Perspective

Eating Motives and the Controversy over Dieting: Eating Less Than Needed versus Less Than Wanted

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Abstract

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Anti-dieting sentiment has grown in recent years. Critics of restrained eating suggest that it evokes counter-regulatory responses that render it ineffective or even iatrogenic. However, restrained eaters are not in negative energy balance and overweight individuals show reduced eating problems when losing weight by dieting. A distinction is often drawn between physiological and psychological hunger, and neuroscience research has shown that there is a neurophysiological reality underlying this distinction. The brain has a homeostatic system (activated by energy deficits) and a hedonic system (activated by the presence of palatable food). The omnipresence of highly palatable food in the environment may chronically activate the hedonic appetite system, producing a need to actively restrain eating not just to lose weight but to avoid gaining it. Just as restricting energy intake below homeostatic needs produces physiological deprivation, restricting intake of palatable foods may produce "perceived deprivation" despite a state of energy balance. In summary, the motivation to eat more than one needs appears to be every bit as real, and perhaps every bit as powerful, as the motivation to eat when energy deprived.

Key words: restrained eating, dietary restraint, appetite, hunger, palatability

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Introduction

Paradoxically, the prevalence of both eating disorders and obesity has been increasing in developed countries during the past few decades. Even more ironic is the fact that dieting has been viewed as contributing to both problems. These apparent paradoxes can be reconciled if one takes into account that the brain has overlapping but nonetheless distinct systems that underlie appetitive motivation. The purpose of this paper is to use current behavioral and neuroscience research related to hunger- and reward-induced feeding to help understand and resolve the controversy that has arisen over the wisdom of dieting (1).

The Controversy over Dieting

Dieting has been criticized on a variety of grounds. It has been viewed as: being a major contributor to the development of binge eating (2) and eating disorders (3); producing an exaggerated reduction in metabolic rate when weight is lost, making weight regain more likely (4); producing cumulative adverse effects on physiological (5) and psychological (3,6) functioning as weight is lost and regained repeatedly; producing a variety of untoward emotional reactions in obese individuals who lose weight (7,8); being ineffective because most people eventually regain their lost weight (7,9); being psychologically unhealthy for women because it promotes unrealistic expectations about the malleability of body weight and shape (10,11); producing a vulnerability to emotional eating and problems with eating regulation in restrained eaters (12); and interfering with the efficient processing of information (13).

These criticisms of dieting have contributed to the development of a widespread anti-dieting movement among laypeople and some professionals (1,7,14–16). As Brownell and Rodin (1) concluded in an article on the controversy over dieting, "we are witnessing a rapid and forceful swing of a pendulum that is moving from an entirely pro-dieting mentality to an anti-dieting fervor" (p. 786).

The basis of the anti-dieting position differs somewhat when applied to people in the normal-weight and over-

weight range. For normal-weight individuals, dieting is viewed as a major source of the rising prevalence of bona fide eating disorders and the spread of body dissatisfaction, binge eating, and extreme weight control practices among otherwise healthy normal-weight people (and young women, in particular). For overweight or obese individuals, dieting is viewed as ineffective in the long run, as generally incapable of overcoming biologically based determinants of body size, as lacking justification because the health risks of mild to moderate obesity are minor or nonexistent, and as generally creating more problems than it solves (7,15).

Brownell and Rodin (1) pointed out a major problem with the anti-dieting position: "It may be important to separate dieting in individuals who are close to normal weight from dieting in those who are heavier. Valid arguments that dieting can be pathological in the former group have been used to discourage treatment for the latter group" (p. 787).

Because critics of dieting by normal-weight individuals assume that dieting is motivated by a drive for thinness (3), relinquishing dieting would presumably have little or no effect on body weight. However, among overweight individuals or normal-weight individuals who are prone to weight gain, the consequences of relinquishing conscious attempts to lose weight or avoid weight gain in an environment that promotes weight gain (17,18) are likely to be quite different. Indeed, when the prevalence of eating disorders is compared with the prevalence of obesity, then from a public health perspective, the most significant diet-related problem may not be that people who should not be restricting their food intake are doing so but that those who need to restrict their intake are not doing so.

