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Craving and Control

"Nature's 'Hunger Thermostat'" reads the ad copy in *Life Magazine*:

> When your blood sugar level is low you are "hungry as a bear." When it is high the healthy person finds it easier to turn down the extra helpings of food that mean extra pounds. It turns your appetite on and off in much the same way that a thermostat regulates the heating system in your house. If you are watching your weight, this is important news—you can raise your blood sugar level, tame your runaway appetite any time you want to. Just eat or drink something with sugar in it. (Sugar Information Inc. 1953)[1]

Sugar Information, Incorporated, the public-facing branch of the international association of sugar producers, ran this helpful dieting tip in popular magazines—*McCall's, Good Housekeeping, Life, Better Homes and Gardens, National Geographic, Redbook, Time, Reader's Digest*—from the early 1950s through the 1960s. Sugar producers funded this advertisement campaign in response to the rising threat that artificial sweeteners posed to sugar's market share. Sugar Information ads featured young white women in pencil skirts watching their waistline, shirtless white boys chugging soda, and white-collar desk men pouring sugar into their coffee.

Ad copy promised a "New Way to Diet without Hunger . . . by keeping your blood sugar up throughout the day." Beware artificial substitutes, they warned; those sweeteners "can't help curb appetite since they have no effect on your blood sugar level" (Sugar Information Inc. 1954). Only pure sugar works to raise blood sugar level and reset the appestat. "Why not try this the next time you get hungry: Take a little sugar—in coffee, tea, a soft drink, ice cream, pastry or candy. . . . Sugar turns down your appestat, and fast. Sugar satisfies hunger" (Sugar

FIGURE 8. "Your Appestat, Sugar and You." *Source*: Sugar Information 1968.

Association Inc. and Kelly 1967, 2). Eat a little sugar, and "your hunger switches off" (Sugar Association Inc. and Tatum Jr. 1968, 2).

This image is a relic from the dawn of the neuro era. A pot of sugar reaches up with its handle arm and flips a light switch in a white man's brain. His thin black tie and white-collar shirt flag him as an Average American Man who values self-control but needs help containing his pressing hungers. He wields a sugar bowl like a pocket calculator or a home improvement device, ready to adjust his blood and lessen the strength of his feelings. Feelings are the disruptors in his bodily system.

He becomes "hungry like a bear": animal desires rise up from his blood, overcoming his mental power (Sugar Information Inc. 1953). Such desires are "runaways" and must be "trained," "tamed," "curbed," "turned down." The appestat controls the call of his animal cravings for pleasure and overconsumption. The brain switch functions unconsciously and automatically, like a mechanical feedback and control center. The body is his house, the blood his furnace, and the brain his thermostat. The brain alters feeling levels in response to information about the internal milieu. When blood sugar is low, "you're apt to eat more than you need, without being conscious of overeating before your blood sugar level is raised to the point where it 'shuts off your appestat'" (Sugar Information Inc. 1954). When blood sugar is high, "the healthy person finds it easier to turn down the extra helpings of food" (Sugar Information Inc. 1953). The ads promise their readers that they do not need to work to control their hungers. When the appestat is properly set, "you do not want so much" (Sugar Information Inc. 1954). The appestat effect is instantaneous, like an electrical circuit. The sugar switch turns desires off, "and fast" (Sugar Information Inc. 1954).

It all happens in the brain. The appestat adjusts levels of circulating substances—blood sugar—which changes the body's feeling. The stomach is curiously ignored in this message: stomachs appear incidentally in the ads as the locus for fat and "weight," the inner contents of a slimming waistline. Taste, too, plays no role in this circuit, as the ads depict it. On the contrary, the intense flavor of artificial sweeteners appears a decoy. They may taste sweet, but they cannot flip the brain switch. These Sugar Information ads invoked bodily control, homeostasis, self-regulation, and informed consumer choice guided by expert scientific knowledge, to *sell people more sugar*. Scientific expertise appeared here in service not of public governance but of commerce. The consumer in these ads did not require skills of self-regulation and self-government. No willpower was required; no reflection needed. All one had to do was to follow the advertiser's instructions. The market would govern our desires for our own good.

Sugar, perhaps more than any other foodstuff, materialized the politics of hunger after the Second World War. Sugar is historically implicated in interconnected forms of malnutrition. Sugar began the Plantationocene era; its production engaged forced enslaved labor in one of the first global agro-industrial systems (Mintz 1986; Haraway 2015). Sugar bears a history of enslavement, colonial land occupation, state support of large landowners, and racist marketing strategies (Hatch, Sternlieb, and Gordon 2019, 596). Sugar workers often could not afford food for their own families. In the Philippines province of Negros Occidental, or "Sugarlandia," nearly half of households were judged in the late 1970s to have insufficient food intake, and more than 80 percent of children malnourished (Jones 1979). Sugar sales tactics were, and still are, marked by class and race. The sugar appestat was colored pure white.

Sugar—this sweet, violent, pleasurable, exploitative substance—was flipping switches in our brains. What were these feelings that the sugar-appestat promised

to contain? Why were they "running away" and out of control? If the "hunger switch" worked automatically to maintain homeostasis, stability, and balance, as the ads promised, why would consumers need to "take" sugar or anything else to "turn hunger off"? The ads' very premise suggested that hunger was out of whack and required an external intervention. Overweight, diabetes, so-called diseases of overconsumption, appeared in the 1950s as rising threats to life and health. Even as they ran these advertisements, sugar producers already were aware by the mid-1950s that their appestat claims were weak.[2] In these ads sugar works to tamp down excess feeling, not to stimulate it. But already in the 1940s, scientists were asking whether sweetness, sensation, pleasure, and taste could themselves *cause* hunger. Researchers funded by the sugar industry discovered that people preferred to eat more sugar, at higher concentrations, than food processors ever had imagined possible. Taste testers even didn't realize that their preferred samples of canned peaches or tomatoes contained lots of sugar; they just liked the taste better. Researchers identified a "peak preference level," which later came to be called the "bliss point." Consumers, they found, chose products first by taste. They considered nutritive value only later, if they ever did at all (Pangborn 1957; Moss 2013).

