

Ozempic

Who gets to escape captive markets and deprived productions? In 2023 a global craze erupted for the hunger-reducing drug Ozempic (semaglutide, also sold as Wegovy). “Vitamin O” invaded the news and social media. Some consumers publicly trumpeted their weight loss success; others hid their use of the drug to avoid social stigma and judgment. Though Ozempic had been approved to treat diabetes, many diabetics struggled to fill their prescriptions as off-label users ate up the available supply (FDA 2023). Many eager weight-loss consumers turned to illicit internet purchases of semaglutide compounds. Users without insurance coverage paid \$900 for a month’s supply of Ozempic, or \$1,350 monthly for the higher dose of semaglutide in Wegovy (Synott 2023).

As one might expect, access and usage followed class divides. Rich neighborhoods were consuming Ozempic at twice the rate of low-income areas that were more impacted by metabolic diseases (Goldstein 2023). Sales grew so high that by late summer 2023, the market value of Danish pharmaceutical company Novo Nordisk, producer of Ozempic and Wegovy, was larger than the rest of Denmark’s entire national economy (Nelson 2023). Ozempic promises consumers a pharmaceutical escape from hunger. This chapter considers the science of hunger suppression, desire, longing, and capitalism. I trace how Ozempic could come to be understood by one of Novo Nordisk’s own research affiliates as a project of cultural Marxist pharmacology. Food companies—in collusion with federal agriculture and trade policies, starvation wages and punitive welfare benefits—are making many people feel terribly hungry. As Julie Guthman (2015) has pointed out, human bodies are a prime target of (food) industrial growth. When “the body becomes an accumulation strategy,” bodies are pressed to absorb surplus production and provide outlets for capital investment. Agro-industrial companies grow by expanding their

market. Not just in competition with other producers, but also by expanding bodies' capacity to consume (Guthman 2015, 2527). Palatability, marketing, and synthetic ingredients all combine to "make ultra-processed products liable to harm endogenous satiety mechanisms and so promote energy overconsumption"—in other words, to make eaters hungry for more (Moodie et al. 2013, 671).

Food companies saturate the world with *nutritus*. Corporate strategy, industrial food design, and global deregulation combine to perpetuate hunger. Food-processing corporations have been designing food to get consumers to eat more since at least the 1950s (recall our discussion of the sugar industry in chapter 5). Starting in the 1970s, trade liberalization and deregulation opened low- and middle-income countries to processed food marketers. Phillip Baker and colleagues (2020, 2–5) have observed that as a country's income rises, ultraprocessed foods shift from a consumer good for the wealthy to a product for the poor. The ultimate frontiers of market expansion, globally, are the economically oppressed. At the same time, as market expansion for processed foods slows in the global North, another outlet for capital investment is growing exponentially: the pharmaceutical market for hunger suppressants (Guthman 2015, 2531).

What work is semaglutide doing? What is the nature of the hunger that it suppresses? "My relationship with hunger, and therefore with eating, is transformed," wrote *Washington Post* commentator Ruth Marcus in 2023 about her experience on Ozempic. "I leave food on my plate, untouched and unlamented, and do not look at the food on yours with the same longing: 'Are you gonna eat those fries?'" For another consumer the drug "shut off the intrusive constant thoughts about food" (Belluz 2023). A user named MandyM wrote in 2019 on *WebMD*: "I've lost [weight] and all interest in food or alcohol. Not caring or even thinking about food or your next meal and not worrying about how you will resist food is incredibly freeing." ("User Reviews for Ozempic" 2023). "No wonder that skinny people think heavy people have no willpower," another Ozempic consumer wrote. "Their brains actually do tell them to stop eating. I had no idea" (Tolentino 2023). A user named High Praise explained on *WebMD* that after starting Ozempic, "I'm not always starving, just feel like a normal person" ("User Reviews for Ozempic" 2023).

A user named Chris posted on *WebMD*: "Ozempic has given me the gift of low appetite" ("User Reviews for Ozempic" 2023). Chris received their lack of hunger as a gift. To no longer want, to no longer desire to consume, is for them a thing of value. Wanting, for Chris and for many other consumers posting on *WebMD*, became something so aversive that they are willing to endure great expense and physical challenges (and to consume a different kind of substance, a drug) in order to stop. Ozempic provides these users relief from thinking, longing, caring, wanting, resisting. In that opening of negative space, in that area of feeling and being that semaglutide suppresses in its users, I believe that new understandings of hunger may become clearer. This kind of hunger is a combination of longing, wanting, and thinking—a constant, fixed, involuntary attention. Hunger here implies a

heightened sensitivity to changes in the nature-culture in and around us: shifts in the climate and built environment, economy, sociability, culture, and affect. Hunger responds to the world entering the body (in the form of food) and orients the body toward the world (by directing thoughts and desires). In an extractive, violent, and stochastically changing world, hunger expresses a gut feeling that things are not right.