The Relevance of Appetitive Motivation for the Dieting Controversy

Most commercial and professional approaches to obesity draw a distinction between physical hunger (resulting from short-term energy depletion) and terms such as psychological hunger, appetite, or eating when not hungry (19). We will refer to this dual-factor perspective as the standard model of hunger. There is a widespread assumption among many researchers (20) and laypeople (16) that the second type of hunger is motivated psychologically; for example, to avoid or soothe painful emotions. Similarly, only physical hunger is viewed as real (i.e., as reflecting a biological need). Psychological hunger, in contrast, is presumably motivated by a desire to avoid or escape from unpleasant emotions and, therefore, is not real because it is being driven by emotional, rather than by energetic, needs. [Although it is conceivable that emotional distress could also be homeostatic in nature (e.g., if a person engages in increased emotional eating after losing weight by dieting), available evidence suggests that weight loss reduces, rather than increases, disinhibitory eating (70,71).] If this viewpoint is accurate, then dieting to lose weight will not only fail to resolve the psychological or emotional eating problem, it will likely create new problems because of the general difficulty of maintaining a weight loss and the specific possibility that dieting may make people even more susceptible to emotional eating (21,22).

However, there is another way of conceptualizing the nature of human appetitive motivation. We will refer to this as the homeostatic-hedonic model of hunger. This alternative approach also recognizes two different motivations for food intake but differs from the standard model in two major ways. First, although the appetitive motivation created by the existence of a short-term energy deficiency is obviously a key homeostatic feature of human eating and weight regulation, in most developed countries appetite is entrained to a schedule of eating that is based more on cultural custom than energy depletion (23). Such eating schedules (e.g., intermeal intervals of several hours or less) usually anticipate and prevent the development of significant metabolic changes (23). According to this view, the physiological alterations that precede meal taking reflect the body's anticipation of a sudden infusion of nutrients (i.e., cephalic phase responses), rather than the effects of acute energy depletion. Thus, even the hunger experienced before meals is not necessarily real hunger in the sense of signaling a clear state of energy deprivation.

The second—and for present purposes more important—distinction between the standard and the homeostatic-hedonic models of hunger is the nature and source of eating when not hungry (24,25). It is well known that people will often eat simply because food is there (26), even if they have recently eaten a meal (24). Indeed, given that few people deprive themselves of energy long enough to experience physiological hunger, it appears that most food consumption is motivated by something beyond the need to counteract physiological signals of energy depletion. Overweight people who eat when not hungry are doing so not only despite the absence of a short-term physiological need but also despite the existence of an overweight condition that involves the storage of many thousands of calories. However, although such eating appears to be irrational and self-destructive, if one takes into consideration the vast differences between the evolutionary environments that shaped humans' appetitive system and the modern environment, then the meaning of eating when not hungry takes on a very different meaning.

According to an evolutionary account, it would be adaptive not only to respond to declining energy stores by seeking sources of food but also to seek out and consume food in the absence of such physiological hunger because doing so would prevent the development of physiological hunger (and the risks it entails) and enhance the consumption and storage of extra energy as body fat (thereby protecting people from periods of food scarcity) (27,28). These

ideas have led to the hypothesis that food consumption can be motivated by brain systems that are activated by energy depletion (a homeostatic or need-based motive) and by the mere availability or presence of food, especially highly palatable food (a hedonic- or want-based motive).

Although it is possible to differentiate between homeostatic and hedonic eating motives, it is important to note that the boundary between them is fuzzy rather than distinct. It is clear that a generally well nourished person who goes 2 to 3 hours without eating is not in a state of energy deprivation but that the same person who goes 24 hours without eating is. However, the point at which lack of energy intake represents a homeostatic challenge is not clear-cut and may vary by individual and level of energy expenditure. As for hedonically based eating, the exact means by which the widespread availability of palatable foods increases energy intake is unclear. A positive energy balance created by the consumption of such foods could be mediated by the frequency of eating, by the amount consumed during individual bouts of eating, and/or by the energy density of the foods consumed.

Because the existence and functioning of the homeostatic and hedonic motivations for eating have been most thoroughly documented in animals, we now turn to animal literature to provide an overview of evidence for the existence of these two brain-based motivational systems underlying appetite.