Could foods that taste good make us hungrier to eat them, even when those foods do not fulfill our needs? The urge to eat sweet, fatty, or salty foods may even work against our own good health. In other words, scientists discovered that living beings hunger for pleasure. Hunger could be made and could be used as a tool for increasing consumption. This (seemingly obvious) idea caused difficulties for appestats and other theories of homeostasis. If animal bodies were designed to regulate and balance their eating to match the needs of physical activity, body temperature, and nutrient needs, why do we have a sweet tooth? How do pleasure and desire fit in? Do animals learn to desire exactly those things, which their bodies need? Some physiologists, following theories of homeostasis, believed that tastes change over time to attract us to sources of the nutrients we are missing (sugar for energy deficiency, salty foods for a salt deficiency, milk for calcium deficiency, etc.). Why doesn't food taste better when it is good for us? Hedonism and homeostasis were at war with each other. We know very well which side won.

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Harvard Medical School professor Walter Cannon (1932) popularized the term "homeostasis" in his influential work *Wisdom of the Body*. Cannon imagined the human body as an automatic regulator, a multifunction thermostat. Bodies maintained stable temperature by discharging excess heat. The lungs and digestive organs kept blood sugar, oxygen, and alkalinity at constant levels by eating and breathing. Kidneys acted as "spillways," evacuating "surplus" sugar, salt, water, and other materials. Cannon called these functions homeostasis, meaning a regulatory process that maintains bodily stability. "In the evolution and behavior of living beings," wrote Cannon (1941, 1), "the trend towards security has been one of the

outstanding features." Hunger played an important role in homeostasis, because it drove people to seek what they were missing. Bodies got hungry when they needed something to balance their internal state.

By the Second World War, problems were beginning to appear with the homeostatic model. Despite their best efforts at education and administration, wartime experts failed to motivate Americans to follow a scientifically balanced diet. The US Quartermaster designed soldiers' ration packets so that each item contained a durable, portable "quota of vitamins, minerals, proteins and calories." But soldiers threw piles of these perfectly nutritious (and presumably gross) items into garbage dumps across the theaters of the world war (Dove 1946, 187). Attempts to rationalize diets on the home front did not fare much better. Anthropologist Margaret Mead (1943, 43), serving as executive secretary of the Committee on Food Habits of the National Research Council, warned in 1943 that Americans needed to change their diets, because "our people are not as well nourished as they could be in view of the food resources we have." The National Food and Nutrition Board suggested that Americans of all classes were suffering from undetected latent malnutrition (Biltekoff 2013, 53). Americans refused to eat the way scientists thought they should. Given wartime labor needs and supply issues, this was a national security problem (Biltekoff 2013, 46–79; LeBlanc 2019, 89–125).

By military directive the Office of the Quartermaster General set up a new Food Acceptance Division to figure out why soldiers were throwing away their rations. "For the first time in history," noted one Division report, US soldiers "lived for long periods of time solely on commercially produced and processed foods" (Dove 1946, 188). Scientists working at the Food Acceptance Division set out to predict which processed products soldiers would eat and which ones they would refuse. The Food Acceptance Division, alongside Mead's Food Habits unit at the National Research Council, became a cradle for the study of taste and pleasure. Scientists at the Division studied the neurophysiology of taste and smell as well as the psychology of food preferences. They measured and classified different odor and taste thresholds. The Division developed a nine-point hedonic scale for taste tests, which could be used to predict food choice and consumption (Meiselman and Schutz 2003, 200–203). After the war Food Acceptance Division scientists brought their consumer research methods to the processed food industry.

One of the first scientists to contract with the US Quartermaster Food Acceptance Division was physiological psychologist Paul Thomas Young, who became a founder of the modern science of pleasure ("Paul Thomas Young" 1965, 1085). Young was convinced that homeostasis was wrong. Hunger was not about needs or bodily regulation. Instead, Young saw hunger as a feeling of anticipation and enjoyment, an expectation of pleasure. Hedonic hunger was fueled not by the stomach nor by contents of the blood but by the sensory, "proprioceptive" pull of tastes like sweetness (Young 1949, 108). Lab rats led the way to Young's world of pleasure and vulnerability. Young ran rats through series of food preference tests:

he set out two foods, usually liquid solutions of sugar and casein (milk protein), in the two ends of a Y-shaped box, so that rats had to choose between one path and the other. He observed what efforts rats would make to get access to sweet stuff rather than the good-for-them source of protein. He measured how fast the rats ran and how much pain they overcame to get to sugar. He observed how much sugar they consumed at different concentrations.

Young's rats ate for sweetness, not for health. Despite attempts to "train" them to prefer other foods, the rats always came back to sugar. Even protein-deficient rats ran faster to sugar than to casein. They ate sugar even when they were satiated. "Psychologists can abandon the view that dietary need is essential for adequate motivation with food," he concluded (Young 1948a, 310). His rats were clearly not eating because they were hungry or needed more energy. They were eating because sugar tastes good. Young called this the "palatability" factor. "One can argue," Young (1948a, 296) wrote, "that rats take what they need to maintain homeostasis. One can also argue that rats take what they like (find palatable) regardless of need."

These rats' hunger served not need but pleasure. Hedonic hunger, as Young imagined it, was provoked by the qualities inherent in foods. "Palatability" was an affect, a feeling of pleasure or displeasure, "the immediate affective reaction (liking or disliking) of an organism which occurs when a food stimulus comes in contact with the head receptors" (Young 1948a, 310). Chemoreceptors in the mouth, nose, and gut lit up in response to a palatable stimulus. In the hedonic theory of hunger, eating is "a chemoreflexive act" (Young 1941, 152). Perhaps, as another researcher suggested, "sweeteners may also act to prime the appetite" ("Session 2B" n.d.). In other words, *the taste of sugar makes us hungry*. Hedonic hunger did not involve scarcity, motivation, or learning. Rats did not learn better when they were hungry for sweet-tasting foods. They might be more "motivated," running faster and overcoming challenges to get to sugar, but they did not learn to get through a maze any quicker for sugar than for protein (Young 1947, 66). Nor could the rats be trained to prefer a less palatable food, when given the choice over time. Hedonic theories disconnected hunger from work and learning. This was all about pleasure (Dror 2016, 247).