Ozempic may release some of the burdens of hunger and longing in a violent, consumerist, and antifat world. In sociologist Scott Vrecko's (2010b, 555) words, obesity drugs offer a "means of protecting individuals from a hostile modern environment" of "hyperconsumption." The drug acts within a social and physiological milieu shaped by global market forces, social norms, expert and lay knowledges, and individual practices. Ozempic reshapes relations of longing. It leaves consumers like Marcus indifferent to the full plate of fries laying before her. For some users semaglutide makes moving through a hyperconsumptive world more bearable. And yet there is a cost to this relief. Hundreds of Ozempic reviewers on *Drugs.com* and *WebMD* recorded experiences of nausea, bloating, vomiting, diarrhea, headaches, and fatigue. Users threw up at work and while driving and had uncontrollable bowel movements that lasted all night. "If you do eat," wrote Stringa007, "you have to force yourself to eat because of the pain, dizzy spells (especially going from sitting to standing), stomach gurgles almost back to back all day, and did I mention how bad the diarrhea is? It's like giving yourself a virus that won't go away" ("User Reviews for Ozempic" 2023). For many, the side effects diminished after a few weeks of use. Some continued to feel mild nausea punctuated by "sulfuric burps." Others found themselves incapable of functioning. "I have been unable to basically leave my house for days," wrote a user named Lola ("User Reviews for Ozempic" 2023). Lola, like many users, tried to power through extreme side effects in the hope that after a few weeks or months they would subside. Those who chose to stop the drug found that its effects lasted for days or weeks.

Some users wrote that they nearly stopped eating altogether. Not only did they lose interest in food, they no longer felt any desire to eat at all. MandyM wrote: "I actually started worrying about how little I wanted to eat." GR posted: "As I began week 4, my appetite is now so reduced that I barely eat anything at all. I have to force myself to eat a meal, and even then I feel sick after a few bites." SRose wrote: "I have actually lost more weight that I intended to and can't seem to make myself eat to pick my weight back up. . . . For anyone wanting to use it for weight loss it's a hell of a way to lose it as you definitely won't have an appetite but you also find yourself feeling weak because you haven't ate anything substantial" ("User Reviews for Ozempic" 2023). Mimi wrote: "I don't have ANY appetite at all and cannot face food. . . . Yes, the 'food noise' has gone, and I no longer overeat or binge, but I feel like everything is suppressed" ("Ozempic User Reviews" 2023). User Susanne Brown told the *Global News* that "taking Ozempic is akin to 'doctor-approved anorexia.' . . . When she first started on Ozempic, it led to dangerous eating habits,

where she would ‘eat two pieces of cauliflower and be full.’ If a patient so desires, they could stop eating entirely by taking more and more of the drug” (Mannie 2023). Some Ozempic users were literally starving. British physicians reported that they treated a woman who had bought semaglutide online, without a prescription, for symptoms of starvation (Sivaraman and Kozhippally 2020).

Reading their posts, I get the impression that these users have internalized, turned outside-in, the violence of the external world, which Ozempic had promised to alleviate. Instead of feeling compelled to respond to and to resist the cues and demands of food out in the world, these users have turned inward. They have ceased to nourish themselves. They are nauseous, bloated, and unable to ingest. They are starving themselves. These semaglutide consumers are starving themselves so that the food market cannot control them and make them always feel starving. Unlike hunger strikers and hunger marchers, who find collective identity and meaning in their actions, drug takers are profoundly alone, except perhaps on forums like *WebMD*. I sympathize. Perhaps nausea and aversion are the most consistent, logical, and preservative responses to this world.

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Hunger is a scientific goldmine for two reasons. First, hunger is a key to solving what has come to be known as a crisis of obesity. Experts by the 1970s routinely characterized obesity as an epidemic (Nordsiek 1964).¹ This language of crisis is fueled by antifat discourse and by claims that “excess weight” causes metabolic, cardiovascular, and respiratory diseases. As many scholars have pointed out, such claims involve a reductive flattening of personal, biological, and cultural difference and complexity (Yates-Doerr 2015a and 2015b; McCulloch and Hardin 2013, 7). The obesity crisis nevertheless mobilizes scientists, public health experts, mass media, and public concern (Guthman 2015, 2531). More than a personal diagnosis, obesity is framed as a collective menace: “Obesity is no longer only the problem of individuals but of whole populations and nations” (Ošancova and Hejda 1975, 57; see Biltekoff 2013, 121). The specter of ballooning health-care costs places responsibility on overweight people for threatening the future fiscal health of states and communities (Dickinson 2019, 120). As the driver of a potentially costly epidemic, excess hunger becomes everybody’s business.

Second, hunger suppression is a financial windfall in a world ruled by the politics of thinness. Body shaping has become a collective project of citizenship, social value, labor productivity, and personal attractiveness. It requires hard work to maintain a body that is capable of being productive in the labor force and that is politically, socially, and personally normative (Dickinson 2019, 122).² People who deviate from a thin, white, hetero, American norm are labeled as “undesirable populations” and are disciplined to “fix” and control their bodies (Choudhury 2022, 18; Strings 2019; Williams-Forsen 2022, 89–140; Harrison 2021). Hunger-suppressing drugs promise to make people desire less and consume less, and

therefore become candidates to be employed and desired (Cottom 2023). An effective drug that reduces hunger can earn billions, as Novo Nordisk has experienced with Ozempic.

Weight loss regimens are designed on the premise that the overweight subject must be motivated to become thin and healthy. The results are often violent and humiliating, when patients are blamed for their own lack of motivation to lose weight (Choudhury 2022, 16). The American Academy of Pediatricians recommends “motivational interviewing” as a first step in the treatment of childhood obesity (the final step being a prescription for diabetic or anticraving medication). In the motivational interview the pediatrician “identifies and reinforces a patient’s [or parent’s] own motivation for change” and “guides families to identify a behavior to change” (Hampl et al. 2023, 47). Readers of chapter 2 in this book will recognize the figure of motivation and all that it implies. Motivation, as a psychological construct, relies on a structure of pleasure and pain, punishment and reward. As we know, the original motivation experiments starved animal subjects and lured them to work with small tidbits of food. Motivation as a frame comes out of a behavioral model of human psychology that is based on deprivation.

