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Neurobehavioral Inhibition of Reward-driven Feeding: Implications for Dieting and Obesity

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OVERVIEW

The development of effective weight loss interventions requires a thorough understanding of the motivational factors that drive and inhibit the overconsumption of food. Obesity was once believed to result purely from disruptions of homeostatic mechanisms controlling food intake. However, it is increasingly recognized that much of the excess caloric intake in obesity is driven by pleasure or the rewarding properties of readily available palatable food (1,2). It is important to note that the motivation underlying this reward-driven or "hedonic" feeding appears to have a different neurophysiological basis than homeostatic controls over energy balance, though the hedonic and homeostatic systems do interact (3). Recently, a surge of research has linked the appetitive motivation to consume palatable food to activation of the mesolimbic dopaminergic system, a neural pathway also implicated in drug addiction and addictive behaviors such as gambling (4). As a result of these findings, a view of obesity as a "disorder of appetitive motivation" has gained prominence among a growing number of obesity researchers. However, a model of hedonic feeding based purely on appetitive motivation has limitations, including an inability to explain instances of feeding behavior parsimoniously in both dieters and nondieters. The aim of this review is to summarize the strengths and weaknesses of the appetitive model, present the extant literature linking control of hedonic feeding to inhibitory processes localized in the prefrontal cortex (PFC), and delineate a theoretical model in which hedonic feeding is viewed as the product of an interaction between appetitive motivation and inhibitory control.

Appetitive motivation in hedonic feeding

The human feeding system leaves room for dessert. Humans will continue to consume palatable food even after energy requirements have been met and homeostatic satiety signals have been engaged. This consumption is clearly not driven by an acute need for calories, and hedonic factors (taste, pleasure, reward) play a large role in this type of feeding. Simply stated, the hedonic feeding model holds that palatable food consumption in the absence of an energy deficit (or the physiological signals associated with energy deficit) is driven by an appetitive motivational state referred to as "hedonic hunger" (2), and not by homeostatic controls over feeding. (For discussion of the role of reward in homeostatic feeding, which may be subjectively associated with escape from hunger rather than the pleasant tastes and textures of palatable food, see ref. (5).)

Evolutionary pressures for hedonic feeding

Hedonic feeding has an evolutionary basis. Modern humans preferentially overconsume sweet and high-fat palatable foods (6). Hominids subsisted as hunter-gatherers for the majority of evolutionary history and access to these types of foods (e.g., fruits, animal meat) was presumably limited. Hunting or prolonged foraging would often have been required to obtain sweet and high-fat foods, and it is likely that the capacity to find these foods pleasurable and highly rewarding motivated ancestral humans to engage in these food-seeking behaviors. After expending many calories to obtain them, consuming palatable foods in excess of one's current need for calories would have allowed some energy to be stored as fat, which could buffer against starvation in instances of short-term food shortage or famine. Thus, the ability of palatable food to promote overconsumption by overriding satiety signals was preserved in our species (3,7). In modern times, when palatable food is abundant and accessible, our evolutionarily derived capacity for hedonic feeding has contributed to an epidemic of overconsumption and obesity.

Neuroscience of hedonic feeding

Hedonic feeding is driven by both sensory and motivational processes that also play a role in drug addiction. The sensory pleasure resulting from tasting and ingesting palatable foods (and drugs) has been referred to as "liking." Liking is distinct from the motivational process of "wanting," which refers to the incentive value of a stimulus (8). Although humans generally want the foods they like, and like the foods that they want, liking and wanting have separate neurophysiological underpinnings

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and are dissociable under several circumstances. For example, drug-addicted individuals report a greater desire to consume a drug over time despite a gradual decrease in the degree to which they find consumption pleasurable, and selective lesioning of the neural system underlying wanting (described below) results in animals who show affective facial expressions of pleasure upon tasting palatable foods but are unmotivated to obtain them (8). Thus, although the sensory process of liking and the motivational process of wanting typically converge with respect to individual foods, instances of dissociation indicate separate neural underpinnings. Some have suggested that wanting plays a stronger role in obesity than liking given that obese individuals do not consistently appear to derive greater pleasure from tasting and ingesting palatable foods (1).