Hedonic pleasure was not the same as the "reward" that Edward Thorndike and the behaviorist psychologists offered to their animal subjects. Young's lab rats were not motivated by lack or deprivation: in most experiments they had eaten a full standard diet before being exposed to the sugar and casein. They ran because they anticipated "enjoyment": "when we use the word enjoyment we are thinking of the palatability of a specific food, the affective response of an animal" (Young 1948b, 284). In his experiments rats consumed far more sugar and salt than their bodies required. They consumed more when the liquids tasted stronger; their greatest intake was at a liquid sugar concentration of 18 percent. The amounts they ingested varied with taste, not with bodily needs (Young 1948a, 301).

Hunger for pleasure might even drive animals to self-injury. Young's rats were willing to submit to strong and painful electric shocks to reach the sugar at the end of their box. Young's colleagues found that magnesium-deficient rats refused to eat magnesium when offered. They speculated that magnesium deficiency actually might feel good: "It is well-known that a feeling of well-being is not always associated with the best possible physical status nor the ultimate welfare of an individual. Examples that may be cited are those of alcoholism, narcotism, and the euphoria that may occur in high altitude anoxia" (Young 1952, 251). Pleasure could lead to dangerous things. Perhaps, Young (1948a, 293) proposed, people who craved sweets, leading to overweight and disease, had inherited more sensitive taste receptors than other people's. Perhaps some people inherited especially strong sensitivity to sweetness. Young pointed to known genetic determinants for taste and olfaction. Some parents and their children could smell a certain substance at low concentrations, while others failed to notice it at all. Perhaps "innate differences in sensory structures, especially in the senses of taste, smell, and touch, may explain why some individuals select food wisely and others do so less wisely" (Young 1948a, 293). Hunger depended on an internal tendency to become aroused, on how strongly one feels a sensation.

Perhaps some people experienced stronger reactions to pleasure than others. Unlike theories of homeostasis, which took all animal bodies as self-knowing and self-balancing, hedonic theories differentiated those who were particularly vulnerable to pleasure, sensation, and overconsumption. Sugar industry–funded researchers found that while both wild and domesticated rats preferred liquids that tasted sweet, they consumed vastly different amounts. Wild rats consumed 12 percent more calories of a sugar solution than a standard liquid; domesticated rats drank 87 percent more (Kare 1969, 49–56). G. C. Kennedy (1952, 579) found that rats with fat bodies responded more strongly to a food's palatability, eating smaller amounts of unflavored foods and far greater amounts of palatable foods than nonobese control rats. Some chemosensors appeared more porous and vulnerable than others.

Processed food producers paid keen attention to this emerging science of taste, pleasure, and preference. The Food Acceptance lab's methods spread through corporate America, as alumni went on to work in the consumer research divisions of the Coca-Cola Company, the Pillsbury Corporation, the Lipton Corporation, and Hunt-Wesson Foods (Meiselman and Schutz 2003, 203). Division scientists ran experiments in the 1960s where panels of taste testers were asked to compare the hedonic value and relative acceptability of "different flavors of a single product type." The study's authors then took their pleasure-magnitude scale to Hunt-Wesson and later worked as consultants to multiple large food-processing companies (Moskowitz and Sidel 1971). Lead author Harold R. Moskowitz (1971, 388) studied perceptions of differences in the taste of sweetness and the feeling of sugar's "pleasantness, or affective dimension." Moskowitz became a renowned expert in teasing

out subjects' preferences for subtle variations in flavor composition and famously innovated new niche varieties of common processed foods from pasta sauce to sodas (Moss 2013, 50–51).

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A sugar pot reaches up and flips a light switch in the brain: the Sugar Information ad (see Figure 8) drew that imagery straight from the emerging field of neural physiology. In the early 1950s neural physiologists located a "hunger switch" in the brain's hypothalamus. By poking brains in specific areas, scientists made animals eat uncontrollably or starve themselves. At first, scientists believed that they had discovered a mechanism for homeostasis. The hunger switch, they thought, would flip on or off in response to bodily needs. But they soon found that things turned out otherwise.

Targeting a single brain site led to dramatic results. For decades, physiologists had noted that patients with damage to the base of their brains began to eat uncontrollably. Animals with damage to the same area grew far fatter than their peers (Hetherington and Ranson 1940). Yale physiologists John Brobeck and Bal K. Anand used a thin needle with an attached electrode to lesion highly targeted areas at the base of rats' brains. They found that injury to a specific site in the middle of the rat's hypothalamus caused it to eat continuously, so much that its body grew extremely obese. Some of those operated rats slept continuously, and others became irritable and aggressive. "Five of them were really vicious," biting their handlers at every opportunity (Anand and Brobeck 1951, 128). When one of Brobeck's rats died, he wrote: "I did an autopsy and found the gastrointestinal tract from the pylorus all the way up to the incisor teeth tightly packed with chewed up chow pellets" (Brobeck 1993, 226). The rat ate to death.

A second kind of operation, which damaged a different area at the sides of the hypothalamus, turned rats into passive ascetics who refused to eat altogether. Rats with lesions on the sides of their hypothalamus stopped eating and starved to death. Anand and Brobeck (1951, 138) believed that they had found the on and off switches for the "urge to eat": one part of the hypothalamus turned hunger on, and the other inhibited, or turned it off. They called these sites the brain's "feeding center." The hunger activated by this brain switch, though, did not resemble the hunger that behavioral psychologists produced by depriving rats of food. None of the usual motivation tests (maze learning, lever pressing, running speed, overcoming painful electric shocks, lifting weights) worked on the lesioned rats. Behavioral psychologists assumed that they could measure the intensity of hunger by how animals reacted to those tests. Food-deprived rats with normal brains increased their performance; the hungrier rats were, the harder they worked to get food.