I should not have been surprised when I discovered that the inventor of behavioral weight control was a psychologist who starved his subjects to get them to work. But I was! In 1962, Charles Ferster published a study on “The Control of Eating.” Ferster was a protégé of B. F. Skinner, in whose lab Ferster trained pigeons to peck incessantly at a small disc or lever to get bits of food. Ferster learned to use rewards and punishments as reinforcers to make his subjects behave in specific ways. Most important, he kept his subjects—whether pigeons, chimps, or children—in a constant state of hunger and food deprivation; then he tracked their “performance” on lever pressing or puzzle solving (Ferster, Nurnberger, and Levitt 1996, 403). In Alexandra Rutherford’s (2009, 7, 43–47) words, Ferster used hunger as a “technology of behavior.”

Ferster spent two years early in his career at the Yerkes Primate Lab, where he deprived chimpanzees of food so that they would do experimental work. He maintained his chimps at 80 percent of their free-feeding weight; in other words, he subjected the animals to nearly the same regimen as the one that the Minnesota Starvation Experiment used to study the effects of extreme food deprivation in people. The chimps were made to respond to rewards (small bits of food) or punishments (in the form of time-outs) (Ferster 1958). Senior researchers at the Yerkes Laboratory were appalled by Ferster’s handling of his primate subjects. One colleague recalled that “there were lots of comments made at weekly staff meetings about the psychology of half-starved animals” (Dewsbury 2003, 257). Yerkes lab director Henry Nissen complained that “you try to starve them into submission and some animals would starve themselves to death. They’d just quit working. It was like they’d lose appetite” (quoted in Dewsbury 2003, 258). Nissen believed that starvation contributed to the deaths of two chimpanzees at the lab (Dewsbury

2003, 258). Skinner later complained that “tender-hearted colleagues frustrated [Ferster’s] efforts to reduce chimpanzees to a satisfactory state of deprivation” (Skinner 1981, 261).

In 1957, Ferster moved to the Indiana University Medical Center. There he applied the same experimental setup to a group of autistic children in the university hospital. Instead of imposing a starvation diet on the children, he forbade them to eat between meals. Then he exposed them to learning tasks that could earn them food or candy (Ferster and DeMyer 1962, 93–95). At Indiana University he also began to study a method of behavioral self-control for weight loss. He recruited twelve nurses from the hospital and led the women through an experimental dieting protocol based on stimulus and response, reward and punishment (Rutherford 2009, 106). Ferster seemingly did not believe in hunger as such. In his view, consistent with behavioral psychology, subjective feelings of hunger were both unmeasurable and irrelevant. “Hunger pangs, which are ordinarily taken as symptoms of hunger (from which the effect of food deprivation is inferred), are more closely related to the conditioned stimuli accompanying past reinforcements of eating than to the level of food deprivation” (Ferster, Nurnberger, and Levitt 1996, 403). In other words, hunger feelings were a result of psychological training rather than physiological need.

Ferster sought to retrain his twelve subjects’ hunger feelings and eating patterns. He set out to make eating—especially pleasurable eating—feel like punishment. By pairing “pies, cakes, cokes, doughnuts, or candy” with a “known aversive event,” these foods “may become conditioned aversive stimuli” (Ferster, Nurnberger, and Levitt 1996, 402). If a psychological link could be created between food and punishment, pain or displeasure, food itself would become undesirable. In practice, this meant focusing the women’s attention on the “ultimate aversive consequences” of their own eating. Ferster and his colleagues trained the women to pause in the middle of a meal and verbally rehearse how eating would bring them pain, misfortune, shame, or exclusion. The researchers “probed” the women to discover “instances where overweight has affected daily life functioning, . . . pleasure, occupation, etc.” (Ferster n.d. [ca. 1958]). Each woman had her own repertoire of ultimate aversive consequences (UACs).

According to archival notes on a group training session, Ferster’s colleague Gene Levitt placed a piece of pie in front of each of the women in turn and asked them to recite their UACs. Miss H- said that her “Spencer corset created an unsightly roll of fat on her body which she felt was noticeable.” Mrs. M- “noted that her husband is losing weight rapidly and that she would ‘die of mortification’ if she stopped losing weight and he caught up with her.” Levitt went so far as to grade the subjects’ recitations; Mrs. M- got “no better than B or perhaps B-” (Ferster n.d. [ca. 1958]). No one ate the pie, of course. Recitations of UACs were supposed to happen in the middle of meals and every time the women encountered a dessert, soda, or piece of candy. Ferster predicted that with repeated aversion training, the subjects’

“feelings of hunger should disappear except just before meal-time” (Ferster, Nurnberger, Levitt 1996 [1962], 405).