Summary of Animal Evidence of Dual Brain-Based Appetitive Motivations

There are a variety of neuroactive substances that impact feeding behavior, including biogenic amines, amino acids, cannabinoids, fatty acids, and neuropeptides. It is now clear that neuroregulators involved in feeding behavior do not act alone in isolated brain areas but rather act through a distributed neural network (29). Also, they do not affect one aspect of feeding but impact feeding associated with energy needs as well as reward (30,31). Although it is clear that animals and humans eat for many reasons, the model we are proposing deals with feeding induced by two of the most important: energy need (hunger) and palatability (reward).

A good deal of data supports the dissociability of these two eating motives. For example, the powerful orexigenic agent neuropeptide Y (NPY)[1] induces a state resembling hunger, inducing feeding of bland laboratory chow in fully satiated rats (32,33). Food deprivation or food restriction induces increased gene expression of NPY in the arcuate nucleus of the hypothalamus, an area containing many NPY cell bodies (34). Furthermore, rats injected intracerebroventricularly with NPY will press a lever to obtain food in an operant chamber at a level observed in rats deprived of food for 24 to 48 hours (35). Thus, NPY appears to be a regulator whose function is related to energy-driven feeding.

In contrast, opioid peptides seem to be involved in the rewarding aspects of eating. Blockade of opioid receptors results in a decrease in consummatory behavior, particularly of preferred, palatable foods. Doses as low as 0.01 mg/kg of the opioid antagonist naloxone will decrease intake of preferred diets in food-deprived (24 hours) rats (36). However, doses as high as 3 mg/kg naloxone have no effect on intake of a nonpreferred diet. In food-restricted (85% body weight) rats, naloxone decreases intake of a highly palatable sucrose-based rat diet, whereas this opioid antagonist does not decrease intake of the less preferred cornstarch-based diet (37). Unlike the elevation in NPY gene expression observed after food restriction or deprivation, opioid gene expression decreases with caloric restriction or deprivation (38). On the other hand, ingestion of a high-fat/sucrose diet increases gene expression of the opioid peptide dynorphin (39). This appears to be due to the hyperphagia associated with the palatable diet.

Of interest, we noted that ingestion of the same number of kilocalories of the fat/sucrose diet as the cornstarch diet resulted in a decrease in opioid gene expression among rats consuming the former diet (39). This might be due to a reward deprivation; that is, the rats ingesting the palatable diet were pair-fed to the number of kilocalories eaten by rats given the cornstarch diet. Thus, yoking the energy intake of a palatable diet to the intake of a bland diet results in a change in opioid circuitry that resembles energy deprivation. If given the opportunity, the rats in the high palatability condition would have eaten much more of the sucrose/fat diet. In a parallel situation in humans, 200 kcal of an unflavored hot cereal might result in short-term satiation, whereas 200 kcal of chocolate cake might not satisfy one's hedonic needs. This phenomenon may occur due to palatability-induced changes in opioid circuitry and other reward-related neurochemicals.

To test this potential dichotomy between NPY and opioids, we allowed rats to choose between laboratory chow and a 10% sucrose solution after injection of either NPY or the μ-opioid agonist [D-Ala^2,*N*-Me-Phe^4,Gly^5-ol]-enkephalin (DAMGO) into the paraventricular nucleus of the hypothalamus (40). After NPY injection, rats ingested ~48% of their energy from the sucrose solution and 52% from the laboratory chow. On the other hand, DAMGO-injected rats ingested only ~12% of their energy from the chow and 88% from the sucrose solution. Thus, DAMGO had a much more potent effect on intake of the palatable solution compared with NPY.

The above data support a two-component model. However, further experimentation has led to a more complex model. Glass et al. (41) examined the effects of naltrexone injected into the paraventricular nucleus (PVN) or central

[1] Nonstandard abbreviations: NPY, neuropeptide Y; DAMGO, [D-Ala^2,*N*-Me-Phe^4,Gly^5-ol]-enkephalin; CeA, central nucleus of the amygdala; PVN, paraventricular nucleus.

nucleus of the amygdala (CeA) on intake of preferred and nonpreferred diets. The PVN is generally regarded as an important site for regulation of energy metabolism and the CeA as a site involved in affect. These investigators noted that lower doses of naltrexone had a greater effect on preferred than nonpreferred diets when injected into the CeA. However, the naltrexone dose effect on intake of the preferred and nonpreferred diets was the same after injection in the PVN. This suggests that although opioids are clearly involved in reward-related feeding behavior, the site of injection is important. Thus, site of action may affect whether regulators impact affect reward or hunger.

