

COMMENTARY

Commentary on: “Neurobehavioral Inhibition of Reward-driven Feeding: Implications for Dieting and Obesity”

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I was a reviewer of Applehans' article (1) and applaud his integration of neurobiological and psychological research on reward-driven eating and its inhibition. Indeed, I am in agreement with the majority of points he makes. The purpose of my comments is to provide some differing perspectives specifically on his treatment of hedonic feeding inhibition to spur further discussion and research on this important topic. My critique is divided into four comments.

First, Applehans assumes that the absence of hedonic feeding when good-tasting foods are readily available is due to the inhibition of reward-driven eating. This assumption seems to apply to everyone (e.g., he notes that it applies to both dieters and nondieters). It appears that the author views satiety and “inhibition of hedonic feeding” as similar phenomena mediated in part by the dorsal prefrontal cortex (DPFC). (Applehans does not distinguish between satiation and satiety, so in this commentary “satiety” will refer to the termination of eating when food is no longer desired.) Though he does not explicitly equate satiety and inhibition of hedonic eating, he also never distinguishes between them. We recently reviewed evidence suggesting that homeostatic eating motives (generated by physiological depletion and

terminated by energy repletion) and hedonic eating motives (generated in the absence of an energy deficit by the presence of highly palatable food and terminated by removing or consuming the food) overlap but are clearly dissociable at neurophysiological and behavioral levels (2). The fact that most of this work has been done with animals (3), and that there are few human neuroimaging studies examining this distinction, indicates that much more research is needed on the applicability of this distinction to humans.

Nonetheless, if one assumes that distinctions between these two appetitive motives documented in animals (2,3) are also applicable to humans, then it is crucial to clearly differentiate between them. A recent review (4) examined human research on whether hyperphagia stimulated by highly palatable foods represented a response to nutritional need or a need-free stimulation of appetite. The authors concluded that the results they reviewed

... not only confirm that satiety and orosensory stimulation have opposing effects on short-term food intake, but also suggest that palatability has a greater influence in conditions where satiety is enhanced... The implication is that palatability may lead to over-

consumption, particularly when sated (p. S8).

Thus the processes responsible for generating inhibitory signals associated with satiety (one form of “inhibited eating”) are likely to be quite different from the processes responsible for restricting hedonic eating (a second form of “inhibited eating”). An example of the author's conflating of these two different phenomena occurs on p. xx, where he says “hedonic feeding results from the dominance of appetitive motivation over inhibitory control, whereas dietary restraint results from the dominance of inhibitory control over appetitive motivation” (p. xx). If the term “satiety” replaced the term “dietary restraint” in the second half of this sentence, then I think it would make sense. Dietary restraint refers to volitional efforts to curb food intake; therefore it does not arise from inhibitory control, it *is* inhibitory control. The *origins* of dietary restraint are psychosocial, not neurophysiological; the *manifestation* of active dietary restraint may be evident in the DPFC or other brain structures, but not its origin. Stated differently, there is a major difference between not wanting to consume highly palatable foods (e.g., when sated) and wanting to consume them despite being sated but inhibiting the desire to do so.

This issue also arises when the author reviews research on the DPFC and the inhibition of eating (see p. xx). The DPFC is widely believed to mediate eating inhibition. The author reviews studies showing that successful dieters (who have maintained a major weight loss) show greater DPFC activation in response to a meal as well as studies showing that lean individuals show greater DPFC activation from before to after a meal than do obese individuals. These examples represent two very different types of “inhibition.” The formerly obese individuals continue

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to engage in very high levels of dietary restraint and physical activity to maintain their new, lower weight – thus their eating inhibition is controlled and conscious. Naturally lean individuals (who are typically nondieters), on the other hand, stop eating automatically when they feel full. Thus it is a stretch to view their maintenance of a normal weight as due to the “inhibition of hedonic eating.” Even though inhibition of eating (i.e., satiety) is occurring at some level, it is neither controlled nor conscious. In sum, for the concept of inhibition of hedonic feeding to make sense there needs to be a demonstrable need to inhibit hedonic eating in the first place.

A second issue is the author’s conflating of two related but distinct dimensions. The first dimension involves the distinction between homeostatic hunger and hedonic hunger (2,5). The second dimension involves the difference between dieting to achieve weight loss and dieting to avoid weight gain (5). Homeostatic hunger refers to the desire for food resulting from the depletion of readily available energy (e.g., glucose) below the level required for immediate energy needs. Hedonic hunger refers to the desire for highly palatable foods in the absence of homeostatic hunger, a desire that is typically elicited by the availability or presence of highly palatable foods.