Needless to say, that hasn’t worked out. Ferster is truly one of the most disagreeable scientists I have encountered in the historical record. Somehow, however, behavioral self-control remains the standard first line of treatment for people judged as overweight. As soon as news emerged about Ferster’s weight loss study, he began to receive letters from women pleading for his help. Mrs. G. R. Zeller (1961) wrote from California: “Recently I read an article . . . [stating] that you and your colleagues had some measure of success, that is in creating the idea of aversion to food. . . . I am at least [many pounds] overweight. It is impairing my health—my mental outlook and certainly my disposition. . . . Is there anything you can suggest to do?” Mrs. S. J. Smith (1961) wrote from Seattle on behalf of her “Fatties Club,” “the members of the club and I haven’t been very successful in taking pounds off or keeping off what we do remove. We were wondering if you could possibly have any other helpful ideas we could use.” Ferster’s self-control method found a broad popular audience in a 1972 mass-market self-help book, *Slim Chance in a Fat World* (Rutherford 2009, 108). Slim chance, indeed.

By the time that Ferster published his behavioral self-control method, the American marketplace was already saturated with weight-loss drugs. A parade of amphetamines and their derivatives—Benzedrine, phenmetrazine, phentermine, fenfluramine, ephedrine—filled pharmacies and medicine cabinets, one after the other. By the late 1960s, some 2 percent to 3 percent of the American population, or four million to five million people, were using amphetamines for weight loss (Rasmussen 2009, 238). As one weight-loss medication was banned because of toxicity, psychosis, or addiction, another took its place. Ozempic is the latest in a series of hunger-suppressant drugs, each derived from a new scientific understanding of hunger and how to stop it.

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Ozempic exists because scientific research on hunger and appetite has become increasingly precise and granular. Researchers manipulate mice genes to knock out specific neuroreceptors or hormones and observe what changes result in the genetically modified mouse’s eating preferences and behaviors. Real-time MRI scans track which brain regions light up when human subjects look at images of food, with or without injections of specific peptides or hormones. Nutrition labs house subjects for a day or a week, expose them to precisely calibrated kinds of food, survey their subjective hunger feelings, and track the peptides and hormones circulating in their blood. Scientists have isolated a dozen hormones and neuropeptides that influence hunger, appetite, and fullness: ghrelin, insulin, and leptin hormones for long-term energy regulation; Neuropeptide Y and CART (cocaine and amphetamine-regulated transcript) neuropeptides that stimulate hunger and fullness, respectively; and gastrointestinal hormones and peptides that sense the

contents of the gut, control gastric emptying, and promote fullness. Semaglutide amplifies the work of one of those gut peptides, GLP-1.

Pharmaceutical appetite suppressants track closely with scientific developments in hunger research. Amphetamines made users more sensitive to the switches that turned appetite on and off. Addiction theories of hunger led to anticraving medications, which were designed to stop consumers from feeling pleasure. Ozempic and comparable drugs replicate the action of peripheral gut peptides. Hopes for a hunger on/off switch were revived with the discovery of the hormone leptin in 1995 (Friedman 2012). Leptin administered directly to the brain reduced food intake, and leptin deficiency caused increased eating. Five years later, the hormone ghrelin was found to produce the opposite effect (Kojima et al. 1999; Gil-Campos et al. 2006, 201). Newspapers and magazines breathlessly reproduced images of rats with and without leptin, one stick thin and the other obese. However, initial hopes quickly tapered. These hormones fail to turn off hunger effectively under conditions of increasing consumption (Schwartz et al. 2000, 662). In addition, proof that human bodies were unable to regulate themselves appeared evident everywhere. Changes in consumer patterns and body sizes across the twentieth century suggested that bodies were not set up to defend any fixed set point, whether of sugar, fat, or body weight.

The first wave of anti-food craving drugs, which came out in the 2000s, targeted the brain's pleasure receptors. As food addicts were defined as dangerously sensitive to sweetness, pleasure, and palatability, anti-craving drugs were designed to reduce pleasure. Certain neuropeptides could turn up or down pleasurable sensations. Neuroscientists identified specific neural circuits for hedonic sensation, "overlapping brain systems . . . [that] mediate drug addiction and feeding" (Dagher 2009, 30–32). Cannabinoid and opioid receptors in the brain respond to both food deprivation and intensely pleasurable tastes (Di Marzo, Ligresti, and Cristino 2009, 3). The brains of animals exposed to a high-fat, high-sugar diet had dysfunctional dopamine pleasure receptors (Hopkins and Blundell 2017, 75). Opioid and cannabinoid antagonists were found to block perceptions of pleasure. When food-deprived subjects took the anti-craving drug naltrexone, which blocks opioid receptors, sugar tasted less delicious than it had previously (Wassum et al. 2009).

Anti-craving drugs allowed people to view themselves, and act on themselves, as diseased and in need of medication (Vrecko 2010a, 43). If a drug can "cure" hunger, the corollary must be that hunger is a biological disease. "It is long past time to stop shaming people with disorders of appetite in a futile attempt to tame our own fears of loss of control," argued columnist Maia Szalavitz (2023). Addiction appears as a biological problem rather than a problem of self-control or will-power. If a drug can turn off the desire to eat quickly and fully, perhaps that desire was never under conscious control to begin with (Sanabria 2015b). Still today, most research on hunger targets the brain, manipulating pleasure receptors in the

central nervous system. But it turns out that tinkering with the brain's cannabinoid, opioid, and dopamine receptors causes unwanted and sometimes severe psychiatric side effects (Vrecko 2010b, 567). Pharmacological innovation in that area has slowed as regulators forced these drugs off the market. Some scientists argued that the focus on blocking pleasure in the brain misses out on a whole realm of bodily experience.