Liking of palatable foods is believed to be mediated by opioid neurotransmission in the nucleus accumbens (9), and pharmacological manipulation of this area results in dramatic changes in animals' intake of palatable foods and their affective facial expressions to the tasting of palatable food (10). In distinction, wanting has been consistently linked to activation of the mesolimbic system, which includes dopaminergic projections from the ventral tegmental area to the nucleus accumbens (8) (see special issue of *Psychopharmacology* (vol. 191, issue 3) for perspectives on the functions of mesolimbic dopamine). Ingestion of palatable food triggers dopamine release in the nucleus accumbens (11), and genetic or neurochemical manipulations of the mesolimbic system influence the motivation to consume palatable food without affecting its perceived pleasantness (liking) (12,13). Connections from the nucleus accumbens to hypothalamic feeding centers provide a pathway through which palatable food can influence feeding behavior by overriding satiety signals (3).

As several authors have articulated (especially refs. (9,14)), the mesolimbic system underlies the motivational drive to consume both palatable food and drugs of abuse, and it seems that drugs of abuse "hijack" the endogenous reward system that evolved to promote hedonic feeding and other motivated behavior. Additional findings also suggest overlap in the neural mediation of hedonic feeding and drug addiction. For example, animal studies have shown that repeated consumption of sugar can produce behavioral withdrawal effects similar to abstinence from addictive drugs (e.g., motor agitation, anxiety (15)). Second, cravings to consume both addictive drugs and palatable food increase with exposure to drug or palatable food cues (respectively), and the mechanism underlying both effects appears to involve cue-induced activation of the mesolimbic system (16,17). Finally, the reinforcing values of both palatable food and addictive drugs increase following psychological stress, which may involve sensitization of the mesolimbic system by stress-induced activation of the hypothalamic–pituitary–adrenocortical axis (19) (see ref. (18) for discussion of stress and reward-driven feeding). Together, the biobehavioral parallels between drug addiction and hedonic feeding have substantiated the view that hedonic feeding represents an "addictive behavior" driven by heightened wanting of palatable food.

Several lines of research suggest that both sensitization and desensitization of the appetitive system can explain individual differences in hedonic feeding. Questionnaire measures of reward sensitivity correlate positively with neural activation of striatal reward centers in response to exposure to palatable food cues (20) and have been used to quantify individual differences in appetitive motivation. Self-reported reward sensitivity is also positively associated with reported food craving and BMI (21). Individual differences in appetitive motivation can also be characterized at the neuronal or even the genetic level. Adiposity has been associated with reduced density of striatal dopamine D_2 receptors in obese individuals (22), suggesting neurophysiological hyposensitivity to reward. To compensate for this hyposensitivity, individuals with this phenotype are thought to overconsume palatable foods to achieve stronger stimulation of the reward circuit. Similarly, Epstein and colleagues (23) reported that individuals with the TaqI A1 allele of the dopamine D_2 receptor, which has previously been linked to deficient dopaminergic reward processing, obesity, and addiction (24), find snack foods more reinforcing than those without this polymorphism. Together, these findings indicate that individual differences in appetitive motivation evaluated at multiple levels of analysis explain engagement in hedonic feeding. However, as discussed below, the appetitive model is less useful for explaining instances in which hedonic feeding does not occur.

Inhibition in hedonic feeding

How do dieters regularly abstain from highly desired (wanted) palatable food, at least for finite periods of time? Can dieting be explained within the appetitive model? Given that one's favorite foods do not immediately become less desired on the first day of a diet, it seems that dietary restraint is the product of active inhibitory control of feeding rather than the passive result of reduced appetitive motivation. In fact, active inhibition of hedonic feeding is reflected not only in instances of successful dietary restraint, but also in the manner in which dietary control breaks down. Studies with chronic dieters ("restrained eaters") show that acute stressful and nonstressful challenges often result in increased food intake (25,26). The most parsimonious explanation is that inhibitory control of hedonic feeding is disrupted during challenges (through mechanisms discussed later), thereby disinhibiting the appetitive motivation to engage in hedonic feeding. Given the large number of individuals struggling to control their weight through dieting, it is important to develop an understanding of the neurobehavioral processes that mediate hedonic feeding inhibition.

