Consumption on BMI Status in 2-5-Year-Old Children. *Pediatr. Obes.* **2016**, *11*, 221–227. [CrossRef]
54. Nguyen, M.; Jarvis, S.E.; Chiavaroli, L.; Mejia, S.B.; Zurbau, A.; Khan, T.A.; Tobias, D.K.; Willett, W.C.; Hu, F.B.; Hanley, A.J.; et al. Consumption of 100% Fruit Juice and Body Weight in Children and Adults: A Systematic Review and Meta-Analysis. *JAMA Pediatr.* **2024**, *178*, 237–246. [CrossRef]
55. Beauchamp, G.K.; Mennella, J.A. Early Flavor Learning and Its Impact on Later Feeding Behavior. *J. Pediatr. Gastroenterol. Nutr.* **2009**, *48* (Suppl. 1), S25–S30. [CrossRef]
56. Mueller, C.; Zeinstra, G.G.; Forde, C.G.; Jager, G. Sweet and Sour Sips: No Effect of Repeated Exposure to Sweet or Sour-Tasting Sugary Drinks on Children's Sweetness Preference and Liking. *Appetite* **2024**, *196*, 107277. [CrossRef]
57. Mennella, J.A.; Bobowski, N.K. The Sweetness and Bitterness of Childhood: Insights from Basic Research on Taste Preferences. *Physiol. Behav.* **2015**, *152*, 502–507. [CrossRef]
58. Stribiţcaia, E.; Evans, C.E.L.; Gibbons, C.; Blundell, J.; Sarkar, A. Food Texture Influences on Satiety: Systematic Review and Meta-Analysis. *Sci. Rep.* **2020**, *10*, 12929. [CrossRef] [PubMed]
59. Søberg, S.; Sandholt, C.H.; Jespersen, N.Z.; Toft, U.; Madsen, A.L.; Von Holstein-Rathlou, S.; Grevengoed, T.J.; Christensen, K.B.; Bredie, W.L.P.; Potthoff, M.J.; et al. FGF21 Is a Sugar-Induced Hormone Associated with Sweet Intake and Preference in Humans. *Cell Metab.* **2017**, *25*, 1045–1053.e6. [CrossRef] [PubMed]

60. Giannini, C.; Feldstein, A.E.; Santoro, N.; Kim, G.; Kursawe, R.; Pierpont, B.; Caprio, S. Circulating Levels of FGF-21 in Obese Youth: Associations with Liver Fat Content and Markers of Liver Damage. *J. Clin. Endocrinol. Metab.* **2013**, *98*, 2993–3000. [CrossRef]
61. Feldstein Ewing, S.W.; Claus, E.D.; Hudson, K.A.; Filbey, F.M.; Yakes Jimenez, E.; Lisdahl, K.M.; Kong, A.S. Overweight Adolescents' Brain Response to Sweetened Beverages Mirrors Addiction Pathways. *Brain Imaging Behav.* **2017**, *11*, 925–935. [CrossRef]
62. Bobbert, T.; Schwarz, F.; Fischer-Rosinsky, A.; Pfeiffer, A.F.H.; Möhlig, M.; Mai, K.; Spranger, J. Fibroblast Growth Factor 21 Predicts the Metabolic Syndrome and Type 2 Diabetes in Caucasians. *Diabetes Care* **2013**, *36*, 145–149. [CrossRef]
63. Jang, H.-J.; Kokrashvili, Z.; Theodorakis, M.J.; Carlson, O.D.; Kim, B.-J.; Zhou, J.; Kim, H.H.; Xu, X.; Chan, S.L.; Juhaszova, M.; et al. Gut-Expressed Gustducin and Taste Receptors Regulate Secretion of Glucagon-like Peptide-1. *Proc. Natl. Acad. Sci. USA* **2007**, *104*, 15069–15074. [CrossRef]
64. Margolskee, R.F.; Dyer, J.; Kokrashvili, Z.; Salmon, K.S.H.; Ilegems, E.; Daly, K.; Maillet, E.L.; Ninomiya, Y.; Mosinger, B.; Shirazi-Beechey, S.P. T1R3 and Gustducin in Gut Sense Sugars to Regulate Expression of Na^+-Glucose Cotransporter 1. *Proc. Natl. Acad. Sci. USA* **2007**, *104*, 15075–15080. [CrossRef]
65. Ma, J.; Bellon, M.; Wishart, J.M.; Young, R.; Blackshaw, L.A.; Jones, K.L.; Horowitz, M.; Rayner, C.K. Effect of the Artificial Sweetener, Sucralose, on Gastric Emptying and Incretin Hormone Release in Healthy Subjects. *Am. J. Physiol.-Gastrointest. Liver Physiol.* **2009**, *296*, G735–G739. [CrossRef] [PubMed]
66. Wallace, D.L.; Vialou, V.; Rios, L.; Carle-Florence, T.L.; Chakravarty, S.; Kumar, A.; Graham, D.L.; Green, T.A.; Kirk, A.; Iñiguez, S.D.; et al. The Influence of ΔFosB in the Nucleus Accumbens on Natural Reward-Related Behavior. *J. Neurosci.* **2008**, *28*, 10272–10277. [CrossRef]