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"There is a tendency to . . . [go] straight to the brain," complained Gerard Smith in 1982. When scientists wanted to understand what made animals and people hungry, they looked to identify the neural mechanisms. "But this move is too neat," Smith cautioned. Looking for hunger and motivation in the brain "[reduced] psychology to neuropsychology." The brain alone could not reveal the full dynamic of what it meant to be hungry. "To go straight to the brain is to study the brain. But . . . motivation is not a thing in the brain but a relationship that involves the brain" (Smith 1982, 134).

In the 1970s, Smith was one of a group of researchers at Weill Cornell Medical Center who began to study appetite and eating from the opposite end of the hungry brain. They focused on the gut and asked what factors make animals stop eating. First, they sham-fed rats who had not eaten all night: food entered the rats' mouths and exited through a tube before it reached their stomachs. Those rats, whose food never touched their gut, ate and ate for hours on end, almost indefinitely (Young et al. 1974). It seemed, then, that something happened in the gut, or in the relationship between the gut and the brain, to make animals stop eating. Evidence of some factor circulating outside of the brain already existed: in the 1950s G. R. Hervey connected the blood flow of two rats, one of which had a lesioned hypothalamus that made it overeat. The second mouse, sharing the over-eater's blood, stopped eating and starved (Hervey 1959). The Weill Cornell group set out to find what it was that traveled between those parabiont rats.

Thanks to the work of gut endocrinologists, the Weill Cornell researchers knew that the stomach and intestines produced their own hormones (Bloom and Polak 1978). They found that cholecystokinin (CCK), a hormone secreted when food enters the small intestine, made rats eat less. The more CCK they injected into a rat's intestine, the less it ate (Gibbs, Young, and Smith 1973, 488). When either the gut hormone or food entered their stomachs, rats performed the same "satiety sequence" of behaviors that rats usually do when they are ready to stop eating (Gibbs and Smith 1982). CCK apparently made rats feel full. Over the following decades, endocrinologists identified more than thirty gut peptides involved in appetite, leading some to call the gut the "largest endocrine organ in the body" (De Silva and Bloom 2012, 12). The gut—the stomach, intestines, and fat tissues—all seem to independently experience hunger and fullness.³

One gut hormone, ghrelin, increases stomach movement and makes one hungry. The other gut peptides act to delay the stomach from emptying, release digestive enzymes, stimulate glucose production in the liver, and reduce hunger. Two of the most important gut peptides, PYY and GLP-1, act through the vagus nerve to the hypothalamus (De Silva and Bloom 2012, 12). Endocrine cells in the gut have receptors that taste food, “the gut equivalent of the taste buds on the tongue that sample various components of ingested food” (Barrett 2014, 34–37). The gut releases hormones in response to specific tastes. An umami tastant alone in the gut (without any food attached) can make people feel less hungry (Cummings 2015, 717). In sum, *hunger is a gut feeling*. What does it mean to say that a gut, rather than a person, is tasting and reacting to food?

Gut feeling is a way of thinking about hunger that attends to sites outside of the brain, peripheral sites of sensing, knowing, and responding. Gut feelings also refer to feelings that escape conscious awareness and control. In the gut model of hunger, intestinal cells and fat cells are agents, which themselves can feel hungry or full. Eaters may not even be aware of what their guts and tissues are sensing, knowing, and feeling (Sanabria 2015b, 135). This model of hunger considers feelings that do not correspond to hedonic pleasure but rather to an unconscious sense of compulsion, of wanting something. Or the opposite of compulsion, a sense of satiety, not wanting. Emilia Sanabria (2015a) has noted that this becomes a problem—when a body can no longer be trusted to know its own limits and needs—in a context of a broken food system, in which a focus on individual eaters obscures broader cultural and corporate forces.

A new theory of affect emerged to describe this situation, when hunger and desire feel compulsive, unconscious, biological. Psychologist Kent Berridge suggested that hunger and desire should be analyzed as a collection of separate components. Berridge distinguished between *liking* something, meaning taking pleasure from it, and *wanting* something, which was more like a craving or a longing. Berridge (1996, 1) argued that these were two entirely separate processes, with distinct neural substrates. “Liking” implies a drive toward pleasure (hedonism). Regardless of bodily needs, whether one has already eaten, the pure pleasure of food can bring one back for more. “Wanting” (sometimes referred to as “implicit wanting”) suggests an unconscious draw to consume something that may or may not fulfill a need or even produce pleasure. One can even want something and not like it (Berridge 2004, 195). Wanting compels one to focus attention and longing on a particular substance. Wanting is a gut feeling.

Researchers performed all sorts of experiments to separate out wanting from liking. They placed tastants (like sweetness without sugar) on rats’ tongues to measure liking. They asked people how much they liked different kinds of food, then tested how hard the same subjects were willing to work to acquire those foods (which was meant to measure wanting). People who scored high on a scale of

binge eating seemed to have an unusually high implicit wanting for high-fat sweet foods, even when they said that they didn't like those foods any more than others. Those people "wanted" sweet and fatty foods more than they "liked" them (Blundell and Finlayson 2011, 1217). Researchers gave other human test subjects the same food to eat repeatedly. The more the subjects ate, the less they liked it (Berridge and Robinson 2003, 509; Finlayson, King, and Blundell 2007, 989). But how much of it they ate did not seem to change how much they wanted it. Implicit wanting, researchers concluded, must be independent of homeostasis. One could keep wanting something even if one didn't *need* it, or even *like* it (Finlayson, King, and Blundell 2008, 126). The opposite of wanting, satiety (not wanting), also appears to function mostly at an unconscious level. Fullness just happens, without thought or will.