But operated rats, with a damaged hypothalamus, showed no interest in working or overcoming challenges. They did not expend any extra effort to get food and

had no interest in food that didn't taste delicious. When food appeared, especially highly palatable food, they just kept eating and did not stop. The operated rats challenged psychologists' belief that they could understand hunger by watching rats work for food. Work played no role in this new experimental setup. Brain switches, not behavior, ruled this new kind of hungry rat (Miller, Bailey, and Stevenson 1950). Something more than food deprivation appeared to make these rats hungry. These rats did not eat because they needed to. They ate in response to neural signals, to chemosensory pleasure, to context and cues. They ate for deliciousness. This kind of hunger had no clear limit or end. At its extreme this insatiable hunger even led to death.

Sugar ads drew their appestat claims from work that began in Anand and Brobeck's lab. The ads boasted that their claims had support from "research scientists at one of our leading universities." Although the ads named no names, they likely were referring to Jean Mayer, professor in the Harvard School of Public Health Department of Nutrition, and his "glucostatic" theory. Mayer did his PhD work at Yale University, around the time when Anand and Brobeck's stereotaxic experiments on brain lesions stimulated rats to overeat or starve themselves to death. Mayer learned to perform these operations and began to search for the mechanism that turns the feeding center on and off. He believed that he had found the "physiologic basis for the hunger state and the hunger behavior," in blood sugar (Mayer 1955, 17). Mayer was a scientist in the mold of Walter Cannon, committed to nutrition as a program of social regulation and reform. After fighting with the Free French forces during World War II, Mayer completed a PhD on vitamin A and became nutrition officer for the newly formed United Nations Food and Agriculture Organization (FAO). "I decided," he wrote, "that my duty was to help build a peaceful and prosperous international order as a complement to my five years of war against Fascism" (Mayer 1977, 179). During his brief tenure at the FAO, Mayer served on committees setting universal standards for protein and calorie requirements. He later traveled with UN delegations to Asia and Africa, including a fact-finding mission to Biafra, which led Mayer to campaign against the use of famine as a weapon of war.

In line with his political commitments, Mayer oriented his research program around homeostasis. He assumed that animal bodies were capable of regulating themselves. The brain, blood, organs, and muscles coordinated to keep inputs level with output. Mayer sought to uncover the chemical and neural pathways for self-regulation. He brought his static sensibility to the emerging science of neurophysiology. Hunger, for Mayer (1953, 16), was a regulatory mechanism: "Feelings involving desire for food or satiety . . . represent a conscious expression of one of the most precise regulatory devices in biology." Mayer, early in his career, used pedometers, stop-motion photos, and respiratory devices to track food inputs and energy outputs. Mayer found that food and activity levels did generally balance out, except that sedentary subjects tended to eat more than they needed (Mayer

1977, 182). This was Mayer's first indication that homeostasis did not function well for all subjects.

In his work on rats, Mayer noted that their brains were particularly sensitive to variations in blood sugar. Levels of glucose in the blood fluctuated more quickly than any other factor or nutrient, like fat or protein. He found that rats injected with glucose (or, relatedly, with amphetamines) would quickly lower their food intake. Mayer proposed that the brain contained glucoreceptors that were sensitive to blood sugar levels. In the mid 1950s he found that the substance gold thioglucose damaged cells in the hypothalamus and also caused obesity in rats—thus establishing a connection between glucose, a specific site in the brain, and obesity (Mayer 1977, 186). He believed that he had found the lever that turned the feeding switch on and off. "In this glucostatic view, hunger would be integrated among the mechanisms through which the central nervous system ensures its homeostasis" (Mayer 1953, 14).

But—this is the part that the sugar ads left out—most animals in Mayer's study failed to achieve long-term equilibrium between their energy intake and output. Some of his rats grew fat. The "day-to-day regulation" of fluctuating blood sugar was "not sufficient to insure constancy of body weight" (Mayer 1955, 18). The rat observations resonated with data showing obesity rates rising in the 1950s United States. Mayer spent the 1960s trying to figure out why homeostasis failed. The answer, he believed, was emotion: emotions cause "metabolic and endocrine changes, which in turn increase or decrease hunger." Emotions worked against homeostasis: they turned hunger on and off but could not "regulate" it (Mayer 1966b, 5).

In contradiction to the sugar ads citing his research, Mayer (1966a, 725) told clinicians treating obesity that "there is little to say for the extensive consumption of sucrose." "The best advice I can give about sugar in any form," he counseled, "[is] eat less" (Mayer 1972). As he took on a public and political role in nutrition policy in the 1960s, Mayer criticized food companies' misguiding messages and manipulation of consumers' emotions. In 1969, President Richard Nixon appointed Mayer chairman of an ambitious White House Conference on Food, Nutrition and Health. Mayer and his congressional allies skillfully maneuvered the conference to achieve lasting impacts on American food policy: easier access to food stamps for the very poor, the end of surplus commodity distribution, national nutrition guidelines, food labeling, and more aggressive controls on false advertising (LeBlanc 2019, 179–198).

Mayer's political work addressed a population increasingly understood as vulnerable to manipulation by food industry interests. The vulnerable person was poor, uneducated, and easily swayed by sensory information from taste to packaging and advertisement. A profile of obese Americans emerged, casting them as damaged in their capacity for managing emotion and judgment. Their vulnerability to pleasure and sensation appeared equivalent to brain damage in animals

(Frohlich 2024, 91–97). Some public health professionals directly accused food processing companies and advertisers of exploiting vulnerable populations for profit. In 1969, Dr. Tore J. Mita, a nutritionist at the Public Health Service Indian Hospital in Pine Ridge, South Dakota, condemned companies for marketing useless products to the poor: "I deplore the fact that millions of economically and educationally disadvantaged consumers are unwittingly spending their scarce money for worthless food products." Nutritionists could not afford to wait hopefully for public education to change consumer habits, in the face of "the million dollar budgets allocated by a segment of the food industry for the far-reaching communications media to glamorize and encourage people to consume innumerable 'nonfoods'" (Mita 1969, 1157). Mita (1969, 1158) demanded that the "burden of responsibility" for a healthy diet be shifted from the shoulders of consumers to the food processors themselves, who should be required to produce, market, and sell only nutritious foods.