67. Salinas-Velarde, I.D.; Bernal-Morales, B.; Pacheco-Cabrera, P.; Sánchez-Aparicio, P.; Pascual-Mathey, L.I.; Venebra-Muñoz, A. Lower ΔFosB Expression in the Dopaminergic System after Stevia Consumption in Rats Housed under Environmental Enrichment Conditions. *Brain Res. Bull.* **2021**, *177*, 172–180. [CrossRef]
68. Lanaspa, M.A.; Sanchez-Lozada, L.G.; Cicerchi, C.; Li, N.; Roncal-Jimenez, C.A.; Ishimoto, T.; Le, M.; Garcia, G.E.; Thomas, J.B.; Rivard, C.J.; et al. Uric Acid Stimulates Fructokinase and Accelerates Fructose Metabolism in the Development of Fatty Liver. *PLoS ONE* **2012**, *7*, e47948. [CrossRef]
69. Cohen, C.C.; Li, K.W.; Alazraki, A.L.; Beysen, C.; Carrier, C.A.; Cleeton, R.L.; Dandan, M.; Figueroa, J.; Knight-Scott, J.; Knott, C.J.; et al. Dietary Sugar Restriction Reduces Hepatic de Novo Lipogenesis in Adolescent Boys with Fatty Liver Disease. *J. Clin. Investig.* **2021**, *131*, e150996. [CrossRef]
70. Douard, V.; Ferraris, R.P. Regulation of the Fructose Transporter GLUT5 in Health and Disease. *Am. J. Physiol.-Endocrinol. Metab.* **2008**, *295*, E227–E237. [CrossRef]
71. Socha-Banasiak, A.; Sakowicz, A.; Gaj, Z.; Kolejwa, M.; Gach, A.; Czkwianianc, E. Intestinal Fructose Transporters GLUT5 and GLUT2 in Children and Adolescents with Obesity and Metabolic Disorders. *Adv. Med. Sci.* **2024**, *69*, 349–355. [CrossRef]
72. Azad, M.B.; Sharma, A.K.; De Souza, R.J.; Dolinsky, V.W.; Becker, A.B.; Mandhane, P.J.; Turvey, S.E.; Subbarao, P.; Lefebvre, D.L.; Sears, M.R.; et al. Association Between Artificially Sweetened Beverage Consumption During Pregnancy and Infant Body Mass Index. *JAMA Pediatr.* **2016**, *170*, 662. [CrossRef]
73. Sewaybricker, L.E.; Schur, E.A.; Melhorn, S.J.; Campos, B.M.; Askren, M.K.; Nogueira, G.A.S.; Zambon, M.P.; Antonio, M.A.R.G.M.; Cendes, F.; Velloso, L.A.; et al. Initial Evidence for Hypothalamic Gliosis in Children with Obesity by Quantitative T2 MRI and Implications for Blood Oxygen-level Dependent Response to Glucose Ingestion. *Pediatr. Obes.* **2019**, *14*, e12486. [CrossRef] [PubMed]
74. Valdearcos, M.; McGrath, E.; Brown Mayfield, S.; Folick, A.; Cheang, R.; Li, L.; Bachor, T.; Lippert, R.; Xu, A.; Koliwad, S. Microglia Mediate the Early-Life Programming of Adult Glucose Control. *Cell Rep.* **2024**, *44*, 115409. [CrossRef] [PubMed]
75. Mendoza-Romero, H.N.; Biddinger, J.E.; Bedenbaugh, M.N.; Simerly, R. Microglia Are Required for Developmental Specification of AgRP Innervation in the Hypothalamus of Offspring Exposed to Maternal High-Fat Diet during Lactation. *eLife* **2025**, *13*, RP101391. [CrossRef] [PubMed]
76. Shearrer, G.E.; Daniels, M.J.; Toledo-Corral, C.M.; Weigensberg, M.J.; Spruijt-Metz, D.; Davis, J.N. Associations among Sugar Sweetened Beverage Intake, Visceral Fat, and Cortisol Awakening Response in Minority Youth. *Physiol. Behav.* **2016**, *167*, 188–193. [CrossRef]
77. Martin, A.A.; Hamill, L.R.; Davies, S.; Rogers, P.J.; Brunstrom, J.M. Energy-Dense Snacks Can Have the Same Expected Satiation as Sugar-Containing Beverages. *Appetite* **2015**, *95*, 81–88. [CrossRef]
78. Serrano, J.; Smith, K.R.; Crouch, A.L.; Sharma, V.; Yi, F.; Vargova, V.; LaMoia, T.E.; Dupont, L.M.; Serna, V.; Tang, F.; et al. High-Dose Saccharin Supplementation Does Not Induce Gut Microbiota Changes or Glucose Intolerance in Healthy Humans and Mice. *Microbiome* **2021**, *9*, 11. [CrossRef]
79. Sylvetsky, A.C.; Rother, K.I. Trends in the Consumption of Low-Calorie Sweeteners. *Physiol. Behav.* **2016**, *164*, 446–450. [CrossRef]

80. Lustig, R.H.; Schmidt, L.A.; Brindis, C.D. The Toxic Truth about Sugar. *Nature* **2012**, *482*, 27–29. [CrossRef] [PubMed]
