The Development of Sweet Taste: From Biology to Hedonics

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Abstract

From the age of two years, an American child is more likely to consume a sugar-sweetened product than a fruit or vegetable on any given day—a troubling statistic, given that food preferences are established early in childhood, as well as the strong association between this dietary pattern and increased risk of developing a number of chronic diseases. Here, we review the ontogeny and biopsychology of sweet taste, highlighting how a biological drive to prefer sweetness at high concentrations during childhood, which would have conferred an advantage in environments of scarcity, now predisposes children to overconsume all that is sweet in a modern food system replete with added sugars. We review the power of sweet taste to blunt expressions of pain and mask bad tastes in foods as well as factors that predispose some to consume high-sugar diets, including experiential learning and taste preferences driven in part by genetics. Understanding children’s unique vulnerability to our current food environment, rich in both nutritive and nonnutritive sweeteners, is highlighted as a priority for future research to develop evidence-based strategies to help establish healthy dietary behaviors early in life.

Keywords
children; sweet taste; biology; psychology; nonnutritive sweeteners; taste genetics

Introduction

From the age of two years, an American is more likely to eat a manufactured sweet than a fruit or vegetable on a given day [1, 2]. By age three, most children consume at least one sugar-sweetened beverage daily [3]. More than 15% of children’s total caloric intake comes from added sugars, levels well above recommendations of 5–10% [4, 5]. In contrast, sweet-tasting fruits make up approximately 5% of calories [6–8]. Ultra-processed foods such as sugar-sweetened beverages, are major sources of added sugars in the diet [9, 10], and the higher the intake, the poorer the nutrient quality of the diet [11]. These dietary patterns are troubling since a diet established early in life that is low in whole fruits and high in energy-dense processed foods [2, 12] increases the risk of developing obesity, cardiovascular

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disease, hypertension, obesity-related cancers, and the most prevalent chronic disease of childhood, dental caries [13–20].

In this article, we review the ontogeny and biopsychology of sweet taste, a critical first step to address the modern patterns of food choices that are antithetical to health, and why it is so difficult to change bad habits and develop good ones. Two factors predispose humans toward obesogenic, high-sugar diets: (a) inborn, evolutionarily driven taste preferences that make individuals vulnerable to the modern food environment rich in sugar, and (b) lack of exposure to a variety of healthy foods during early childhood. Evolution has shaped the taste of foods children initially prefer or reject. In an environment with limited nutrients and abundant poisonous plants, sensory systems evolved to detect and prefer perceptions that specify crucial nutrients such as the once rare energy (carbohydrate)-rich plants that taste sweet [21, 22], while rejecting those that taste bitter [23]. By focusing on the biology and psychology of sweet taste, as well as the contextual learning about the sweetness of foods, we gain insights into why children as a group are more vulnerable to a food environment rich in sweetened processed foods and why some children are more vulnerable to this environment.

1. The Biology of Sweet Taste

In nature, carbohydrates are a source of energy often equated with sweetness, which is associated with powerful hedonic appeal. The emotions experienced upon tasting something sweet are complex processes mediated by sweet taste receptors in the periphery and by multiple brain substrates, which phylogenetically are remarkably well conserved [24]. Progress has been made in identifying the initial events underlying sweet taste recognition [25, 26] and the brain mechanisms underlying the strong hedonic responses experienced upon tasting something sweet [27, 28].

Sweetness as a sensation starts on the tongue. When sweet-tasting (nutritive and nonnutritive) ligands stimulate a receptor on taste cells, the resulting signal is conducted via G proteins [29], which activate pleasure-generating brain circuitry, where sweet taste perception and hedonics arise (reviewed in [30]). The hedonic “liking” and motivation “wanting” signals for a sweet taste are distinctly modulated through brain mesocorticolimbic circuitry involving the nucleus accumbens and ventral pallidum [28].

At the receptor level, two proteins, T1R2 and T1R3 (taste receptor family 1, proteins 2 and 3), combine to create a sweet taste receptor; their associated genes are TAS1R2 and TAS1R3. Among the G proteins, the one associated with intracellular sweet signaling is gustducin (Gα protein subunit), encoded by the GNAT3 gene [29, 31]. These receptor and transduction proteins are underexpressed in some people due to inborn genetic variation, and genotype explains, in part, differences in sweet taste sensitivity and preferences [32–38], at least among adults.

