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Time to abandon the notion of personal choice in dietary counseling for obesity?

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Current approaches to dietary counseling for obesity are heavily rooted in the notion of “personal choice” Typically, patients receive education about dietary contributions to energy balance and are then encouraged to make dietary choices (e.g., food selections, portion sizes) consistent with weight loss. Yet even highly motivated and nutritionally-informed patients often struggle to refrain from highly palatable, energy-dense foods available in the modern environment, and ultimately, only a small percentage of individuals achieve sustained weight loss through dietary modification (1–4). Failed attempts at weight control are frustrating to patients and providers alike. Studies show that both parties frequently attribute obesity to poor “personal choices” or insufficient “willpower” on the part of the patient (5,6). For example, a sample of British dietitians ranked “lack of willpower” as more important to the development of obesity than genetic factors (7), even though adult body mass is 55–75% heritable (8,9). The suggestion that individuals become or remain obese due to their unhealthy personal choices, or a lack of willpower to make healthy choices, is stigmatizing to patients and unlikely to motivate patients to lose weight (10). De-emphasizing the role of personal choice in dietary counseling for obesity would reduce stigma, but doing so carries the risk of undermining patients’ perceived control over their

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weight loss success. The goal of this commentary is to help dietitians negotiate this dilemma by presenting a scientifically-informed framework that views the personal choices relevant to obesity counseling in terms of three neurobehavioral processes. We argue that applying this framework in dietary counseling can both minimize patient stigma and preserve patients' sense of empowerment.

Personal choice: a problematic framework for obesity

The term “personal choice” implies that human behavior derives from conscious, volitional decisions, and connotes that humans have “free will” to decide between alternative courses of action - independent of biological and environmental forces. An implication of this definition of “personal choice” is that individuals can be considered causally, financially, and morally responsible for their behavior (11,12), a notion firmly embedded in the folk psychology of many cultures (13–15). The ethical and policy implications of personal choice in health have been discussed at length (11,16,17). In contrast to the notion of personal choice, some argue that human behavior is explained by neurobiological processes and their interaction with environmental stimuli (18). Supporting this deterministic¹ model of “personal choice” are studies demonstrating that 1) future actions can be predicted by brain activation patterns up to 10 seconds before individuals become aware of having made a decision (19), 2) behavior is strongly influenced by processes outside of conscious awareness (20), and 3) individuals can be led to believe that they have caused actions outside of their control (21–24). Others dismiss the apparent conflict between free will and neurobiologically-based explanations of behavior (25–27). Whether human behavior is ultimately rooted in free will, neurobiology, or a combination of both will not be settled anytime soon. Yet, there is still considerable value for understanding how neurobehavioral processes interact with the environment to influence eating behavior for the purposes of understanding obesity's etiology and reducing stigma.

As dietitians cannot control patients' environments or their genetic vulnerabilities, and effective non-surgical weight loss treatments do not currently exist, it is understandable why many seek to instill responsibility for change in their patients. However, rather than engendering a sense of empowerment, the suggestion that sufficiently motivated patients can *choose* to engage in a healthier lifestyle can often lead to guilt and stigmatization by implying that individuals are responsible for failing to control their weight. Indeed, obese patients often feel stigmatized in healthcare settings, which can result in avoidance of the health care system, increased eating pathology, and even weight gain (10,28). Adopting a scientifically informed framework that clarifies how personal choice is affected by biological and environmental factors may reduce obesity stigma in healthcare settings (29) and empower patients by drawing attention to the environmental drivers of obesity that are within their control.

A scientific framework of personal choice in obesity

Building on emerging research, we propose that “personal choice” in obesity can be understood as a composite of neurobehavioral processes influenced by biological and environmental forces. Though a number of existing neurobiological models are potentially relevant to understanding personal choice in obesity [e.g., (20,30–32)], we focus on three neurobehavioral processes that have been most consistently implicated in obesity and overeating: food reward, inhibitory control, and time discounting.

¹Determinism is the view that behavior is caused by previous mechanistic processes.