81. Stanhope, K.L. Sugar Consumption, Metabolic Disease and Obesity: The State of the Controversy. *Crit. Rev. Clin. Lab. Sci.* **2016**, *53*, 52–67. [CrossRef]
82. Colley, D.L.; Castonguay, T.W. Effects of Sugar Solutions on Hypothalamic Appetite Regulation. *Physiol. Behav.* **2015**, *139*, 202–209. [CrossRef] [PubMed]
83. Petersen, K.F.; Dufour, S.; Mehal, W.Z.; Shulman, G.I. Glucagon Promotes Increased Hepatic Mitochondrial Oxidation and Pyruvate Carboxylase Flux in Humans with Fatty Liver Disease. *Cell Metab.* **2024**, *36*, 2359–2366.e3. [CrossRef]
84. Greenhill, C. Not so Sweet—Artificial Sweeteners Can Cause Glucose Intolerance by Affecting the Gut Microbiota. *Nat. Rev. Endocrinol.* **2014**, *10*, 637. [CrossRef]
85. Mandrioli, D.; Kearns, C.E.; Bero, L.A. Relationship between Research Outcomes and Risk of Bias, Study Sponsorship, and Author Financial Conflicts of Interest in Reviews of the Effects of Artificially Sweetened Beverages on Weight Outcomes: A Systematic Review of Reviews. *PLoS ONE* **2016**, *11*, e0162198. [CrossRef] [PubMed]
86. InterAct Consortium; Romaguera, D.; Norat, T.; Wark, P.A.; Vergnaud, A.C.; Schulze, M.B.; van Woudenbergh, G.J.; Drogan, D.; Amiano, P.; Molina-Montes, E.; et al. Consumption of Sweet Beverages and Type 2 Diabetes Incidence in European Adults: Results from EPIC-InterAct. *Diabetologia* **2013**, *56*, 1520–1530. [CrossRef]
87. Pacheco, L.S.; Tobias, D.K.; Haslam, D.E.; Drouin-Chartier, J.-P.; Li, Y.; Bhupathiraju, S.N.; Willett, W.C.; Ludwig, D.S.; Ebbeling, C.B.; Hu, F.B.; et al. Sugar-Sweetened or Artificially Sweetened Beverage Consumption, Physical Activity and Risk of Type 2 Diabetes in US Adults. *Diabetologia* **2025**, *68*, 792–800. [CrossRef]
88. Malik, V.S.; Li, Y.; Pan, A.; De Koning, L.; Schernhammer, E.; Willett, W.C.; Hu, F.B. Long-Term Consumption of Sugar-Sweetened and Artificially Sweetened Beverages and Risk of Mortality in US Adults. *Circulation* **2019**, *139*, 2113–2125. [CrossRef] [PubMed]
89. López-Pascual, E.; Rienda, I.; Perez-Rojas, J.; Rapisarda, A.; Garcia-Llorens, G.; Jover, R.; Castell, J.V. Drug-Induced Fatty Liver Disease (DIFLD): A Comprehensive Analysis of Clinical, Biochemical, and Histopathological Data for Mechanisms Identification and Consistency with Current Adverse Outcome Pathways. *Int. J. Mol. Sci.* **2024**, *25*, 5203. [CrossRef]
90. Emamat, H.; Ghalandari, H.; Tangestani, H.; Abdollahi, A.; Hekmatdoost, A. Artificial Sweeteners Are Related to Non-Alcoholic Fatty Liver Disease: Microbiota Dysbiosis as a Novel Potential Mechanism. *EXCLI J.* **2020**, *19*, 620–626. [CrossRef]
91. Vos, M.B.; Abrams, S.H.; Barlow, S.E.; Caprio, S.; Daniels, S.R.; Kohli, R.; Mouzaki, M.; Sathya, P.; Schwimmer, J.B.; Sundaram, S.S.; et al. NASPGHAN Clinical Practice Guideline for the Diagnosis and Treatment of Nonalcoholic Fatty Liver Disease in Children: Recommendations from the Expert Committee on NAFLD (ECON) and the North American Society of Pediatric Gastroenterology, Hepatology and Nutrition (NASPGHAN). *J. Pediatr. Gastroenterol. Nutr.* **2017**, *64*, 319–334. [CrossRef] [PubMed]
92. Tseng, T.-S.; Lin, W.-T.; Ting, P.-S.; Huang, C.-K.; Chen, P.-H.; Gonzalez, G.V.; Lin, H.-Y. Sugar-Sweetened Beverages and Artificially Sweetened Beverages Consumption and the Risk of Nonalcoholic Fatty Liver (NAFLD) and Nonalcoholic Steatohepatitis (NASH). *Nutrients* **2023**, *15*, 3997. [CrossRef]
93. World Health Organization. Oral Health Data Portal. *The Global Health Observatory.* 2023. Available online: https://www.who.int/data/gho/data/themes/oral-health-data-portal (accessed on 10 July 2025).