A recent study of 1,901 adolescent and young adult twins found that a common genetic factor accounted for 30% of the overall variance in their sensitivity to the sweet taste of two monosaccharides (glucose, fructose) and two nonnutritive sweeteners (neohesperidin...
dihydrochalcone, aspartame) [39]. While this twin study did not determine whether the
common factor was the sweet taste receptor gene, other data reveal that (a) variation in the
TAS1R3 subunit of the sweet taste receptor gene and (b) variation in the GNAT3 gene that
codes for the intracellular sweet transduction protein are related to sucrose detection
thresholds (defined as the lowest concentration of a stimulus needed by a subject to detect its
presence relative to water). Adults with the TAS1R3 CC genotype (rs1015443) or GNAT3
TT genotype (rs7792845) detected sugars at lower concentrations (were more sensitive) than
those with the T or C allele, respectively [34–38]. Adults with the TAS1R3 CC genotype
were more sweet sensitive and preferred lower levels of sweet than others [34]. Thus, at least
for adults and older children, genotype explains why not all people perceive sweetness
equally strongly [30].

Taste receptor proteins appear in many cell types including those with a secretory or other
endocrine function like ductal and beta cells of the pancreas [40, 41] and enteroendocrine
cells of the gut [42]. Likewise, some taste receptor cells in the oral cavity have an endocrine
role in the body. These cells produce and secrete hormones such as serotonin,
cholecystokinin, vasoactive intestinal peptide, glucagon-like peptide 1, ghrelin, and
neuropeptide Y [43–46]. The effect of these molecules may be to modulate the function of
their own or adjacent cells. Beyond these autocrine and paracrine effects, they may also
enter the bloodstream, where they have hormonal effects [47]. The biological utility of these
hormonal responses may be part of the cephalic-phase response, whereby the body prepares
for incoming food, which is logical because the first contact of food with cells of the body
occurs in the taste cells [47]. While most studies have made these endocrine observations in
model organisms, mostly mice and rats, the close ties between the rodent and primate taste
systems suggest these endocrine effects would be similar in humans. From a developmental
perspective, early experience with excess sweet taste stimulation may prime the cephalic-
phase effects and affect nutrient absorption from the gut in ways that could persist into
adulthood.

2. The Biopsychology of Sweet Taste in Children

The study of the ontogeny of human sweet taste perception has been the focus of scientific
investigations for many decades. Table 1 highlights the variety of scientific approaches,
methodologies, and outcome measures that have been used to assess (a) sweet taste
perception and preferences and (b) the consequence of tasting something sweet on
behavioral responses in infants and children.

The sensory pleasantness derived from tasting something sweet is inborn. Before birth, the
ability to detect sweet tastes is functioning and interacting with systems controlling affect.
Upon tasting something sweet, infants born preterm produced stronger and more frequent
sucking responses [48, 49] and were less likely to exhibit spontaneous crying when
compared to tasting water [50]. Within hours of birth, newborns can differentiate varying
degrees of sweetness [51] and will ingest a greater volume of a solution that tastes sweeter
[52]. When something sweet tasting is in the oral cavity, their faces relax and this relaxation
is often accompanied by a smile [53–55] and they begin sucking more voraciously (i.e.,
longer sucking burst, decreased latency to suck) [56, 57]. That sweet taste per se promotes
feeding is suggested by the finding that tasting either nutritive sweeteners (e.g., sucrose) or nonnutritive sweeteners (NNS; e.g., aspartame) elicits mouthing and sucking movements and hand-to-mouth contact [58–60], all of which are feeding-related behaviors.

Perhaps related to its effects on promoting ingestion, tasting something sweet blunts expressions of pain [61–64]. A small amount of a sweet solution placed on the tongue of a crying newborn exerts a rapid calming effect that persists for several minutes [58, 60] (see Stevens [61] for review). Because the NNS aspartame mimics the effects of sucrose [59, 65], and because the administration of sucrose by direct stomach loading is not effective [66], afferent signals from the mouth, rather than gastric or metabolic changes, appear to be responsible. Tasting sucrose, but not water, delayed 8- to 11-year-olds’ reporting of pain onset when undergoing the cold pressor test, a cold-induced pain stimulus test [67–69], but was not evident in adults [69]. However, the pain-reducing effect of sucrose in children is context dependent, since tasting something sweet was ineffective during needle-related procedural pain [70].