Food reward

Obesity has been viewed almost exclusively as a disorder of energy homeostasis in which overeating results from insufficient satiety signaling or amplified hunger signaling (33,34). However, research conducted over the past decade has demonstrated that the sensory experience of palatable food can easily override homeostatic controls of energy balance, leading to overeating in the absence of true physiological hunger (35,36). It is also appreciated that the palatability of our food supply has been greatly enhanced by the food industry through the infusion of increasing amounts of sugar, fat, salt, and flavorings. This food engineering has been implicated as a key contributor to the obesity epidemic (37). Food reward includes both the *experience of pleasure* one receives from eating and the *motivational drive* to obtain and consume palatable food (38). Of these two aspects of reward, the motivational component may be more relevant to obesity since obese individuals do not report experiencing greater pleasure from palatable food than normal weight individuals (39,40). Of particular relevance to personal choice in obesity are findings that one's sensitivity to food reward is grounded in genetics and neurobiology and is strongly linked to obesity (41).

The neural processing of food reward has been traced to the mesolimbic system (Figure 1), the brain's "reward circuit" which also mediates the motivation to engage in sex, gambling, and substance use (42). The hedonic pleasure associated with eating is linked to opioid neurotransmission in several small "hotspots" in the nucleus accumbens and other regions, whereas the motivational aspect of food reward is primarily mediated by dopamine pathways from the ventral tegmental area to the nucleus accumbens (38,42–46). Interestingly, *blunted* mesolimbic system neurotransmission (47), and biologic and genetic markers associated with *diminished* dopamine signaling (48–50) are linked to higher adiposity. The prevailing hypothesis is that this blunted mesolimbic signaling represents a deficiency in neural reward processing for which affected individuals compensate by overconsuming palatable food. In this way, deficient neural reward processing appears to equate with *greater* reward sensitivity at the level of behavior.

Greater sensitivity to reward is linked to stronger food cravings (51), preferences for sweet and fatty foods (52), greater *ad libitum* food intake in laboratory studies (53), and higher body weight among adults and children (40,52,54,55). Sensitivity to reward is hypothesized to explain vulnerability to aspects of the "toxic food environment." For example, living in areas with greater access to fast food outlets has been linked, albeit inconsistently, to an obesity-promoting diet and higher body mass (56,57), but these effects appear to be strongest among those most sensitive to reward (58). Essentially, high reward sensitivity combined with convenient access to highly palatable, energy-dense foods represents a biology-by-environment interaction that makes one extremely vulnerable to overeating and weight gain.

Inhibitory control

Food reward accounts for the "pull" towards palatable food that can drive overeating even in the absence of true physiological hunger. Beyond the intensity of food cravings, the question remains whether we can ignore or suppress such urges. After all, isn't the ability to *override* our hedonic motivations the essence of "choice," "self-control," and "willpower"? The fact that we can refrain from eating palatable food (if only occasionally) while still finding food extremely tempting indicates that the capacity to refrain from eating is a distinct process from food reward. In other words, inhibiting our food intake is not simply a matter of *reducing* the motivation to eat; it involves actively controlling behavior *despite* a strong motivation to eat. Though exercising inhibitory control over eating has long been considered

the central task in weight management, a scientific description of inhibitory control of eating at the behavioral and neurobiological levels is only now emerging.

We (59) and others (60) have proposed that inhibitory control over eating is supported by executive functions mediated by the prefrontal cortex (PFC). The PFC is considered critical for self-control, planning, and goal-directed behavior more generally (61,62), and the inhibition of eating can be considered a special class of behavior under its governance. There is ample evidence linking the functioning of prefrontal regions to performance on tasks measuring inhibitory control (31) and clinical syndromes characterized by impulsivity such as attention-deficit/hyperactivity disorder (ADHD) and drug addiction (63). In particular, the dorsolateral region of the PFC (Figure 1) has been implicated in the “decision” to engage inhibitory processes for the purpose of self-regulation (63–65). Several recent neuroimaging studies link differences in dorsolateral PFC function with the ability to inhibit eating. Hare et al (64) asked dieters to choose between pairs of 50 food items varying in taste and perceived healthiness. Dieters who consistently selected health over taste showed greater dorsolateral PFC activation when choosing the healthier options compared to those who more often selected taste over health. Further, there was evidence of functional connectivity between the dorsolateral PFC and brain areas associated with reward processing, consistent with the notion that the dorsolateral PFC inhibits the influence of reward on behavior. Other studies have shown that the dorsolateral PFC is activated following the ingestion of food (66,67), and that greater postmeal activation is associated with reduced adiposity (68,69), decreased food craving (70), and successful weight loss (71). Taken together, these findings indicate that the dorsolateral PFC supports active suppression of the motivation to eat palatable food. Unfortunately, life stress and other factors can easily disrupt inhibitory control (72) and lead to weight gain (73). Given this, it is not surprising that weight loss interventions, which largely rely upon persistent inhibitory control of eating, have meager long-term success rates (1–4).