The notion that inhibitory processes play an important role in feeding is not a novel idea, at least among the lay public. The view that overeating results from a lack of willpower or poor self-control is surprisingly prevalent, and has had the unfortunate consequence of promoting stigmatization of overweight and obese individuals. In addition to its scientific value, systematic investigation of the role of inhibitory control in hedonic feeding will hopefully redirect attention away from the "character" of overweight and obese individuals and

toward the biological and environmental factors that influence feeding behavior. Curiously, the scientific understanding of hedonic feeding inhibition is only just beginning to emerge. At this early stage, tentative answers to three key questions about hedonic feeding inhibition can be gleaned from the existing literature.

1. Which evolutionary pressures favored the capacity to inhibit hedonic feeding?
2. What nervous system structures mediate hedonic feeding inhibition?
3. How does hedonic feeding inhibition manifest in cognitive and affective processes?

Evolutionary pressures to inhibit feeding

As mentioned earlier, hedonic feeding likely evolved to promote the storage of energy as fat which would buffer against starvation during food shortages. The dramatic rise in obesity prevalence in modern times characterized by readily available palatable food is widely attributed to this evolutionary adaptation. However, the notion that hedonic feeding was generally adaptive for ancestral humans does not preclude the possibility that inhibiting hedonic feeding on a situational basis may also have been adaptive. In other words, the human feeding system may have evolved to follow the "standing rule" to increase fat stores through hedonic feeding when opportunities arose, but to make exceptions to this rule (i.e., inhibit hedonic feeding) in certain circumstances.

One can imagine several scenarios in which hedonic feeding inhibition would have been adaptive for ancestral humans. For example, the ability to inhibit hedonic feeding would have allowed one to conserve food during periods of anticipated food shortage (27), such as during prolonged expeditions in nonproductive areas or periodic (e.g., seasonal) reductions in food availability. In addition, hedonic feeding inhibition would have played a valuable role in the social context of ancestral human life. Ancestral humans commonly engaged in systematic food sharing among group members, which allowed ancestral humans to subsist on the more reliable food procurement success of the group rather than one's own efforts (and luck) (28). Given its importance to survival among ancestral humans, it is not surprising that food sharing had many social implications, such as fostering reciprocity among group members and forming the basis for social alliances (28). Within this context, the ability to inhibit hedonic feeding would have been critical. As the recipient of shared food, hedonic feeding inhibition would allow more food to be shared with one's immediate family, thereby contributing to inclusive fitness by supporting the caloric needs of one's current and future (via one's mate) offspring. As the procurer of food, inhibiting one's desire to consume the most prized (and typically most palatable) portions of a game animal could allow these portions to be exchanged for valuable social or tangible rewards. Finally, hedonic feeding inhibition would have allowed ancestral humans to follow social feeding norms. Hunter-gatherer groups typically ate together in a central location (28), and individuals who were witnessed consuming more than one's "fair share" would likely have been viewed as a burden to the group (27). Those perceived in this way could have suffered significant social costs which bore upon survival and reproduction. Thus, it would have been adaptive to inhibit careless bingeing on the fruits (or meats) of the group's labor.

Although it may be challenging for us to appreciate the impact of social norms on hedonic feeding in ancestral times, social influences on hedonic feeding are visible in modern humans. Regardless of whether hungry or satiated, individuals adjust their food intake to match those around them and consume less food when they perceive that their feeding is being monitored or evaluated (29). The fact that humans have the capacity to inhibit hedonic feeding according to social factors fits well with notion that many functions mediated by the PFC which promote behavioral inhibition and self-control were shaped primarily by social pressures that indirectly influenced inclusive fitness (30). It is argued that several functions of the PFC are critical to achieving hedonic feeding inhibition.

Neuroscience of hedonic feeding inhibition

The human PFC is considered the primary neuroanatomical basis for self-regulation, and is the region of brain that prevents us from automatically responding to our environment with unchecked emotionally driven behaviors by exerting top–down organizational and inhibitory control over automatic cognitive and affective processes (31). For example, regions of the PFC are implicated in planning and organization, delaying or inhibiting stimulus-driven behavioral actions, mental flexibility, self-monitoring, and regulating affective impulses (31,32).