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Sugar Inc.'s appestat advertisement might not evoke laboratory rats and brain lesions for most subscribers to *McCall's* or *Reader's Digest*. However, most viewers of the sugar ads likely would have shared some of the "pharmacological optimism," which infused 1950s American culture (Campbell 2007, 84). Tranquilizers, from chlorpromazine to Miltown, circulated widely and promised to smooth out consumers' mental rough edges. Pharmaceuticals appeared poised to solve and regulate nonconformities of all kinds. Who knows, perhaps a little spoonful of sugar just might do the same for food-related nonconformities. The notion of a substance "curbing" or "turning down" problematic desires and feelings would have been very familiar to magazine readers of this era.

The "appestat" shared many underlying ideas with mid-twentieth-century neuropharmacology. Neuropharmacology combined older psychoanalytic assumptions and more recent brain science. Desires, cravings, and behaviors could be traced to specific sites in the brain. Feelings were understood as chemical functions. Chemical substances (drugs or sugar) served as technologies for investigating the brain and altering behavior. Some people (and animals) were seen as more vulnerable than others to the pull of pleasure and "the external forces of suggestion, substance, and impulse." Those forces drew them to perform acts beyond and despite their own free will and self-control into a spiral of psychopathology. They were unable to stop, despite the negative consequences. Nancy Campbell (2007, 20) has named these factors as the defining qualities of the twentieth-century American drug addict. The same qualities were applied to overeaters.

Hunger and drug addiction research in the 1940s and 1950s co-created a template of the vulnerable, hypersensitive, damaged, and irrational brain. Excessive hunger for food and for drugs could be understood as parallel (even equivalent) biochemical-psychological "disorder[s] of desire" (Campbell 2007, 25). Many of

the same research scientists and sites were implicated in both hunger and addiction science. Access to the brain, in this era before imaging technologies, relied on direct stimulation of specific brain sites or on administration of chemicals. Two tools and two disciplines converged around drugs and food: behavioral observation and neurophysiology. Scientists poked at brains or administered substances—drugs, hormones, sugar—and observed the resulting changes in animal behavior. Behaviors and brains were reconfigured as metabolic.

This encounter of brain and behavior, drugs and food, converged in the 1940s at the Yerkes Laboratories of Primate Biology in Florida. Yerkes Lab researchers mobilized hunger as an experimental and epistemological tool for understanding brain and body function. In 1940, S.D.S. Spragg published an experimental protocol for producing morphine addiction in four Yerkes Lab chimpanzees. Spragg's work suggested that chemical addiction could happen in nonhuman animals and was not unique to the human psyche. Addicted animals appeared to challenge psychoanalytic theories of addiction as a character defect rooted in early childhood experiences or as a social-cultural construct. Spragg described addiction in purely physiological and behavioral terms. Addiction gained "a firm organic basis" (Spragg 1940, 125). He designed his experimental setup as a paired test of hunger and drug addiction. Chimpanzees were presented with two boxes, each with a color-matched key. The black box contained a banana and the white box a syringe. Food-deprived chimps unlocked the black box and ate the banana. Morphine-deprived chimps, whether or not they were food-deprived, went to the white box and removed the syringe. Chimps often handed the syringe to the researcher, sometimes physically pulling their human handlers toward the injection room. Chimpanzees in withdrawal chose the syringe over the banana, even when they were hungry. Spragg (1940, 14) interpreted both chains of action, unlocking and opening either the white or the black box, as behavioral indications of "desire."

Hunger drive tests, in which food-deprived animals solve puzzle boxes to access food "rewards," lay the groundwork for midcentury drug addiction research. Spragg suggested that drug addiction involved an "appetitive component" (Dewsbury 2003, 253). Addiction, like overeating, represented a departure from homeostasis. "Drug addiction, whether in the human or the chimpanzee subject," Spragg (1940, 125) wrote "can be considered as a state of equilibrium, the departure from which creates a condition that generates powerful motivations to restore that equilibrium—motivations that pervade the behavior of the organism and predominate over other, normally primary, desires." Hunger for bananas, hunger for morphine, desire and behavior, converged in the Yerkes Lab chimpanzee cages. In 1944, Yerkes Lab director Karl Lashley invited his former PhD student, Donald Hebb, to take up a research position there. Hebb spent the next three years meandering from studies of chimpanzee phobias to dolphin social intelligence. He expended most of his intellectual energy on a theoretical attempt to bridge the

fields of behavioral psychology and neurophysiology. This effort, published in 1949 as *The Organization of Behavior: A Neuropsychological Study*, attempted to explain what was going on in the brains of hungry, addicted, and other animals.

Hunger, wrote Hebb (1949, 200–202), was "equivalent to addiction": "Hunger established in the presence of lowered blood nutrients, and having the effect (through eating) of raising them, would be physiologically the equivalent of an addiction—biologically valuable, but still an addiction." Hebb imagined what was happening in the brains of humans and animals like Spragg's chimpanzees. Their brain cells, stimulated repeatedly by the same chemical agent (like morphine), began to interact in a fixed set of patterns. Metabolic changes, stimulated by food or morphine, set off that preformed pattern. The brain cells responded to sensations and internal chemical changes and formed regular repetitive reactions. Brain cells' patterning and direction were more complex than on-off switches, stimulus and response, arousal and reaction (Hebb 1949, 72). Brain activity was not a simple reaction to animals' bodily needs. No steady regulator guaranteed equilibrium and homeostasis (Hebb 1949, 179). What an animal did depended on the activation of already formed cell assemblies, both recurrent and anticipatory (Hebb 1949, 135). Instead of one-way switches, Hebb's brains were continuous feedback loops.