During the cold pressor test, the more a child liked the taste of sucrose, the better it increased pain tolerance. Of interest, the pain-reducing properties of sugars were attenuated among children who were overweight or at risk for overweight [69]. Whether this attenuation is due to impairment of hypothalmo-pituitary control and higher than normal beta-endorphin plasma levels, which have been observed in obese children [71], is not known. Nor do we know whether these children consumed carbohydrates and sweet-tasting foods chronically or in higher amounts, which could in turn modify opioid and dopamine receptor binding and thus the efficacy of its analgesic properties [72–77].

When it comes to sweet tastes, children live in different sensory worlds than adults. Both cross-sectional and longitudinal studies have revealed that the preference for sweet taste remains heightened throughout childhood, declining to adult levels during mid-adolescence [33, 35, 68, 78–83]. Like adults [84, 85], children exhibit a wide range in their taste sensitivity to sweet (i.e., lowest concentration of sweetness that can be detected), and such variation is due, in part, to variation in taste receptor genes and measures of obesity [86, 87]. But it does not appear that children prefer higher levels of sweet taste because they are insensitive to its taste. What causes the age-related decline in sweet preference and consumption between adolescence and adulthood remains a mystery, but it has been observed in other mammals [88]. There is evidence that the heightened sweet preference may be linked to the growing child’s need for calories that begins during infancy and continues until the cessation of puberty [89]. Two studies found that levels of sweetness most preferred by children positively correlated with urinary concentrations of N-telopeptides of type I collagen [35, 90], a biomarker for bone resorption and growth [91], thus suggesting that the age-related decline in sucrose preferences may be related to the cessation of physical growth.

Children’s preference for sweet taste is evident not only in the level of sweetness they prefer in a liquid as measured in the laboratory but also in the foods they consume. The higher the level of sweetness in solution most preferred, the higher the sugar content of their favorite foods, including breakfast cereals [80, 92] and beverages [80, 93]. Children will drink more
of a beverage (e.g., Kool-Aid [94]) and eat more of a food (e.g., spaghetti [95]) when it contains a higher amount of added sugar and thus tastes sweeter. Sweet taste drives consumption not just because children like its taste but because adding sugar masks the unpleasant tastes (e.g., bitter [96, 97]) inherent to a food or beverage or which develop during manufacturing and processing [98].

During childhood, children learn the rules of cuisine: what to eat, how to eat, when to eat, and how sweet a food is supposed to taste [99–104]. The sensation of sweetness is context dependent and can acquire meaning through associative learning [105, 106]. Through familiarization and repeated exposure, children develop a sense of what should, or should not, taste sweet [94, 102, 107]. Longitudinal studies revealed that babies who were routinely fed sweetened water (e.g., water sweetened with table sugar or honey) during the first months of life exhibited a greater preference for sweetened water when tested at 6 months and then again at 2–10 years of age, compared with children who had little or no experience with sweetened water as infants [81, 94, 108]. Perhaps feeding sugar water during infancy is an indication of the likelihood that sugar-sweetened beverages and other foods will be part of their family diet, or perhaps the children have been programmed to have heightened preferences for sweet tastes (see [109–111]).

In recent years, the food environment in which children live has changed, with increasing numbers of foods, beverages, and liquid medications geared for pediatric populations sweetened with NNS or blends of nutritive sweeteners and NNS [112], which contribute sweetness with few to no calories. Because of the paucity of research on how children respond to the taste of NNS, we expanded the study of sweet preferences in children to include the NNS sucralose and compared preference ratings between the two stimuli. The most preferred concentrations of sucrose and sucralose were determined separately among 6- to 14-year-olds via the Monell forced-choice, paired-comparison tracking procedure [79]. Sucrose solutions ranging in concentration from 90 to 1050 mM, and sucralose solutions ranging from 0.1 to 1.5 mM, were presented during two separate test sessions; concentration ranges were based on equated sweetness intensity of the two stimuli; psychophysical testing revealed sucralose is approximately 600 times sweeter than sucrose on a weight-by-weight basis [113]). All procedures were approved by the Office of Regulatory Affairs at the University of Pennsylvania, and informed consent was obtained from their mothers and informed assent from the children.