It is also important to point out that although taste and hunger are major determinants of food intake, eating in animals and humans is impacted by a variety of other factors as well. Environmental stress, physical activity, time of the day, social influence, impulse control, and other factors clearly affect eating behavior. A priority for future research is to determine the degree to which these diverse influences are mediated by common neurophysiological mechanisms.

Yeomans et al. (42) reviewed evidence indicating that there are dissociable systems mediating homeostatically and hedonically based eating in humans as well. For instance, in one study, the serotonin drug D-fenfluramine reduced hunger but had no effect on ratings of a food's pleasantness (43). Opioid antagonists have also been shown to reduce a food's pleasantness ratings without affecting hunger (44).

The Genesis of Hedonically Driven Eating

Because humans have few innate taste preferences, food palatability is mostly a learned phenomenon (42). Liking of particular foods develops, in part, through associations between ingestion of foods and the delivery of metabolizable energy during digestion (45). However, foods and flavors that acquire positive valence through conditioning seem to retain their motivational properties independently of one's current level of energy repletion (e.g., when dessert is desired and consumed after a satiating meal). Similarly, consumption of highly palatable food is greater than consumption of less palatable food even when the energy and macronutrient content of the meals are the same. For instance, consumption of pasta is greater when the tomato sauce is appropriately spiced than when it is unspiced and bland (42). Thus, once acquired, the palatability of a food appears to be maintained, at least in part, by orosensory reward, not simply by the delivery of energy associated with the food (42). The same process is presumably responsible for the habitual consumption of foods (e.g., sugarless gum, diet soda) that are pleasant tasting but deliver few or no calories.

Potential Interactions among Needing, Wanting, and Liking

Up to this point, the concept of needing food has been tied to an energy deprived state and the concept of wanting food has been tied to its reward value (independently of need). However, it is also important to consider how need- and reward-based motivations to eat may interact. Our summary of this topic is drawn from a recent review paper by Yeomans et al. (42). As these authors point out, it has long been known that energetic need (i.e., physiological hunger) enhances food palatability (46,47). However, the fact that food palatability is enhanced by energetic need does not mean that food palatability depends on a need for energy. There is now considerable evidence that the influence of palatability on intake can simply be a function of orosensory stimulation of reward pathways (42). For instance, although it might be expected that there would be an inverse relationship between the energy content of a preload and the rated palatability of foods eaten after the preload, most studies have found no relation between a preload's energy content and change in palatability of subsequently eaten food (42). Furthermore, consumption of highly palatable foods can actually delay the reduction in hunger that normally occurs as a meal is eaten. Yeomans et al. (42) conclude that these results: ". . . 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, contradicting ideas that satiety and orosensory reward have either additive or positively interacting effects. The implication is that palatability may lead to over-consumption, particularly when sated" (p. S8).

The distinction made in this paper between needing to eat (because of an energy deficit) and wanting to eat (because of the rewarding properties of a food) is somewhat reminiscent of the distinction that Berridge and Robinson have made between wanting and liking a food (48). Although wanting and liking a food are usually viewed as indistinguishable, these investigators have demonstrated that the two appetitive motives are dissociable. Liking for tastes is mediated by opioid neurotransmission in brain circuits similar to those implicated in drug reward. In contrast, manipulation of dopamine systems in the brain markedly increases motivated behavior to obtain food rewards but does not change liking for the food (assessed by animals' affective facial expressions). Wanting and liking have also been distinguished in research with humans. Epstein et al. (49) recently demonstrated that food deprivation increased the reinforcing properties of food (as measured by the amount of work performed to obtain food) but did not influence subjects' hedonic evaluation of the food.

It is clear that the distinction between wanting and liking, although relevant to food intake and to ingestion of drugs, does not adequately address the motivational impact of the

biological need for food. That is, unlike addictive drugs, there is an innate need to consume food. Thus, although the notion of needing food is inherent in life itself, the notion of needing addictive substances (e.g., alcohol, nicotine) makes no sense in the absence of ingestion of and eventual dependency on these substances. The need for alcohol, nicotine, and other drugs is acquired and at least partially reversible, whereas the need for food is innate and irreversible (except, perhaps, in some disease states such as anorexia nervosa). Therefore, although the distinction between wanting and liking is just as pertinent to food intake as to drug use, the concept of innate need is unique to food intake.