Hunger, like addiction, could not be understood as simple homeostasis. No automatic meter existed in the blood or stomach, adjusting metabolic levels to meet bodily needs (Hebb 1949, 204).. Hebb (1949, 190) cited Paul Thomas Young's argument that hunger was more than homeostasis. Some brain activity, "something like thinking," had to intervene between external sense perception and internal bodily movements (Hebb 1949, xvi). Hunger and addiction disorganized brain activity, which translated into unstable and disturbed behavior. "Even in experienced subjects the need of food has disintegrating effects," Hebb (1949, 192, 205) wrote. "The relation of hunger to emotional disturbance is notorious. . . . There is an inescapable relation between drug addictions, food habits, and chronic emotional disturbance." Deprived animals were disturbed, restless, uncomfortable, in pain. Only when hunger or addiction was sated could brains stabilize and function.

Hebb drew inspiration from Spragg's chimpanzee experiments to conceive hunger and addiction as brain-based events. Hunger and addiction were functions not of homeostasis but of neural circuitry. Neural assemblies formed associations between sensory events—the taste of food or the sensation of a syringe's needle—and feelings of euphoria or satisfaction. By repeating the same association over and over, feelings and behaviors became locked in. Spragg's chimpanzees showed some signs of relief and satisfaction even from a drugless saline injection. That simple sensation connected to past relief triggered neural cells to follow a fixed pattern. Hebb suggested that the same effect appears in the act of tasting, chewing, and swallowing: the sense experience of food brings relief long before the body's needs

are met. Hunger and addiction, for Hebb, both functioned as disrupters of cell assembly. Brain processes could easily be thrown off.

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If hunger and addiction were parts of the same scientific-neurological complex, American drug policy and food policy in the 1950s stood (and today still stand) at opposite extremes. In the realm of food, producers and marketers are free to promote their wares unfettered. In the realm of drugs, the public response is total and violent. Cold-turkey abstinence is often the only option for addicts, whether by rehabilitation or incarceration. At the Rockefeller Institute for Medical Research, Vincent Dole imported hunger research into the study of addiction, with large-scale policy implications. Dole and his collaborator, Marie Nyswander, challenged the legal and punitive dichotomy between food hunger and drug hunger.[3] Their research did not use animal subjects; it focused entirely on humans, mainly addicts in search of recovery. Dole and Nyswander's work culminated in a historic push to move drug policy away from abstinence and toward maintenance, functioning, and harm reduction.

Dole began his medical research career in the 1940s steeped in questions of drive, motivation, and deregulated homeostasis. He studied the role of human fat tissues and protein deficiency in weight gain and loss, and confirmed the value of low-salt diets in treating hypertension (Dole et al. 1954; Dole 1959; McCarty 1984). "I'd been interested in the appetite control systems," Dole (1989, 332) recalled," and I had a feeling that there was absolutely open territory in the whole question of behavior, and to what extent metabolism had to do with drive." His move from obesity to drug addiction in the early 1960s occurred serendipitously: a colleague about to begin a sabbatical year asked Dole to fill in as chairman of the Health Research Council's Committee on Narcotics. But the move fit perfectly with his research concerns at the time.

Dole questioned why researchers and policy makers treated narcotics differently than food, when both kinds of substance increasingly appeared to stimulate the same physiological processes. At the outset of his drug research, Dole (1989, 334) recalled, "the question I asked myself was, 'What's so bad about narcotics?'" Stimulating pleasure was not on its own a reason to condemn consumption. After all, he reasoned, a glass of wine or a nice meal also produced euphoric effects, to no one's concern. The only logic behind the differential treatment of the two types of substances, which both produced metabolic effects, had to be ideological (Dole 1989, 339). With Nyswander, an experienced rehabilitation clinician, Dole set up a clinical program in 1964 to put heroin and morphine addicts on a stable daily dose, to replace cravings and disorder with stability and equilibrium. This approach paralleled his experience with obesity treatment, in which clinicians adjusted levels of protein in patients' diets and observed the effect on their weight. Quickly it became clear that adjusting protein levels and morphine doses did not cause analogous effects. Narcotics acted too rapidly for the body to establish equilibrium.

But Dole and Nyswander found that patients taking the opioid agonist drug methadone were able to reach a stable state, free of the extremes of euphoria, craving, and withdrawal. Methadone patients lost their appetite for heroin and had no withdrawal symptoms; many regained stable work and family lives. Very quickly, Dole and Nyswander's methadone maintenance protocol ballooned in size. Methadone treatment spread from one clinic to a network of New York City clinics treating tens of thousands, to a national program promoted and funded by the Nixon administration (Dole 1989, 340–341). He attempted to convince a skeptical law-enforcement community, and the general public, that narcotic addiction was a disease and not a moral failing. Treating opioid addicts with methadone, Dole (1989, 341) argued, was like treating diabetics with insulin.

In his later writings, Dole drew a line straight from his research on obesity and abnormal food consumption to drug addiction. He understood both behaviors as "symptoms of metabolic defect" (Dole 1989, 338). Although we like to think that we choose what we eat, Dole (like Hebb) believed that "all ingestive behavior is in some way responsive to the biochemical state of the body." Eating and drug-taking stemmed from biochemical effects. Hunger, for narcotic drugs or food, produced metabolic changes in the body and brain. This chemical dynamic directed what people choose to do. In this, Dole drew from the lineage of Spragg, Hebb, and Young. "Behavior, even the apparently free-willed decisions of man," Dole (1965, 211) wrote, "is powerfully determined by chemical events in the organism. The addict, slave to a chemical, illustrates this dramatically."

Methadone produced an effect in Dole and Nyswander's heroin addicts, like the effect that Sugar Information Inc. promised would result from a teaspoonful of sugar. "Somewhere in the body a simple chemical change induced by the narcotic [or alimentary] chemical—a depletion of transmitter substances in neurones, for instance, or the release of hormones—changes mood and motivation" (Dole 1965, 211). A spoonful of sugar, like 40 grams of methadone, could eliminate craving, desire, and deregulation. Hunger and addiction control designed a new form of self-maintenance: biochemical maintenance of the neurophysiological self. The metabolic brain needed constant maintenance. It was vulnerable to destabilizing influences, sensations, or substances. Far from self-regulation and homeostasis, hunger appeared as a physiological disorder. Hunger captures vulnerable brains, rendering those brains' carriers unable to control their own eating. Cravings may not be under control of the conscious will, and containing them required hypervigilance. Vulnerability, here, refers to hypersensitivity and heightened pleasure. Vulnerability is reinforced by environmental seductions, palatability, sweetness. All these feelings were promised to disappear with a spoonful of the right corrective substance.