Children most preferred a 700 mM sucrose solution and a 1.03 mM sucralose solution. While sucralose is sweeter than sucrose on a weight-by-weight basis, from these data one cannot conclude that children like sucralose more than sucrose. To put this in perspective, this concentration of sucrose most preferred by these children is equivalent to approximately 14 teaspoons of sugar in 237 ml of water (i.e., an eight-ounce glass), nearly twice the sugar concentration of a typical cola. When the sweetness of sucralose is equated for the sweetness of sucrose [113], the level of sucralose most preferred is nearly identical (720 mM sucrose). That the level most preferred for these two taste stimuli are highly correlated [r(36)=0.70, p<0.001], suggests that, at least for sucrose and sucralose, children have a particular bliss point range for sweetness.
3. Concluding Remarks

The convergence of scientific evidence suggests that the ability to detect and prefer sweet taste is evident early in life and reflects both biology and experiential learning. Evolution has shaped the child’s response to sweetened foods and beverages, and our sensory systems evolved to detect and prefer the once rare calorie-rich foods that taste sweet [114]. While this “sweet” attraction may have served children well in a feast-or-famine setting, attracting them to mothers’ milk and then to energy-rich foods during periods of growth, today it makes them vulnerable to food environments abundant in processed foods rich in added sugars and lacking healthy sweet foods, such as fruits [33, 114, 115]. Indeed, the more fruits in a child’s diet, the less added sugar the child consumed [9, 116].

We hypothesize that in the current food environment, fruits are competing with “supernormal foods” [117]—that is, foods and beverages rich in added sugars [118] and/or NNS—which children may be especially vulnerable to overconsuming, given their proclivity for sweet taste. Once children become accustomed to a highly sweetened diet, they may find that the very tastes that their sensory systems evolved to be attracted to (e.g., fruits) don’t taste “sweet enough” [22]. This is supported by experimental studies in animal models showing that initially preferred sweet solutions became less preferred after rodents experienced more intense sweetness [88].

Given the increased prevalence of NNS in the food supply, including products intended for children (of which parents are often unaware [119]), how much added sugar is in the child’s diet may no longer be a good proxy for the diet’s overall level of “sweetness.” This changing food supply brings to light a number of questions regarding the impact of sweetness on the growing and developing child that are priorities for future research [120–122]. Because some taste receptors are located in the endocrine system of the gut [42], what are the consequences of exposure to NNS (sweet but few to no calories) in growing children [17, 123, 124]? What are the consequences of children learning to associate sweet taste with foods that typically are not sweet but, because of added sugars and/or NNS, have been processed to taste sweet? Since children prefer a more intense sweet sensation than do adults, are they more likely to overconsume NNS? Given the current evidence suggesting that reducing overall intake of sweetness (both caloric sweeteners and NNS) may be a better strategy to lower rates of obesity than simply replacing the former with the latter [17], can children who have learned that foods should taste sweet relearn to like a particular food in its natural, unsweetened form?

Understanding the unique vulnerability of children to the modern food system is a critical first step for developing informed, evidence-based strategies to address what has become an issue of great public health importance, because many chronic diseases plaguing modern society derive in large part from poor food choices, dictated by our flavor preferences and the types of foods that are available and deemed appropriate for children. Increasing healthy dietary behaviors (and decreasing unhealthy habits), beginning in childhood, is recognized as the most important aspect of reducing an individual’s risk of lifestyle-related diseases [125].
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Table 1

Summary of representative pediatric research on sweet taste, from preterm infants to children, illustrating the diversity of methods and variety of outcomes