As for dieting, we and others have recently made a distinction between controlling food intake to achieve weight loss and controlling food intake to avoid weight gain (5–7). There is a necessary connection between these two types of dieting and the two types of hunger described above. If someone is losing weight via dieting, then by definition they are frequently in a state of homeostatic hunger. If someone is dieting to avoid weight gain, it is assumed that their dietary restraint is mostly directed at avoiding consumption of highly palatable foods, a conflict that defines hedonic hunger (5). Although someone who is on a weight loss diet may desire to eat more food than the diet allows, and though some of those foods may be highly palatable, such hunger appears to be driven more by a need for calories than by a need for pleasure. Conversely,

when a person in energy balance has recently eaten to repletion, but experiences a strong desire for a donut when walking past a donut shop, such hunger is driven more by a desire for pleasure than by a need for calories.

However, Applehans conflates these two dimensions by referring to the need to inhibit hedonic feeding while dieting. One problem here is that he does not differentiate between the fact that *all* voluntary food intake is presumably rewarding but that the *nature* of the reward process may be very different – even qualitatively different – for individuals who are energy deprived vs. energy replete. Thus the implicit assumption that inhibiting food intake to sustain an energy deficit and inhibiting food intake to avoid an energy surfeit are interchangeable does not appear to be supportable.

A third issue arises in regard to the obesity epidemic and how much this epidemic is attributable to an obesogenic environment as opposed to individuals’ inability to sufficiently inhibit their intake. The author suggests (on p. xx) that obesity, like drug addiction, should be viewed in part as a “disorder of failed inhibition”; he goes on to say that “it is hoped that this review allows obesity researchers to integrate the notion of inhibition with the broader literature on hedonic feeding” (p. xx). A problem with this viewpoint is that it overestimates humans’ capacity for inhibiting hedonic eating in an obesogenic environment (8) and under-emphasizes the power of ever-present palatable foods to continuously stimulate appetite (5) regardless of short-term hunger status. As the author notes, an appetite system that capitalized on the occasional opportunity to overconsume energy during eons of human evolution presumably represented an adaptive advantage and therefore would have been strengthened over evolutionary time. What has changed dramatically in recent decades is not our appetitive or inhibitory systems, but the modern food environment. From this perspective overweight individuals represent the “cream of the evolutionary crop” because they capitalize on plentiful eating opportunities and tend to store extra energy as body fat; naturally thin people, on the

other hand, have a “disorder of failed satiety” because they fail to capitalize on opportunities to overconsume calories and store it as body fat. Applehans then notes that “the hedonic-inhibitory model suggests that the impact of inexpensive, readily available palatable food on overconsumption is not simply dependent on appetitive motivation,” and also “that transient disruptions of inhibitory control are more likely to result in overconsumption when food is highly accessible” (pp. xx). Though there are indeed many such “disruptions of inhibitory control” that occur every day (watching TV, feeling stressed, being cognitively absorbed in a task, etc.), putting the emphasis on the inhibition side of the equation appears to be misdirected because the problem is not the omnipresence of disinhibitors like TV and stress but the omnipresence of highly palatable foods that create hedonic eating drives and the need to resist them.

A fourth and final comment relates to the meaning of the terms “dieting” and “restrained eating”. Recent research has shown that restrained eaters identified by all of the most commonly used measures are not in negative energy balance and do not consume fewer calories in the natural environment than do unrestrained eaters (7,9,10). Throughout his paper, Applehans, like others before him (11), fail to differentiate between restrained eating and dieting to lose weight, despite substantial past evidence that these two constructs are associated with different and even opposite behavioral responses (12,13). As suggested above, actual weight loss dieting represents a challenge to the homeostatic rather than to the hedonic appetite system. That is, eating for reward presumably takes a back seat to eating for calories when the body is in a state of negative energy balance. Indeed, there is evidence that food cravings actually *decrease* during a very low calorie weight loss diet (14), which contradicts the author’s suggestion that “individuals do not crave (want) palatable food less intensely after starting a diet” (p. xx). The converse is true of hedonic eating – when energy replete, obtaining energy is presumably much less important than obtaining pleasure. Engaging in *either* type of eating

is presumably rewarding but the behavioral and neurophysiological mechanisms involved in inhibiting the two types may be quite different (2,5).

In sum, Applehans' has skillfully integrated studies of neurophysiology, evolution and eating control to advance our understanding of hedonically driven eating and its inhibition. My aim in this commentary has been to refine and extend his points to more fully characterize reward-based eating and its management.

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DISCLOSURE

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