Studies have revealed associations between PFC activation and general indices of feeding inhibition. The PFC, particularly the dorsolateral region, is activated following ingestion of a meal (33,34). The dorsolateral PFC has previously been linked to behavioral inhibition and matching behavior to environmental cues (32), and it is reasonable to speculate that it also plays a role in inhibiting reward-driven feeding in response to interoceptive satiety signals, but not necessarily in generating the satiety signals. Ideally, the involvement of the PFC in hedonic feeding inhibition (not just in general satiety) would be shown through neuroimaging studies linking patterns of PFC activation with motivation to consume palatable food following a caloric preload. Along these lines, Small *et al.* (35) examined neural activation patterns in response to repeated chocolate ingestion in "chocolate lovers." Although a standardized caloric preload was not included in their protocol, participants had recently eaten a meal (about 4 h earlier on average) and all denied acute hunger. Increasing activation of the lateral PFC was associated with decreasing motivation to consume additional pieces of chocolate over the course of the session, suggesting a role for lateral PFC activation in hedonic feeding inhibition.

The PFC's role in hedonic feeding inhibition should be reflected in success at dieting, which requires substantial inhibition of hedonic feeding. DelParigi *et al.* (36) examined patterns

of brain activation following food intake in formerly obese individuals (i.e., BMI > 35) who have achieved and maintained significant weight loss through dieting (i.e., current BMI < 25), and nondieting controls. Successful dieters showed greater activation of the dorsal PFC relative to nondieters following consumption of a liquid meal, and dorsal PFC activation was positively associated with self-reported dietary restraint in this sample. Others report that greater dorsolateral PFC activation following a meal is associated with reduced adiposity in men and women (37,38). Taken together, these findings suggest that dorsolateral regions of the PFC mediate hedonic feeding inhibition, thereby supporting successful dieting and weight maintenance.

Manifestations of hedonic feeding inhibition

A host of cognitive and affective processes are recruited by the brain to promote goal-directed behavior and reward acquisition, including subjectively experienced affect, reward salience, attentional focus, and behavioral activation. As a product of the organism's overall motivational state, these processes reflect the antagonism between PFC-mediated inhibition and appetitive motivation driven by the mesolimbic system. Two neurocognitive processes are presented which reflect antagonism between appetitive motivation and inhibition as it pertains to hedonic feeding or drug addiction (described below). Although there are important differences between addiction and hedonic feeding, the addiction literature provides a useful template for developing a model of hedonic feeding inhibition.

Decision making and delayed rewards. One PFC-mediated process that is likely critical to hedonic feeding inhibition is the ability to overcome the tendency to undervalue postponed rewards (called delay discounting). Humans generally prefer immediate rewards over delayed rewards, even when the delayed reward is of greater incentive value. For example, most of us would prefer to receive $10.00 today instead of $12.00 a year from now. It can be said that the brain "discounts" delayed rewards, or that rewards decrease in their ability to control behavior with increasing temporal delay (39). As the probability of actually obtaining a reward often decreases with a temporal delay in the real world, delay discounting may represent an adaptation selected to deal with the uncertainty inherent in delayed rewards (40). Despite the notion that delay discounting may have been evolutionarily adaptive, the tendency to discount delayed rewards too steeply is considered a facet of impulsivity and a risk factor for maladaptive behavior (39). Delay discounting can be quantified by presenting participants with a series of choices between two rewards differing in their objective reward value (one small, one large) and temporal availability (one immediate, one postponed), and determining the steepness of the hyperbolic curve characterizing their devaluation of rewards with increasing postponement. Using such tasks, researchers have linked greater discounting of delayed rewards with a number of appetitively driven behaviors, including various forms of substance abuse and gambling (41,42).

Another measure of impulsive decision making is the Iowa Gambling Task (43). In this task, individuals choose cards that result in monetary gains or losses from four decks. Cards in two decks sometimes yield large immediate monetary gains but produce long-term losses, whereas cards in the other two decks produce smaller immediate rewards but have the potential for long-term monetary gain. The task is conceptually similar to delay discounting tasks in that it taps into a decision-making process in which immediate rewards are favored over more advantageous eventual rewards. Impulsive decision making on the Iowa Gambling Task has been reported in those with bulimia nervosa (44) and individuals addicted to illicit drugs, alcohol, or gambling (4,45).