Well before Ozempic, medical interventions in the gut made patients stop wanting food. Before there were gut peptide drugs, there was bariatric surgery. In the early 1950s surgeons in Sweden and the United States noticed that patients who received bowel restriction surgery, for treatment of various diseases, tended to lose a significant amount of weight. Thus began decades of restrictive surgeries, which cut up and bypassed parts of the stomach and intestine, to get patients to lose weight. By 1970 more than thirty thousand intestinal bypass operations had been performed, although it soon became clear that the side effects of intestinal surgery were too dangerous to tolerate. The gastric bypass (diverting part of the stomach and intestine) was first practiced in 1967 and quickly became the most prevalent weight loss surgery (Celio and Pories 2016, 656–664). In the year 2018, some 250,000 bariatric surgeries took place in the United States (Lynch, Kozak, and Zalesin 2022, 1).

Following bariatric surgery, many patients no longer felt hungry. They stopped wanting food. Researchers found that bariatric surgery altered gut peptide activity (Imamura et al. 1984). It wasn't just that a smaller stomach stopped them from absorbing as much food. They no longer had a desire to eat. Mentally, they knew that they needed food, but affectively they did not feel the need (Lynch, Kozak, and Zalesin 2022, 4).⁴ Even more powerfully, postsurgery patients felt satiated, overfull, every time they ate. Bariatric patients interviewed by Amanda Lynch postsurgery complained that they experienced "painful feelings or 'being sick' from taking 'one last bite'" (Lynch, Kozak, and Zalesin 2022, 5). Patients after surgery said to Lynch and her colleagues, "I don't think my body knew what hunger was" before surgery, and "I never really understood full" (Lynch, Kozak, and Zalesin 2022, 6). "Eleanor," who underwent a sleeve gastrectomy, told Lynch, "I either didn't recognize the signals before or they are much more acute now. The stomach I have now, I think it has that brain connection" (Lynch, Kozak, and Zalesin 2022, 6). Patients postsurgery said that they no longer felt guilt or regret when they were full, and that their hunger feelings were less intense and emotional (Lynch, Kozak, and Zalesin 2022, 7).

Drug researchers looked to bariatric surgery for clues to how to suppress hunger. Some asked, “Could the altered gut hormones following gastric bypass be sending ‘fullness’ signals resulting in sustained weight loss?” (Wren and Bloom 2007, 2125). In rat models, directly injecting gut hormones produced similar effects as bariatric surgery. If a drug could manage to deliver a combination of gut hormones that “mimic natural satiety mechanisms,” it could replace surgery with the same effects (Wren and Bloom 2007, 2126). Ozempic answered that call. Enter the Gila monster. Gila monsters are the largest lizards in the United States and one of a few species of venomous lizard. They live in desert arroyos and semiarid rocky regions of the Southwest United States and northern Mexico. According to the National Zoo, Gilas have been known to eat only three or four large meals (of birds, eggs, small mammals, and amphibians) in an entire year. They carry stores of fat, move slowly, and have very low metabolism (Smithsonian National Zoo n.d.). Gila monster saliva contains the hormone exendin-4, a particularly powerful peptide that resembles human GLP-1, but is stronger and longer lasting.

A synthetic form of the Gila hormone, Exenatide, was the first GLP-1 drug to be approved for diabetics. Exenatide reduced blood sugar, slowed the stomach from emptying, and reduced hunger (University of North Carolina 2007). But early GLP-1 drugs metabolized quickly and required daily injections. Novo Nordisk figured out how to prolong the effect for a week at a time, and Ozempic was released in 2017. “Injecting ourselves with this lizard juice,” worried a user named Sane on *Drugs.com*, “can’t be good for us” (“User Reviews for Ozempic” 2023). Gila monster saliva (or rather, a synthetic version of it) is now teaching human bodies, especially the gut, how to stop feeling hungry all the time.

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Physiological psychologist John Blundell, working with Novo Nordisk, tested semaglutide on human subjects. He found that semaglutide made users want less. They lost appetite, felt fewer food cravings, and avoided fatty, energy-dense foods (Blundell et al. 2017, 1249). Blundell and his colleagues at the University of Leeds specialized in research on wanting and satiety (not-wanting). They applied Berridge’s schema of liking versus wanting experimentally and developed tools to measure the strength of implicit (unconscious) wanting (Finlayson, King, and Blundell 2007, 2008). At some point in the 2000s, Blundell and members of his research group seem to have become convinced that something extreme and dramatic had to be done about overeating. This is where it becomes really interesting for someone like me, who studies science, technology, and society. Blundell came to view this specific form of hunger, wanting and longing, as a product of late-capitalist overproduction. Furthermore, a solution, at least in Blundell’s view, is to defend oneself from capitalism with pharmaceuticals. Blundell’s work on Ozempic is a form of cultural Marxist pharmacology.

The fundamental problem, as Blundell and his colleagues see it, is capitalism. Desire and longing, in late-capitalist society, are sick. Overconsumption is everywhere, powered by the economic drive to expand markets. People are enticed to buy ever more clothes, gadgets, cars, appliances, and processed foods; as Blundell points out, only the latter is blamed for a global health emergency (Blundell and Finlayson 2011, 1216). The commercial marketplace pushes out ever tastier foods to attract ever more consumption. As a result, the drive to consume, perpetuated by abundance, advertising, and ease of access has damaged the general structure of desire and longing. Consumers are wanting more, far more than they need, and are never satiated. “Given this situation (abundance, palatability and promotion), together with the operation of a powerful and well- functioning reward system in the brain, it is a surprise to us that the level of obesity is not even higher than its current level” (Blundell and Finlayson 2011, 1217). Obesity is only one side effect of an all-encompassing consumer society.