These observations give rise to the need to differentiate the way in which the needing/wanting distinction made in this paper differs from the wanting/liking distinction made by Berridge and Robinson (48). The needing/wanting distinction made here refers only to a person's current energy status, with need-based eating referring to intake motivated by an energy deprived state and want-based intake referring to intake driven primarily by external factors such as time of day or the availability of palatable food. Berridge and Robinson's wanting/liking distinction, on the other hand, has, to date, been based on the different brain circuits mediating the two motivations or on the operations used to assess them (for wanting, the degree of work expended to obtain food; for liking, the existence of positive affective facial expressions when tasting palatable substances). When applied to the motivation to eat, the wanting/liking distinction of Berridge and Robinson appears to be theoretically orthogonal to the dimension of need as used in this paper. That is, animals and humans may want food (i.e., be willing to work for it) and like food (i.e., readily consume it when available and show positive affective reactions to it) both when an energetic need is present (after food deprivation) and when it is absent (when desert is desired after a filling meal). At an empirical level, however, energetic need at least sometimes interacts with wanting and liking [e.g., Epstein et al.'s (49) demonstration that food deprivation increased willingness to work for food but not affective evaluations of the food].

Implications of a Dual-Factor Model of Hunger for Eating Disorders and Obesity

The homeostatic-hedonic model of hunger described here has implications for eating disorders and obesity that are quite different from those that have been drawn from the standard model of hunger (3,7,50). The homeostatic hunger system is obviously activated by the massive weight losses shown by anorexic patients and by equally large weight losses (51,52) often shown by bulimic patients in the early stages of their disorder. This sort of major long-term energy deficit is vastly different from the type of mild, short-term dieting shown by the great majority of restrained eaters and dieters studied in restrained eating research (53–57). Reviews of literature on restrained eating and dieting have shown that neither current dieting nor a history of repeated past dieting (so-called yo-yo dieting) can account for the disinhibitory eating demonstrated by restrained eaters identified by Herman and Polivy's (1980) Restraint Scale (56,58,59) or for binge eating in obese individuals (8). This research suggests that the Restraint Scale reflects tendencies toward disinhibition that are not driven by dieting behavior. In fact, the label disinhibition to refer to eating induced by social and emotional cues (60) appears to be a misnomer because such eating does not depend on prior eating restraint (61). Conversely, restraint measures that seem to better describe actual attempts at food restriction do not predict disinhibitory eating (56). Recent research has also shown that both measures of unsuccessful dieting (i.e., the Restraint Scale) (55) and measures of successful dieting (e.g., the Cognitive Restraint scale of the Eating Inventory) (60) do not reflect hypocaloric dieting in the natural environment when intake is assessed objectively and unobtrusively (rather than by self-report) (54,57) and prospectively predict weight gain, not weight loss (62–66).

All of these findings indicate that self-reported restrained eating is not associated with sufficient restriction of energy intake to induce an energy deficit or engage the homeostatic mechanisms that might counter such a deficit. Even overweight individuals who report themselves to be restrained eaters (67) or dieters (68) demonstrate eating regulation, rather than counter-regulation, after consumption of a high-calorie preload. Indeed, the only study that has produced clear-cut counter-regulatory eating in obese individuals was carried out just before the start of a dieting program (69), when obese participants are typically at or near the highest weight they have ever been.

Experimental studies that manipulate energy balance point to the same conclusion. When obese individuals lose weight in a formal weight loss program, binge eating dramatically decreases (8) and remains below pretreatment levels for years (70). Even individuals who are in or close to the normal-weight range show decreased, rather than increased, bulimic pathology when they are put on a diet to lose weight (71) or to avoid weight gain (101).