It is noteworthy that the brain regions implicated in choosing between immediate and delayed rewards are also implicated in hedonic feeding. The nucleus accumbens and interconnected cortical regions are selectively activated when human participants are presented with an opportunity to choose an immediate reward. In contrast, the lateral PFC is activated during all choice trials, including those between two delayed rewards, consistent with its role in general decision-making (46). Not only does the magnitude of nucleus accumbens and dorsolateral PFC activation in response to reward predict participants' preferences for immediate and delayed rewards, respectively (47), but also the greater relative activation of the lateral PFC compared to that of the nucleus accumbens and related cortical areas is observed when participants choose delayed over immediate rewards. These findings clearly support the notion that the mesolimbic system and PFC antagonistically guide our preference for immediate versus delayed rewards.

There is an intuitive link between hedonic feeding inhibition and impulsive decision making as measured by delay discounting and gambling tasks. To the degree that individuals are motivated to refrain from hedonic feeding to achieve a larger future benefit (e.g., weight loss), they are choosing between immediate and delayed rewards. Just as drug-addicted individuals must forego the immediate reward of a drug-induced "high" for the larger financial, social, and physical rewards of a sober lifestyle, dieters must forego the immediately rewarding experience of ingesting palatable food for the larger reward of weight loss and improved health. Thus, dieting can be considered a series of decisions to pursue a delayed reward of weight loss by sacrificing the immediate reward associated with hedonic feeding (48). It is reasonable to hypothesize that the high failure rate of weight loss diets partially stems from our tendency to discount delayed rewards. By extension, one might expect that those who discount delayed rewards more steeply are less likely to maintain long-term dietary weight loss.

Despite compelling theory and consistent empirical associations between impulsive decision making and multiple manifestations of "appetitive behavior," only a handful of studies have examined associations between performance on decision-making tasks and constructs related to hedonic feeding. Increased adiposity has been associated with poorer performance on the Iowa Gambling Task in two studies (49,50) and steeper delay discounting of monetary rewards (51). Such findings have led some to suggest that impulsive decision-making influences hedonic feeding (52), but studies

linking rigorously defined hedonic feeding at the behavioral level (e.g., food consumption following caloric preload, motivation to work for palatable relative to bland food) with performance on decision-making tasks are clearly warranted to validate this claim.

Selective attention. Attention allocation is another process which reflects antagonism between appetitive motivation and inhibition and is implicated in hedonic feeding. Attention allocation is influenced by one's motivational state (more precisely, attentional processing is co-ordinated with affective, physiological, and other processes that support motivated behavior), and humans selectively allocate attentional resources to motivationally salient stimuli like food (53) and conditioned drug cues (54) at the expense of other sensory information. It has even been suggested that a primary function of mesolimbic dopamine is to draw the brain's attention to motivationally salient stimuli (8,55). Selective attention promotes processing of sensory information needed to pursue goals or reward, and also serves to keep the organism "locked in" to appetitive pursuit by promoting continued processing of appetitive cues. In turn, continued appetitive cue processing results in continued activation of the extended mesolimbic system (17,55), sustained appetitive motivation, and further promotion of selective attention. This positive feedback loop (middle portion of **Figure 1**) maintains goal-directed behavior until the organism attains the goal or the loop is otherwise disrupted (e.g., by competing stimuli). Selective attention has been implicated in maintaining drug addiction and triggering relapse (54), and it is reasonable to speculate it similarly promotes hedonic feeding. The role of selective attention to food cues in feeding is suggested by its associations with homeostatically driven hunger and eating disorders (53,56,57). Yet, only one study has found that selective attention to food cues predicts subsequent consumption of palatable food (58). Replication and extension of these findings are warranted.

Fortunately, selective attention can be inhibited. Shifting attention away from motivationally salient stimuli is critical for planning, self-monitoring of behavior, and perceiving sensory information that might indicate the need to abort appetitive pursuit of a reward or goal. Attentional shifting breaks the feedback loop described above by disrupting continued cue-induced stimulation of the mesolimbic system, thereby allowing appetitive motivation to subside. For example, training participants to allocate attention away from alcohol cues on a dot-probe attention allocation task reduces alcohol cravings (59). Whether one selectively attends to a stimulus or flexibly shifts attentional resources among multiple stimuli is determined by an interaction between appetitive motivation and inhibition. The anterior cingulate cortex, a component of the medial PFC, appears to be the locus of this antagonism. The anterior cingulate cortex receives inputs from the mesolimbic system and other limbic areas (60) and has strong connections with the lateral PFC (61), thereby positioning it at the intersection of appetitive motivation and inhibitory control. Neuroimaging studies have shown increased anterior cingulate cortex activation following the presentation of motivationally significant stimuli (55) and during volitional efforts to control attention allocation (61,62). Collectively, preliminary evidence indicates antagonism of the mesolimbic system and PFC in the control of attentional focus, and by extension, the regulation of hedonic feeding.