Industrial food processors have our number (Sanabria 2015a; Moodie et al. 2013, 671). They know the “bliss points” for doses of sugar, fat, and salt that make foods hyperpalatable (Drewnowski and Greenwood 1983; Moss 2013). They know that liquid sugar drinks and membranous cheese puffs slip past our mouths so quickly that our taste sensors miss how much stuff just went in and leave our stomachs so quickly that the gut barely tastes them (Cassady, Considine, and Mattes 2013; Mozaffarian 2022, 1447). (The Sugar Research Foundation has known this since 1969 [55].) Food companies know that people tend to eat more ultraprocessed foods, more quickly, than minimally processed foods, even when the two types of food are similar in calories, fat, and sugar (Hall et al. 2019; Small and DiFeliceantonio 2019, 347). Processors know that they can get away with substituting cheaper synthetic alternatives—artificial flavorings, preservatives, enzymes, emulsifiers, gums, trans fats—for whole ingredients (Guthman 2015, 2529; Baker et al. 2020, 10–12). They know that human bodies can adjust to accommodate increasing food intake but tend to forcefully resist eating restriction and weight loss (Blundell and Finlayson 2004, 23; MacLean et al. 2017, 9). Companies know that environmental cues and tastes learned in childhood shape lifelong appetites (Halford et al. 2008; MacLean et al. 2017 11–12). They know that many people are stressed, overworked, with little time to care for themselves and their families, getting paid starvation wages and inadequate welfare benefits (Eyer and Sterling 1977; Chilton and Rabinowich 2012; Cheon 2021).

Food processors know that ultraprocessed meals, drinks, and snacks at bargain prices (subsidized by federal agricultural policy) offer affordability, convenience, and pleasure, and sometimes they are the only feasible alternatives in a captive marketplace. The only possible response to this situation must be to repair the whole psychological structure of desire and wanting. Current approaches that focus on behavior change and weight loss, or even taxing sugary sodas, will not help much. Telling people to lose weight and exercise more just makes them feel bad about

themselves and does nothing to move the needle. The only social intervention to date that significantly reduced obesity, Blundell notes, was the economic blockade of Cuba in the 1980s and 1990s. National shortages of food and gas forced Cubans to eat less and to walk instead of driving. But that kind of violent social engineering is neither generalizable nor desirable. Besides, the world economy depends on easy access to food and fuel (Blundell 2018, 1305; see Garth 2013). Attempts to reduce the supply of unhealthy food, or to intervene on the consequences of wanting, like weight gain, are doomed to fail. Instead, the structure of desire itself must be altered. People must be helped to “manage their drive to consume” (Halford et al. 2010, 255).

It does not help simply to exhort people to eat less. This is not within any one individual’s mental power. For Blundell and his colleagues the issue is “a hedonic process operating at an unconscious, compulsive level (measurable by implicit wanting)” (Blundell and Finlayson 2011, 1217). To change the way people eat, one must change what they want, lessening desire for some foods and reinforcing desire for others (Halford et al. 2010, 256). Desire, longing, and wanting must be redirected away from capitalist productions and promotions. In other words, to suppress an out-of-control hunger, one requires the tools of the Frankfurt School. Blundell cites Marxist philosophers Herbert Marcuse and Max Horkheimer to suggest that the capitalist system “controls people’s behavior to the same extent as an overtly authoritarian regime.” Capitalism drives people to behave in ways that do damage to themselves. Under such conditions, Blundell asks, is a scientific solution to obesity even possible? “Can biological mechanisms or psychological processes be revealed that are strong enough to resist the political and economic forces of a capitalist system, which is the basis for the world’s business?” (Blundell 2018, 1305). Could scientists ever come up with a drug, a surgery, or a psychological intervention, for resisting capitalism?

Blundell wrote this reflection at roughly the same time as his research group was working for Novo Nordisk to investigate the effectiveness of semaglutide. I imagine that he must have had semaglutide in mind when he asked whether a biological mechanism exists, which was strong enough to counter the effects of capitalism. The Leeds group had for some time been calling for an antiobesity drug that would alter “our innate attraction to food and susceptibility to over-consumption” (Halford et al. 2010, 265). Ozempic, it appears, is (at least for some of its developers) a technology for surviving capitalism. I did not see that coming from a physiological psychologist working for a drug company.

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Three conclusions emerge from this Ozempic story: First, industrial food producers design their products to increase consumption—in other words, to keep people hungry. This has serious consequences for health and well-being. Some people suffer deeply and experience their relationship with food as a constant battle

against their own bodies. Fat shaming, diet culture, and motivational weight loss programs amplify the harm. Responsibility for managing this situation falls on the backs of individual consumers.