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Methodology and Outcomes</th>
<th>References</th>
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<tbody>
<tr>
<td>Preterm Infants</td>
<td>Increased strength and frequency of sucking of sucrose-sweetened nipple compared with unsweetened nipple</td>
<td>Maone et al., 1990 [48]</td>
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<tr>
<td></td>
<td>Increased strength and frequency of sucking when tasting glucose-sweetened drops of solution compared with water</td>
<td>Tatzer et al., 1985 [49]</td>
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<td>Attenuated pain response (as evidenced by decreases in crying duration) during venipuncture procedure when tasting a sweet (sucrose or glucose) solution compared with water</td>
<td>Abad et al., 1996 [62]; Deshmukh and Udani, 2002 [63]</td>
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<td></td>
<td>Decreased spontaneous crying relative to baseline when tasting sweet (sucrose or glucose) solution compared with water</td>
<td>Smith and Blass, 1996 [50]</td>
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<tr>
<td>Infants</td>
<td>Increased strength and frequency of sucking of a sucrose-sweetened compared with unsweetened nipple</td>
<td>Maone et al., 1990 [48]</td>
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<td>Attenuated pain response (as evidenced by decreases in crying incidence and duration) during and after painful procedures (e.g., immunization, heel lance, circumcision) when tasting sweet (sucrose or glucose) solution compared with water</td>
<td>Harrison et al., [64]; Blass and Hoffmeyer, 1991 [60]</td>
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<td></td>
<td>Decreased spontaneous crying relative to baseline when tasting a sweet (sucrose or glucose) solution compared with water</td>
<td>Smith and Blass, 1996 [50]</td>
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<td></td>
<td>Increased sucking burst length and decreased sucking latency with a sucrose-sweetened compared with unsweetened nipple</td>
<td>Crook, 1978 [56]; Crook and Lipsitt, 1976 [57]</td>
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<td>Increased sucking-like and mouthing responses, facial relaxation, smiling, and hand-mouth contact when tasting sweet (sucrose) solution compared with water</td>
<td>Rosenstein and Oster, 1988 [55]; Steiner et al, 2001 [53]</td>
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<td></td>
<td>Decreased crying and increased hand-mouth contact when tasting sweet (sucrose) solution compared with water</td>
<td>Barr et al., 1999 [59]</td>
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<tr>
<td></td>
<td>Decreased crying and increased hand-mouth contact when tasting sweet (aspartame) solution compared with water</td>
<td>Barr et al., 1999 [59]</td>
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<td></td>
<td>Increased intake of sweet solutions (sucrose, glucose, fructose, lactose) compared with water</td>
<td>Miller and Moran, 1982 [108]; Desor et al., 1973 [32]</td>
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<td>Children</td>
<td>Increased pain threshold during cold pressor test when tasting sweet (sucrose) solution compared with water</td>
<td>Miller et al., 1994 [67]; Pepino and Mennella, 2005 [69]; Mennella et al., 2010 [68]</td>
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<td></td>
<td>No effect on pain induced by needle-related procedures when tasting something sweet compared with water</td>
<td>Harrison et al., 2015 [70]</td>
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<td>Concentration of sucrose most preferred higher among children than among adults, with the changeover to adult pattern occurring during mid-late adolescence</td>
<td>de Graaf and Zandstra, 1999 [82]; Mennella et al., 2005 [80]; Mennella et al., 2011 [79]; Mennella et al., 2014 [35]; Desor and Beauchamp, 1987 [78]; Desor et al., 1975 [83]</td>
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<td>Concentration of sucrose most preferred higher during periods of growth</td>
<td>Coldwell et al., 2009 [90]; Mennella et al., 2014 [35]</td>
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<td>Children learn through repeated exposure the most appropriate level of sweetness for a particular food</td>
<td>Beauchamp and Moran, 1984 [94]; Sullivan and Birch, 1990 [102]; Liem and de Graaf, 2004 [107]</td>
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<td>Increased consumption of a sweetened version of a food or beverage (e.g., spaghetti, Kool-Aid, vegetables) compared with unsweetened counterpart</td>
<td>Filer, 1978 [95]; Beauchamp and Moran, 1984 [94]; Sharafi et al., 2013 [97]</td>
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<td>Sucrose masked the bitterness and increased liking of a range of bitter-tasting stimuli (urea, caffeine, denatonium benzoate, propylthiouracil, and quinine)</td>
<td>Mennella et al., 2015 [96]</td>
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<td></td>
<td>Sucrose detection thresholds decreased with age and central obesity (the older the child or the more central obesity, the more sensitive the child was to sweet taste)</td>
<td>Joseph et al., 2016 [87]</td>
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This review is not meant to be representative of all research conducted to date; rather, it highlights the breadth of methodologies employed and the convergence of findings.