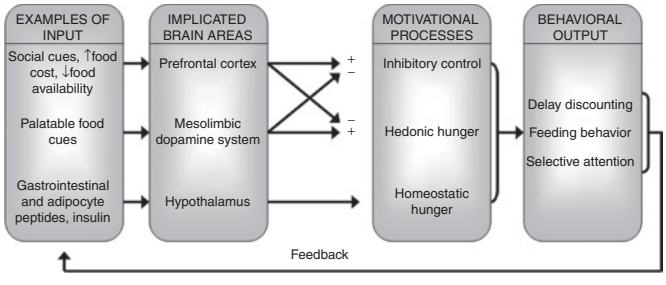

Figure 1 Simplified schematic representation of neural, motivational, and behavioral aspects of hedonic feeding and inhibitory control.

Synthesis—a hedonic-inhibitory model

Hedonic feeding has been conceptualized as an appetitive behavior based on its neuroanatomical and neurobehavioral parallels with drug addiction, but mounting evidence indicates that PFC-mediated inhibitory processes also play a critical role in hedonic feeding. This review argued that ancestral humans living as hunter-gatherers faced ecological and social pressures that called for occasional inhibition of hedonic feeding. This inhibition is represented at the neural level in dorsolateral areas of the PFC, at the motivational level in decision-making and attention allocation, and at the behavioral level in dietary restraint.

The evidence reviewed above is consistent with a hedonic-inhibitory model in which overconsumption of palatable food is a product of the antagonistic balance between appetitive motivation mediated by the mesolimbic system and active inhibitory control mediated by the dorsolateral PFC (**Figure 1**). Hedonic feeding results from dominance of appetitive motivation over inhibitory control, whereas dietary restraint results from dominance of inhibitory control over appetitive motivation. An implication of the model is that weight loss through dieting requires sustained inhibitory control over the appetitive system. This relatively straightforward prediction is consistent with the neuroimaging studies of successful dieters presented above (36) and, unlike the appetitive model, fits with the common observation that individuals do not crave (want) palatable food less intensely after starting a diet. Conversely, instances of dietary failure would be expected when the appetitive system dominates over the inhibitory system. It has been shown that stimulation of the appetitive system through exposure to the sights and smells of food is often enough to trigger an increase in feeding in dieters (63). Transient disruption of inhibition through exposure to stress and nonstressful cognitive load also leads to increased feeding (25,26). Although mechanisms through which these situational demands disrupt hedonic feeding inhibition are unclear (some studies suggest that stress-induced secretion of the glucocorticoid hormone cortisol may drive hedonic feeding following acute stress (18), but cortisol

would not be expected to drive hedonic feeding following nonstressful cognitive challenges), it has been suggested that such challenges monopolize attentional and working memory resources necessary to maintain inhibition over hedonic feeding (64). This notion is consistent with the fact that both inhibition and working memory are mediated by PFC (31,32), and that cognitive load leads to impulsive responding on the Iowa Gambling Task (65).

A potential criticism of the hedonic-inhibitory model is that inhibition might only manifest in active dieters, and that appetitive motivation alone is adequate to explain hedonic feeding and common obesity. There are at least two refutations to this argument. First, the fact that dieting is so common in modern societies warrants its incorporation into any comprehensive model of feeding. In a large national (United States) survey, >50% of obese men and women reported that they were currently trying to lose weight (66), and dietary restriction (i.e., "eating fewer calories," "eating less fat") was the most commonly cited strategy. A model which only explains feeding behavior in nondieters is therefore of limited use for the majority of obese individuals. Second, inhibition plays a significant role in hedonic feeding in "nondieters." Many individuals inhibit their intake of certain palatable foods for reasons unrelated to weight loss, such as health promotion (e.g., reduce cholesterol), social presentation (29), avoidance of food allergens, or to save food for later consumption (e.g., when guests arrive). Even if a firm distinction between active dieting and nondieting was tenable (rather than a continuum of dietary restriction), the notion of hedonic feeding inhibition extends well-beyond efforts at weight loss.