Public discussions of Ozempic and obesity almost never mention the companies that make the food, which makes people sick. Commentators argue over whether people are or are not to blame for what they eat, whether overeating is an addiction, a disease, or a bad habit, and whether Ozempic is a proper or viable response. Some commentators lament racial and economic inequalities, the ubiquity of fast food joints, the paucity of fresh food in some neighborhoods, or the generally unhealthy food environment. But they almost always lay the burden—and shame—of managing all this on individual consumers. This situation is not new, and is not coincidental. When experts in the 1970s and 1980s warned about the ill health effects of eating too much sugar, the sugar industry responded with targeted interventions focused on the importance of daily tooth brushing to avoid cavities (Kearns, Glantz, and Apollonio 2019, 15). Corporate-funded narratives emphasize uncertainty about the causes of weight gain, even when experts agree that there is a clear correlation between the rise of metabolic disease and industrially processed foods. Food companies are systematically and scientifically manipulating our taste sensors, hormonal responses, and brain reward circuits. They are treating our sensory systems as “alienable commodities” (Spackman and Lahne 2019, 145). Their foods make us hungry by design. These corporations should be held responsible.

The second conclusion is that hunger, desire, and longing are historically determined. This is what Blundell decided when he read Herbert Marcuse while his lab was testing Ozempic’s psycho-physiological effects. Consumer culture channels desires, wants, and needs in directions that reinforce domination. This is a broader way to make the point above about food companies manipulating the body’s hunger responses. As Marcuse (1991 [1964], 5–6) has argued, the dominant culture creates and reinforces needs that perpetuate toil, misery, and “the struggle for existence.” Hunger is manipulated and mobilized in the service of consumerism, wage suppression, overwork, and cultural norms like anti-Blackness and fat shaming, all of which cause damage. People struggling to stretch their paycheck, catching something fast to eat between shifts, or punishing themselves for perceived food transgressions have that much less energy to devote to things that will make them truly happy and well. Marcuse (1991 [1964], 5) called this situation “false needs,” when people’s desires are manipulated in the service of their own misery. But you don’t need to subscribe to totalizing, binary judgements about true and false needs to recognize that our hungers are being played with in ways that do not serve our collective well-being. Even, in fact, in ways that perpetuate violence and harm.

The third conclusion: Ozempic tells us to pay close attention to gut feelings. If hunger and desire are historical, collective, and mediated by capitalism, then liberation goes with changing the structure of desire. Ozempic shows that such a

radical restructuring is possible and that many thousands, even millions, of people are yearning for it. If only it could be done collectively, without inducing permanent nausea and astronomical medical expenses. Kent Berridge's (1996) work on wanting and liking suggests that there are subtle distinctions in the structure of desire, distinctions that many help us to recognize and respond to manipulative cues. Wanting, as Berridge describes it, is a feeling of craving or compulsion. You can want something, strongly, terribly, even when it gives you no pleasure, you don't like it, even when it is damaging. As Blundell recognized, that kind of wanting lines up with something like what Marcuse called false needs. If we take liking in its broadest sense—not just a fleeting sensation of pleasure but full enjoyment—liking offers a counterfeeling to the compulsion of wanting. What might happen if people were able to carefully, slowly, mindfully pay attention to what they truly like?

As Da'Shaun Harrison (2021, 20) has written, "thinness, as a politic, demands that one consume less, desire less, rather than make the demand that we end a World where what one desires would leave others without." Is another politic possible? Is there a different, collective mechanism, a different hunger, strong enough to resist the political and economic forces of a capitalist system? Gut feelings might lead us away from hungers that harm and toward desires that bring enjoyment. The first step toward this kind of desire is to be able to truly feel—to recognize and to analyze—our gut feelings. What is the nature of this particular hunger? Is it a numbing of pain? Which parts of this hunger feel compulsive, which parts feel pleasant, and when? Before, during, after consuming something? Listening to gut feelings requires developing the skill of interoception (Chen et al. 2021; Harshaw 2008; Berntson and Khalsa 2021). Could a careful survey of gut feelings inform a critical analysis of one's own desires?⁵

Antidieting experts encourage eaters to respect and respond to their own gut feelings. The authors of the popular antidieting book *Intuitive Eating* propose a set of principles including "Reject the Diet Mentality," "Make Peace with Food," "Respect Your Body," "Honor Your Hunger," "Feel Your Fullness", and "Discover the Satisfaction Factor." Intuitive eating encourages readers to feel and follow their own hunger cues; the authors ask readers to measure their feelings on a ten-point Hunger Scale, ranging from (1) famished to (5) satisfied to (10) painfully overfull (Tribble and Resch 2012, 68–69). Intuitive eating practitioners propose a form of "gentle nutrition," by which the body's feelings of pleasure, satisfaction, and wellness guide eaters toward foods that are good for nourishment and well-being. What if listening to gut feelings went beyond a scale of strength of feeling? What if gut feelings could also tell us something about the ways longing and desire are channeled?

What if this technique expanded beyond individual bodies, to include inquiry about the whole structure of food production, consumerism, and desire? Gut feelings are physical, social, emotional, and cultural. Gut feelings are also triggered

by food shaming, fat shaming, white cultural norms, and anti-Blackness. Scholar Psyche Williams-Forsen challenges the moralistic, commodified messages that direct African Americans to eat or not to eat certain foods. Williams-Forsen (2022, 126) has argued that “we, as African Americans, need to be transparent about how we feel after eating certain foods: sluggish, satisfied, satiated, happy, cranky, and so on. . . . We should decide how foods feel in our bodies.” What gut feelings might a universal basic income produce? What about legally mandated one-hour meal breaks and regular, predictable working hours? What hungers might be relieved by redirecting federal agricultural subsidies to support sustainable food systems? Ozempic shows that a different kind of wanting is possible. But we don’t have to be satisfied with the version sold to us in the form of an injectable pen.