Introduction to the Special Issue: Homeostatic vs. Hedonic Feeding

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Guest Editors

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The notion that food intake is controlled by two parallel processes - so-called homeostatic and hedonic feeding - is a well-established dichotomy in the feeding literature (e.g., 1 - 4). Within this conceptualization, homeostatic processes ensure that we eat when hungry and stop eating once sated, so that food intake matches energy expenditure. It has been proposed that these processes act in parallel with, and can be overridden by, hedonic processes, which encompass the rewarding, palatable and pleasurable properties of food, often involving learning and habit. As such, hedonic feeding has been invoked to explain how obesity has become rampant despite the presence of adaptive homeostatic mechanisms that should guard against excess consumption (e.g., 5 - 6). As befits the idea of parallel, often competing processes, it was thought that homeostatic and hedonic feeding are generated via distinct underlying mechanisms. Homeostatic feeding has been described as the function of peripheral signals (e.g., gastrointestinal satiation and satiety signals, adiposity signals), and certain brain regions (e.g. hypothalamus and nucleus of the solitary tract), while hedonic feeding involves other brain areas including mesolimbic circuits and the neurotransmitter dopamine.

Although this parallel process model has been highly influential and permeated the field, an ever growing body of evidence suggests that the distinction is less clear-cut than previously thought. In fact, recent research points to homeostatic and hedonic processes being intertwined and heavily dependent on the same underlying structures (e.g., 7 - 10). These interactions are evident in the abundance of reciprocal projections between brain regions traditionally considered to be part of the homeostatic or hedonic circuitry. Further, many of the peripheral hormones that influence feeding, traditionally considered to be homeostatic signals, act more broadly in the brain than previously appreciated, including effects in the mesolimbic reward system. This special issue of *Physiology and Behavior* provides a collection of papers that should challenge and inform thinking about the dichotomy between homeostatic and hedonic feeding, and consider whether other frameworks for the interaction between homeostatic drive states and reward mechanisms may be more useful to the field in the future.

Many papers in this issue address the ways in which hormones and brain circuitry traditionally considered to be homeostatic mechanisms control feeding, at least in part, through actions on reward systems, and affect broader aspects of learning and motivated behavior. Kern and Mietlicki-Baase (11) review literature on the pancreatic hormone amylin and describe how it acts at receptors throughout the brain to impact motivated behavior including eating and alcohol intake. Konanur and colleagues (12) report that glucagon-like peptide 1 receptor activation suppresses phasic dopamine responses to food-predictive cues, providing new insight into how this caudal brainstem neuropeptide may impact motivation. The lateral hypothalamus has long been acknowledged as a site where homeostatic and hedonic signals interface (13), and the paper by Lee and colleagues (14) proposes a role for hypothalamic orexin and melanin-concentrating neurons in mediating behavioral transitions necessary for feeding. The review by Burdakov and Peleg-Raibstein (15) argues that despite its longstanding association with homeostasis and other regulatory functions, the hypothalamus has a primary role in memory updating. Carr (16) reviews cellular mechanisms by which food restriction modules the rewarding value of drugs and associated cues, and hypothesizes that this is an adaptive response to food scarcity.

Some of the papers in this collection address how non-homeostatic factors such as context and cues may impact eating and food choices. Greiner and Petrovich (17) present data suggesting that while rats initially show neophobia to novel food, they come to prefer that food with repeated testing, and in contrast, novel environment has a robust intake-suppressive effect that appears to be longer-lasting in females. Sadler and colleagues (18) explored the characteristics associated with sensitivity to food reward in humans, and report that BMI and susceptibility to food cues may be important factors.

Two papers deal with the ways in which fatty acids impact ingestive behavior. Figlewicz and Witkamp (19) provide a comprehensive review on the role of fatty acid signaling in the control of feeding. Zhao and colleagues (20) report evidence that the sequalae of gastrointestinal infusion of fatty acids alters sensitivity to food but not to other types of reward.

Finally, several reviews in this collection focus on disorders including obesity. Ferrario (21) reviews data supporting the idea that individual differences in incentive motivation and NAc plasticity play a role in vulnerability to obesity and difficulty in maintaining weight loss. The influential concept of “liking” and “wanting” as dissociable components of reward is reviewed by Morales and Berridge (22), who discuss neural mechanisms and clinical implications. Berthoud and colleagues (23) examine the question of why overeating and obesity happen given the existence of homeostatic regulation of eating, and put forth a hypothesis for how obesogenic environments impact the brain circuitry central to both energy homeostasis and food reward. Lowe and colleagues (24), review the literature on individual differences in weight variability, and suggest that higher weight variability, independent of baseline BMI, is predictive of future weight gain and may be a risk factor for poor clinical outcomes.

Together, the papers of this special issue provide an update on the traditional homeostatic vs. hedonic model, and suggest a number of future directions for new research.

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