94. American Dental Association. Dental Erosion 2021. Available online: https://www.ada.org/resources/ada-library/oral-health-topics/dental-erosion (accessed on 9 September 2025).
95. Reddy, A.; Norris, D.F.; Momeni, S.S.; Waldo, B.; Ruby, J.D. The pH of Beverages in the United States. *J. Am. Dent. Assoc.* **2016**, *147*, 255–263. [CrossRef] [PubMed]
96. Teng, A.M.; Jones, A.C.; Mizdrak, A.; Signal, L.; Genç, M.; Wilson, N. Impact of Sugar-sweetened Beverage Taxes on Purchases and Dietary Intake: Systematic Review and Meta-analysis. *Obes. Rev.* **2019**, *20*, 1187–1204. [CrossRef] [PubMed]
97. Roberto, C.A.; Lawman, H.G.; LeVasseur, M.T.; Mitra, N.; Peterhans, A.; Herring, B.; Bleich, S.N. Association of a Beverage Tax on Sugar-Sweetened and Artificially Sweetened Beverages with Changes in Beverage Prices and Sales at Chain Retailers in a Large Urban Setting. *JAMA* **2019**, *321*, 1799. [CrossRef]
98. Latner, J.D.; Stunkard, A.J. Getting Worse: The Stigmatization of Obese Children. *Obes. Res.* **2003**, *11*, 452–456. [CrossRef]
99. Puhl, R.M.; Heuer, C.A. The Stigma of Obesity: A Review and Update. *Obesity* **2009**, *17*, 941–964. [CrossRef] [PubMed]
100. Gortmaker, S.L.; Long, M.W.; Resch, S.C.; Ward, Z.J.; Cradock, A.L.; Barrett, J.L.; Wright, D.R.; Sonneville, K.R.; Giles, C.M.; Carter, R.C.; et al. Cost Effectiveness of Childhood Obesity Interventions. *Am. J. Prev. Med.* **2015**, *49*, 102–111. [CrossRef]
101. Powell, L.M.; Wada, R.; Persky, J.J.; Chaloupka, F.J. Employment Impact of Sugar-Sweetened Beverage Taxes. *Am. J. Public Health* **2014**, *104*, 672–677. [CrossRef]
102. Micha, R.; Karageorgou, D.; Bakogianni, I.; Trichia, E.; Whitsel, L.P.; Story, M.; Peñalvo, J.L.; Mozaffarian, D. Effectiveness of School Food Environment Policies on Children's Dietary Behaviors: A Systematic Review and Meta-Analysis. *PLoS ONE* **2018**, *13*, e0194555. [CrossRef]

103. Pallan, M.; Murphy, M.; Morrison, B.; Sitch, A.; Adamson, A.; Bartington, S.; Dobell, A.; Duff, R.; Frew, E.; Griffin, T.; et al. National School Food Standards in England: A Cross-Sectional Study to Explore Compliance in Secondary Schools and Impact on Pupil Nutritional Intake. *Int. J. Behav. Nutr. Phys. Act.* **2024**, *21*, 123. [CrossRef] [PubMed]
104. Schwartz, A.E.; Leardo, M.; Aneja, S.; Elbel, B. Effect of a School-Based Water Intervention on Child Body Mass Index and Obesity. *JAMA Pediatr.* **2016**, *170*, 220. [CrossRef] [PubMed]