Time discounting

A third factor that likely contributes to the low success rates of dietary interventions for obesity is the human tendency to devalue delayed rewards. Most of us would prefer to receive \$200 today rather than \$300 a year from now. This decision illustrates the fact that the brain discounts the value of money and other rewards over time, resulting in impulsive, short-sighted decision-making. Time discounting provides a framework for understanding why we sometimes knowingly make choices that are not in our best long-term interest [i.e., why the ‘will’ breaks down (74)]. For some individuals and not others, the immediate rewards of smoking, gambling, and drug use have a more potent influence on decision-making than the long-term social, financial, and physical costs of such behavior. Numerous studies have found that individuals who engage in these “addictive behaviors” assign disproportionate weight to the immediate pleasure derived from these activities compared to those who abstain (75–79). The link between time discounting and body weight is reflected neuroanatomically, with time discounting being governed by the same brain regions associated with food reward and inhibitory control. Time discounting is influenced by an impulsive, appetitive system that promotes pursuit of immediate rewards, as well as a reflective, deliberative system that seeks to maximize long-term gain. Neuroimaging studies indicate that these neural systems are composed of the mesolimbic dopamine system and its extensions, and the dorsolateral PFC, respectively (80–82). In fact, the reciprocal activation of these two brain regions predicts performance on time discounting tasks (83). The relevance of time discounting to obesity is substantial. In a very literal sense, weight loss requires consistent selection of delayed rewards [e.g., health benefits of weight loss (84)] over the immediate rewards associated with palatable food (85). In other words, the process of weight loss is directly at odds with the human tendency for time discounting. Consistent

with this notion, several studies have linked higher body weight (86–88) and intake of palatable food (89) to greater time discounting on behavioral choice tasks.

Summary and Implications for Counseling

Thus far we have highlighted three neurobehavioral processes that promote overeating and obesity: 1) neurobiologically-based behavioral sensitivity to the rewarding properties of food, mediated by the mesolimbic dopamine system, 2) relative weakness in inhibitory control, mediated by the PFC (particularly dorsolateral regions), and 3) steeper discounting of the delayed rewards of weight loss relative to the immediate pleasure associated with eating, reflecting the interaction between the mesolimbic system and the PFC. There are at least three implications of this neurobehavioral model for dietary counseling for obesity.

First, the model explains eating behaviors which promote obesity without invoking character flaws (e.g., lack of willpower). By emphasizing genetically-influenced neurobiological processes that confer vulnerability to overeating in a toxic food environment, the model enables dietitians to more effectively address obesity (as discussed below) without promoting stigma.

Second, the neurobehavioral model preserves a sense of individual control. Though it may seem counter-intuitive, shifting the focus away from “personal choice” and towards the environmental and neurobehavioral processes involved in eating can encourage patients to take an active stance in their approach to weight management. We recommend that dietitians simultaneously convey two messages about weight control to their patients: 1) obesity is heavily influenced by genetic and environmental factors, and an epidemic of obesity is precisely what would be expected given the genetic heritage of our species and the omnipresence of palatable food in the environment; and 2) successful weight management can be achieved by taking active steps (such as those described below) to minimize the impact of the environment on eating behavior. The first message acknowledges that patients are working against potent genetic vulnerabilities and a toxic food environment, and normalizes patients’ (and dietitians’) frustration with failed attempts at weight control. The second message signals that patients can better control their weight through strategies focused on the interaction between the brain and the environment. For the majority of dietitians, this second message constitutes a shift in strategy from urging patients to make the “tough choices” required for weight control to helping patients minimize the number of tough choices they encounter. This differs from the traditional approach to obesity counseling, which by simply encouraging patients to eat fewer calories than they expend, ignores the very processes that make this advice so difficult to follow.