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Scientific research on food cravings led to a serious problem: If hunger works like an addiction, hooking people on foods that their bodies may not even need, what does this mean for markets? How can a free market work if consumers are

incapable of making rational choices? The ideal of the market depended on individual freedom of choice. What could that freedom really mean if consumers were misdirected by sensory manipulation, glucose dependency, and "trained" or fabricated hungers? What if the marketplace created hungers that destroyed consumers' health? *What if the market itself made us sick and hungry?* Drawing on techniques developed at the Food Acceptance Division, food processors test for the right combinations of sugar, salt, and fat that reach consumers' "bliss point" (point of strongest liking) and maximize consumption (Moss 2013). It is fair to say that many products on our grocery shelves, impoverished in nutritive content and overabundant in sensation, are food-deprived. Could foods themselves be sources of food insecurity?

Experts by the 1960s increasingly came to believe that misinformation, advertising, and branding could themselves be making Americans hungry and malnourished. Conveners of the 1969 White House Conference on Food, Nutrition and Health recommended that government agencies like the Federal Trade Commission (FTC) be empowered to control both food content and information (Frohlich 2024, 97). The Conference Final Report warned that "gaps in our public knowledge about nutrition, along with actual misinformation carried by some media, are contributing seriously to the problem of hunger and malnutrition in the United States" (White House 1970, 179). Experts called for strong government regulations on food producers' access to consumers' minds. Simply educating people about proper nutrition was not enough. The nature of the marketplace itself had to change.

In 1971, emboldened by the White House Conference, the FTC went after the sugar "appestat" ads. The FTC opened a case against Sugar Information Inc. and its advertising agency, Leo Burnett, for falsely claiming that sugar helps with weight loss. The commission charged that "respondents have represented and are now representing, directly and by implication, that . . . consumption of sugar and foods containing sugar, such as soft drinks, ice cream cones, or candy bars, before meals will result in reduced daily caloric intake." These advertisements "were, and are, false, misleading and deceptive" ("United States of America" 1972, 15). Sugar Information Inc. eventually settled the case and its two decades-long "appestat" ad campaign came to an end.

In that same year, Gerald Thain, assistant director of the FTC Food and Drug Advertising Department, publicly blamed advertisers for hunger and malnutrition: "Recently, it has become clear that, although we are a part of the most affluent society in the world existing at any time, we are not a well fed nation." False advertisements, he warned, undermined the "basic right" of all Americans to "proper food and proper nourishment" (Thain 1971, 3). False advertising prevented consumers from accessing a nutritive and healthy diet and made American hunger worse. Thain praised the White House Conference on Food, Nutrition and Health for enabling the FTC to pursue food companies who publish misinformation.

As a result of the White House Conference, the FTC opened complaints against multiple food producers for making false and unscientific statements about their ingredients. In a move that seems impossible today, the FTC did more than force companies to cease publication of misleading ads. Companies were required to publish "corrective advertisements" in the same media outlets where the original ads appeared, to inform consumers that they had been misled. Thain admitted that "the proposed corrective advertising remedies has had [*sic*] enormous repercussions. One article in the trade press recently described corrective advertising as a 'doomsday' weapon." But, Thain (1971, 7–8) argued, it was the only way to undo the harms to consumers and to the market, caused by lies and manipulation. Choices and desires stimulated falsely had to be contained and undone.

Echoing the White House Conference report, Thain listed three interconnected forms of malnutrition: deprivation, unbalanced diets, and diet-related diseases. The ills of poverty, misguided spending, and overconsumption were all expressions of the same problem: a market out of whack. This was a collective, national problem in need of a collective solution. To emphasize that Americans share a universal stake in the problems of hunger and malnutrition, Thain appealed to an imagined tradition: "Today many concerned Americans, viewing what they consider to be the deterioration of the quality of life, yearn for the life pattern of an earlier, simpler era . . . [when] we were a nation which expected and presumed pure, healthy foods flowing forth from the rich land." Since that imagined time (the memory of which itself effaces the violence inherent in obtaining land), the complexity and expansion of food markets had complicated consumer choices. People learned about food indirectly and via mass media channels. Malnutrition had become an American problem, in part, because of corporate manipulation (Thain 1971, 2–3).

In testimony opposing the new, more activist FTC policies, General Mills Corporation chairman C. W. Cook echoed the same wistful origin story but with a different ending. Cook presented the big food producers' objections at an FTC hearing in October 1971. In his testimony he recalled his mother buying milk and butter for her family from the local farmer. Those days, however, were in the past. "Tremendous changes in the technology of food processing and distribution . . . have made us a far better fed people than we would have been, despite the fact that we still have a way to go before we achieve optimum national nutrition." Advances in food production, Cook warranted, could not have happened without advertising to inform consumers how to find products. Advertising brought new and beneficial goods to consumers. "We in business . . . feel that the public interest is also served when the consumer is offered a maximum of freedom of choice in the marketplace under a system of fair competition" (Cook 1971, 3–4).

The FTC debates articulated a basic question: How does consumer choice respond to hunger? What does freedom of choice mean in the context of sensory manipulation, media messaging, palatability, and preference curves? Thain and

Cook recognized that something had shifted in postwar America. Sugar producers themselves noted that by 1960 two-thirds of sugar consumed in the United States came in the form of processed goods, in "a complete reversal of conditions which prevailed before World War II" (Hickson and Sugar Association Inc. 1960, 33). Nourishment was mediated in new ways, which both produced and alleviated hunger. Market-generated hungers generated financial opportunity and consumer choice, and also ill health and malnutrition.

