

PERSPECTIVE

In defense of pleasure: We need to rethink food reward and obesity

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Is overeating driven by hedonism? We argue that the pleasure of eating food is not a driver of the obesity epidemic, but rather the regular consumption of an unhealthy diet blunts sensitivity to interoceptive signals that drive food reward.



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Abbreviations : GLP-1, glucagon-like peptide-1; HFD, high-fat diet; UPFs, ultraprocessed foods.

All organisms need to acquire energy to sustain life. To optimize this behavior, the values of foods are learned and updated based on experienced physiological benefit. For example, illness following food consumption leads to the formation of a conditioned taste aversion so that the food can be avoided in the future. By contrast, when consumption leads to nutrient and energy availability, a conditioned preference (flavor nutrient learning) is formed, and the value of food cues and flavors increases, motivating acquisition and intake at a level that equates to physiological value. Importantly, whereas conditioned taste aversion is associated with a host of unpleasant conscious sensations, nutrient and energy sensing is largely subliminal and interoceptive, being triggered when nutrients are detected in the upper intestine or energy use is detected in the hepatoportal vein. These subliminal neural signals are carried by the spinal and vagus nerves to the central nervous system to release dopamine and support learning and value updating, acting as a primary reinforcer [1].

Critically, although such subliminal reinforcers can influence conscious perceptions (for example, by increasing liking), they can also cause conditioning and influence behavior and intake independently of conscious “hedonic” correlates. For example, in humans, such post-oral interoceptive reinforcers condition increased intake [2] and responses in the nucleus accumbens and hypothalamus to flavors [3], and they do so completely independently of the effects of conditioning on the conscious perception of liking. This demonstrates a dissociation between circuits coding for what we refer to as “high road” (e.g., delicious flavors) versus “low road” (subliminal post-oral) reinforcers [1]. Moreover, despite being subliminal, these low road signals are both necessary and sufficient to sustain food reward and therefore act as the critical

unconditioned reinforcer behind food reward [4]. By contrast, the conscious sensation of food, including encounters with food cues and delicious flavors, are conditioned secondary reinforcers that are neither necessary nor sufficient to sustain reward in the absence of post-oral interoceptive reinforcement [4].

Although both high road and low road pathways and their integration are critical for adaptive food decisions, the bulk of evidence indicates that high road hedonic signals are not to blame for the obesity epidemic. Nevertheless, researchers continue to promote the idea that hyperpalatable foods lead to obesity and to target “hedonic” systems in the search for new therapeutic targets. We believe that this framework is problematic and would urge a shift of focus towards interoceptive reward signals and gut–brain learning, rather than hedonic behaviors.

If hedonic (i.e., pleasurable) signals are driving the obesity epidemic, then foods linked with the obesity epidemic should be more pleasurable, and those who experience more pleasure from these foods should have higher body mass indices.

Following the same logic, the glucagon-like peptide-1 (GLP-1) agonists that are so effective at reducing food intake should blunt the pleasure derived from food. However, although ultraprocessed foods (UPFs) have been linked to the obesity epidemic and found to promote overconsumption compared to minimally processed foods, the UPFs used in these studies were not rated as more liked, and therefore must promote food intake by some other means [5]. Likewise, although increased sugar intake is related to increased rates of obesity, extreme sweet-likers have greater lean mass, but not fat mass. They also have higher interoceptive accuracy for both generic (heartbeats) and gut-based (gastric satiation and fullness) signals [6]. This suggests that increased liking reflects a sensitivity to higher metabolic requirements rather than representing a driver of obesity. Furthermore, GLP-1 agonists do not appear to reduce food intake by reducing food liking [7]. Therefore, it is unlikely that eating for pleasure drives the obesity epidemic.

Instead, there is strong evidence from animal models that habitual consumption of unhealthy diets rewires both hypothalamic and midbrain-striatal circuits to promote overeating and obesity risk by reducing sensing of nutrients by the digestive tract, thereby decreasing the value of less energy-dense foods, producing compulsive and impulsive behavior, impairing memory, and reducing taste perception [8]. Many of these effects are observed in the absence of weight gain or metabolic change [9]. While experimental use of diets analogous to those used in animals is challenging in humans, studies that use mild, naturalistic dietary interventions do show neurobehavioral alterations that are similar to what has been reported in rodent models. For example, healthy weight individuals randomized to consume a high-fat and high-sugar versus an equicaloric low-fat and low-sugar snack daily for 8 weeks show decreased liking of a low-fat test food, despite no differences in weight gain or metabolic measures [10]. In animals, a high-fat diet (HFD)-induced decreased preference for low-fat foods can be recovered by increasing post-oral signaling [9], suggesting that the decreased preference for low-fat food observed in humans [10] may reflect reduced post-oral sensitivity to fat. Accordingly, nasogastric delivery of lipids to humans with healthy weight, but not to those with obesity,

decreases striatal response, an effect that does not reverse following a 10% reduction in body weight [11]. Exposure to the high-fat/high-sugar versus low-fat/low-sugar diet also revealed enhanced neural responses to food cues and enhanced sensory association learning. This is consistent with a large literature linking food cue reactivity in humans to risk for weight gain [12], as well as with animal studies showing that exposure to habitual HFD increases impulsive responding (an effect that is associated with decreases in dopamine receptor subtype 2 signaling) and can do so without increases in body weight [13].

Collectively, the findings from human and animal studies converge to suggest that it is decreased sensitivity to the subliminal interoceptive signals that are generated during nutrient digestion, coupled with increased stimulus-driven intake, that drives overeating, rather than a heightened hedonic signal. This conclusion is consistent with the incentive-sensitization theory of addiction [14]. In the case of feeding, it also highlights a critical contribution of interoceptive sensitivity. However, much remains unknown regarding this gut–brain system. Characterizing the trajectory, dose, and reversibility of diet-induced neuroplasticity will better inform the development of effective treatments that restore signal fidelity, the creation of regulations on the food industry that limit obesogenic foods, and the design of dietary guidelines that protect interoceptive function. This reorientation from hedonic pleasure to gut–brain communication represents a necessary next step in addressing the global obesity epidemic.

Author contributions

Conceptualization: Dana M. Small.

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