Finally, the framework presented above supports an increased emphasis on several behavioral strategies that have been considered adjuncts to dietary counseling for many years (90) (Table 1). Dietitians should assist patients in manipulating their environments to minimize exposure to palatable food cues, a step that is essential to reducing energy intake by preventing activation of the brain’s reward circuitry that generates the motivation to eat. For example, patients should remove tempting, high-calorie foods from their home and workplace. Of course, the decision to bring high-calorie foods into the home is made at the food store, and shopping from a grocery list or using online grocers (e.g., Peapod) can help reduce one’s susceptibility to the torrent of food cues at the supermarket (91). Another strategy involves learning to minimize exposure to stress and developing more effective stress reduction strategies, as stress promotes overeating and obesity by enhancing food reward processing (92,93) and disrupting inhibitory control (94,95). Dietitians may briefly review stress management techniques, encourage exercise as a stress reduction strategy, and refer patients to appropriate behavioral specialists. Finally, consideration of time discounting

would suggest that increasing the delay to food rewards and decreasing the delay to weight loss rewards promotes better adherence to dietary goals. Consistent with this idea, patients should be encouraged to prepare healthy foods in advance to make them immediately accessible, keep tempting snacks out of the home (thus requiring a trip to the food store to obtain them), and focus on achieving short-term behavioral weight control goals (e.g., meeting a daily calorie goal) rather than focusing exclusively on long-term weight loss. The focus on short-term behavioral goals may be especially important considering that the rate of initial short-term weight loss is predictive of long-term weight loss outcomes (96), and that unrealistic long-term weight loss expectations are sometimes associated with poorer outcomes and higher attrition from weight loss treatment (97; also see 98). Focusing on achieving short-term behavioral goals would likely have the dual benefits of promoting early weight loss through behavior change and de-emphasizing any unrealistic weight loss expectations patients may have.

As the neurobehavioral basis of eating behavior advances, so will our understanding of obesity and weight control. However, enough progress has been made to enable dietitians to shift from a model of obesity counseling grounded in personal choice to one rooted in the brain processes that govern eating behavior in an obesity-promoting environment. In addition to providing nutrition education and encouragement, dietitians should more heavily focus on helping patients overcome the brain-based processes that make dietary modification so difficult, largely through strategies that have been considered “behavioral adjuncts” to dietary obesity counseling for many years. Dietary lapses or failures should be conceptualized as the result of brain systems interacting with a toxic food environment, and not as a reflection of poor personal choices or lack of willpower. Even if this approach is no more effective in producing weight loss than current practices, it is much less likely to elicit patient stigmatization.

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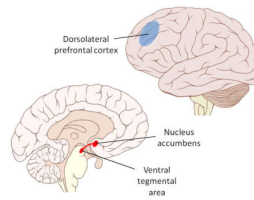


Figure 1.

Brain regions implicated in eating behavior. Food reward is largely mediated by the mesolimbic reward pathway (red), whereas inhibitory control of eating involves functions governed by the dorsolateral prefrontal cortex (blue). Delay discounting appears to be influenced by the functional interaction(s) between these two regions. Images modified from original productions by Patrick J. Lynch and C. Carl Jaffe, obtained under creative commons license.

Table 1

Summary of a neurobehavioral model of personal choice in obesity

Behavioral process	Neural basis	Impact on personal choice	Clinical implication(s)
Food reward	Mesolimbic dopamine system	Increases motivation to consume palatable food Mechanism by which the highly engineered food supply overrides homeostatic controls of energy balance	Removing palatable food cues from personal environments (e.g., home, workplace) reduces overeating by preventing activation of reward circuitry Limit the impact of reward on food choice by shopping with a grocery list, using online grocers, planning restaurant menu selections in advance
Inhibitory control	Prefrontal cortex, especially dorsolateral regions	Supports restraint from eating, which is a core component of weight management Inhibitory control can be disrupted by stress and demanding mental tasks, leading to overeating	Avoid situations (e.g., buffets, restaurants) that challenge inhibitory control Counsel or refer for stress management Keep high-calorie foods out of reach where stress is anticipated
Time discounting	Interaction between mesolimbic system and prefrontal cortex	Immediate pleasure from eating has a greater impact eating has a greater impact on decision-making than delayed benefits of weight control	Focus on achievement of short-term goals (e.g., meeting a daily calorie goal) Advise patients to prepare healthy foods in advance to increase their accessibility relative to unhealthy convenience foods