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In the wake of the Sugar Information Inc. settlement with the Federal Trade Commission, the sugar industry turned to more subtly effective information campaigns. The newly formed International Sugar Research Foundation subsidized scientific research favorable to the industry. Foundation-sponsored research directed public attention away from sugar's most dangerous effects on heart disease, diabetes, and obesity, and instead focused on the single issue of tooth cavities. The solution to a high-sugar diet, they proposed, was to brush your teeth. Sugar's critics were branded as "food faddists." As Christine Kearns and her colleagues found in their analysis of advertisements and publications from the 1970s and 1980s, the Sugar Association followed the tobacco industry playbook for "information laundering" (Kearns, Glantz, and Apollonio 2019, 15).

In 1973 popular magazines printed a full-page spread of a white woman extending her arm toward the reader, her hand holding a glistening caramel-topped ice cream sundae. The heading above her promised to give "the plain truth about your sweet tooth." The ad copy read: "Do you recall the messages we brought you in the past about sugar? How something with sugar in it before meals could help curb your appetite? We hope you didn't get the idea that our little diet tip was any magic formula for losing weight." Dieting is "complicated," it continued, and "research hasn't established that consuming sugar before meals will contribute to weight reduction or even keep you from gaining weight" (Sugar Information Inc. 1973). This was the corrective advertisement mandated by the new FTC guidelines, as part of its settlement with Sugar Information Inc. The advertisement's stated purpose was to reeducate the consumer and undo the harms of misguided appeals. And yet the lustrous image of an ice cream sundae beckoned. . .

Sugar might not switch off the appestat, the ad concedes. But it feels good. Having printed a retraction statement as required by its settlement, Sugar Information Inc. pivoted to a new theme. The FTC settlement order specified that future ads were allowed to include "accurate representations of refined sugar's role in and contribution to a balanced diet . . . [and] accurate representations of any non-nutritional characteristic of refined sugar" ("United States of America" 1972). This characteristic, in the 1973 ad, was pleasure. Sugar, the ad continued, "is a food you enjoy." With this corrective ad, Sugar Information Inc. left behind the electrical switches and homeostatic regulation of the 1950s and openly embraced hedonism.

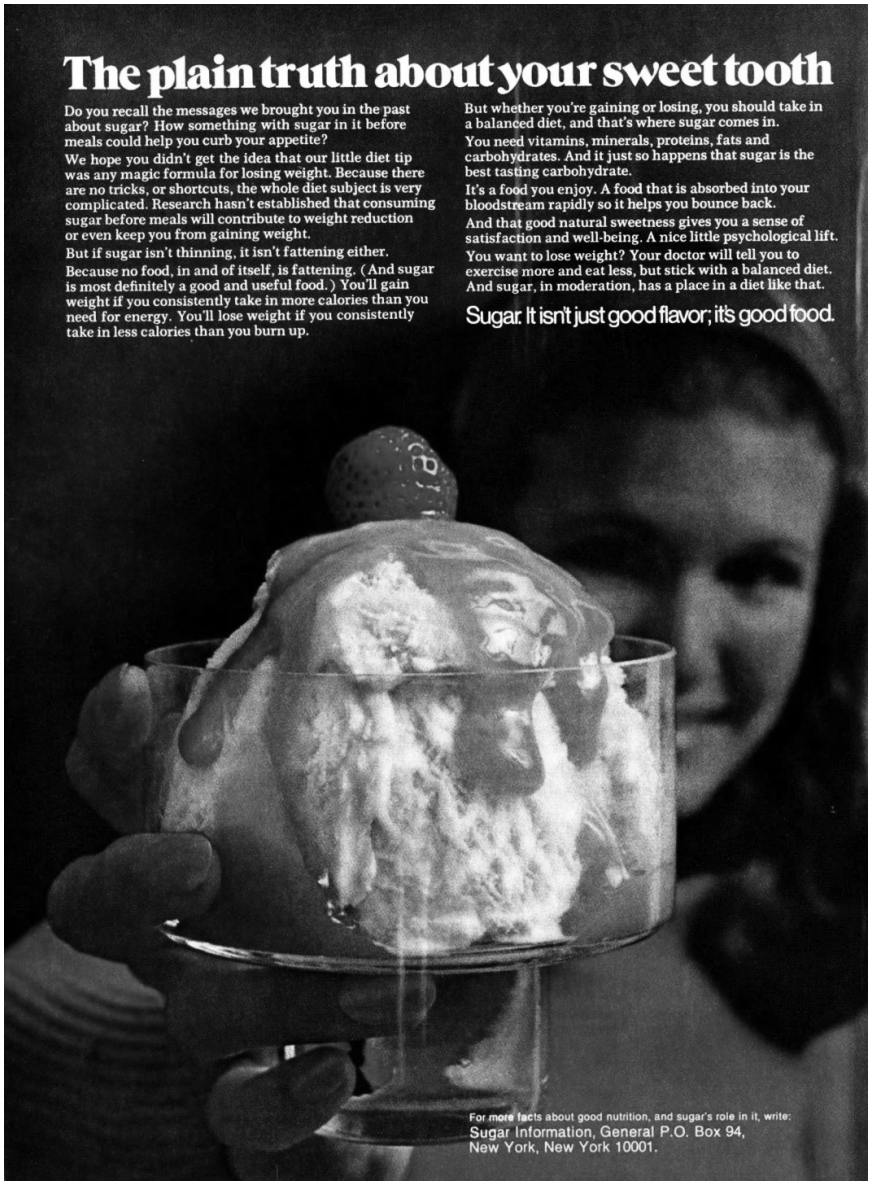

FIGURE 9. "The Plain Truth about Your Sweet Tooth." *Source*: Sugar Information 1973.

The ad promises that sugar will make you feel good. "It helps you bounce back." Drawing from psychological language, Sugar Information offered consumers pleasure. Sugar "gives you a sense of satisfaction and well-being. A nice little psychological lift" (Sugar Information Inc. 1973). In the end, hedonism and marketing, as we all know, won.