It is important to note that both between-person (trait) and within-person (state) variability in mesolimbic system and PFC function contribute to the balance between appetitive motivation and inhibitory control at any given moment. For example, genetic variants of the dopamine D_2 receptor (23,24) and cue-induced activation of the mesolimbic system (17) are known trait and state influences on appetitive motivation, respectively. Variants of the catechol-*O*-methyltransferase gene which codes for the enzyme that metabolizes brain dopamine and norepinephrine (67), neuroadaptations in the PFC with prolonged drug use (68), and increased situational demands on attentional and working memory resources (65) are probable trait and state influences on PFC-mediated functions. Importantly, neurobehavioral processes that reflect the antagonism of appetitive motivation and inhibitory control, such as impulsive decision making and selective attention, show both trait and state variation as well (39,53,65). Thus, a multitude of factors potentially contribute to imbalances between appetitive motivation and inhibitory control to determine an episode of hedonic feeding, and studies to further elucidate these factors are warranted.

Although hedonic feeding was originally conceptualized as an addictive behavior based on its shared neurobiology with drug addiction, several authors (e.g., (68,69)) have argued for a conceptualization of drug addiction as a disorder of failed inhibition. For unclear reasons, this "disinhibition model" has not yet gained as much attention as the appetitive model among addiction researchers, and very few obesity researchers (e.g., (52,70)) have argued for a corresponding conceptualization of feeding behavior. It is hoped that this review allows obesity researchers to integrate the notion of inhibition with the broader literature on hedonic feeding. For example, the hedonic-inhibitory model suggests that the impact of inexpensive, readily available palatable food on overconsumption is not simply dependent on appetitive motivation, but also that transient disruptions of inhibitory control are more likely to result in overconsumption when food is highly accessible. Finally, the hedonic-inhibitory model may serve as a neurobehavioral framework within which behavioral weight loss interventions that focus on restraint and "self-control" can be recast, and as an impetus to develop pharmacological agents that increase inhibitory control over hedonic feeding.

Caveats and future directions

This review has attempted to answer three basic questions about hedonic feeding inhibition. Many additional questions remain, such as: which genetic and environmental factors influence the balance between appetitive motivation and inhibitory control? Does hedonic feeding inhibition prospectively predict adiposity? Is inhibitory control over hedonic feeding reflected in questionnaire measures of dietary restraint? Can hedonic feeding inhibition be enhanced through behavioral or pharmacological interventions?

In addition to answering new questions, methodological issues related to the study of hedonic feeding inhibition must be addressed. Many studies cited in support of PFC-mediated hedonic feeding inhibition measured constructs closely related to hedonic feeding (such as adiposity, successful dieting, or other appetitive behaviors) rather than rigorously defined hedonic feeding behavior in a laboratory or naturalistic setting. Although these studies have been incredibly important in building a case for a hedonic-inhibitory model of feeding, future research should seek to isolate hedonic feeding methodologically from that driven by homeostatic energy requirements by measuring food consumption after a caloric preload, as suggested by Lowe and Butryn (2). Such studies are critical as hedonic and homeostatically driven feeding have distinct neural representations and likely play very different roles in the etiology of obesity and successful dieting. Additional research linking indices of inhibitory control with hedonic feeding through behavioral, genetic, and neuroimaging methodologies would be invaluable in expanding and refining the hedonic-inhibitory model.

It has not escaped attention that inhibitory processes are typically invoked to achieve certain goals (such as weight loss), and that these goals likely invoke appetitive motivation. Although inhibitory processes (e.g., selecting delayed over immediate rewards, shifting attention away from food cues) are neuroanatomically and functionally distinct from the motivation to inhibit feeding, the process by which the brain recruits inhibition to support acquisition of delayed rewards such as weight loss requires additional study.

CONCLUSIONS

A model in which mesolimbic dopamine underlies an appetitive motivational state that substantially contributes to overfeeding and obesity has been met with enthusiasm from some obesity researchers. However, this appetitive model does not offer a conceptualization of abstinence from hedonic feeding, which is necessary to understand both dietary restraint and the daily feeding behavior of nondieters as well. A more complete model of feeding incorporates both appetitive motivation and inhibitory processes which are mediated by the PFC and most likely evolved under social and ecological pressures. It is hoped that this hedonic-inhibitory model of feeding will serve as a useful framework to guide future research on the etiology and treatment of obesity.

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