The implication of these findings is that much of the increasing prevalence of restrained eating and dieting in children (72) and adults (73) during the past 20 years is likely a consequence of passive overconsumption (74) and hyperphagia produced by the obesigenic environment, not a cause of such behavior (59,75). That is, most dieting appears to represent a response to the effects (i.e., a positive energy balance and weight gain) of hedonically-based mechanisms that are activated by the omnipresence of highly palatable foods in the environment. Furthermore, although any weight that is lost on a diet is unlikely to be maintained, this simply means that dieting is an ineffective means of curbing chronically activated hedonic motivations

to eat (59,75), not that it creates homeostatically driven counter-regulatory responses to an energy deficit. The fact that highly palatable foods are often eaten in the absence of an energy deficit means that dietary restraint may be necessary to prevent weight gain in people who are prone to it, which appears to be the majority of people in many developed countries (42).

The long-term ineffectiveness of dieting may reflect the fact that, among obesity-prone individuals, people's self-regulatory skills are not up to the task of perpetually counteracting the pernicious effects of an obesigenic environment (75,76). This conclusion is supported by a study by Hensrud et al. (77). They put overweight women on weight loss diets until they reached a weight in the normal-weight range. The women were neither seeking weight loss nor taught anything about how to lose weight or maintain the weight they lost. Hensrud et al. then compared the weight regain of these women with the average weight regain of participants in 16 previous clinical outcome studies of lifestyle programs for long-term weight loss. The rate of weight regain shown by the two groups over a 4-year period was indistinguishable, despite the fact that the latter group had sought weight control treatment and had spent several months learning how to lose weight and maintain the loss.

The disappointing long-term results of weight loss trials are usually interpreted to mean that dieting is ineffective (15,78) or that dieters are prone to relapse (79). However, from the dieter's point of view, the most immediate and compelling goal of dieting is weight loss (which usually happens), not weight loss maintenance (which usually does not). Thus, the tendency for dieters to regain lost weight could just as well be taken as evidence of the potency of the food environment as of the impotence of diets. Furthermore, little consideration has been given to the possibility—indeed the likelihood—that those individuals who go on weight loss diets are precisely those whose appetitive system is most sensitive to a food-laden environment (80). There is evidence that overweight people find eating more rewarding than do normal-weight individuals (81). A relationship between the apparent rewarding effects of sweet taste and obesity was supported in a study by Stunkard et al. (82), who showed that sucking avidity for a nonnutritive sweet solution in 3-month-old infants predicted weight gain at 2 years of age. Drewnowski and Schwartz (83) have shown that obese individuals have higher preferences for dietary fat than those of normal weight, and several studies have found a relationship between preference for higher fat foods and level of adiposity (42). Several neuroimaging studies (84–87) have also found evidence consistent with the hypothesis that overweight individuals generate weaker satiety signals after eating than do normal-weight individuals, which would mean that when lean individuals have stopped eating, overweight people continue to find eating rewarding. The aforementioned findings again suggest that overweight individuals, above and apart from the difficulty they may have in eating less than they need (i.e., to lose weight), will often experience difficulty in eating less than they want (i.e., to avoid weight gain or, after weight loss, weight regain).

A final point about hedonically-driven eating is that there is likely more than one type of such eating. For instance, a normal-weight person may develop a habit of snacking after lunch and consume an average of 50 kcal/d beyond his energy needs, thereby gaining several pounds in a year. An obese individual with Binge Eating Disorder (88) may consume thousands of calories in a short period of time, causing considerable gastrointestinal discomfort and emotional distress. Although both types of overconsumption would presumably be provoked by hedonic (rather than homeostatic) motives, the particular motives involved likely differ considerably. One frequently cited motive that may be relevant in the latter case is eating-induced reduction of negative affect (20,89).

Implications of the Homeostatic-Hedonic Model for Weight Control

Much of the anti-dieting sentiment that has arisen in the past 30 years is based on the idea that attempting to restrict food intake to conform to societal norms for thinness has a variety of adverse psychological and behavioral effects. For instance, restrained eating has been blamed for counter-regulatory eating, hyperemotionality, distractibility, stress-induced eating, salivary hyper-responsiveness, and other problems (6,90). However, research has demonstrated that such outcomes are not due to hypocaloric dieting (8,59,91,92). Therefore, it appears more likely that these adverse correlates of restrained eating stem from heightened appetitive responsiveness to the food environment, which is essentially what many obesity researchers asserted over 30 years ago (93–95). Indeed, Rodin (93) concluded that restraint "is only a descriptive term and not a mechanism. Restraint is what some people do if they feel compelled by external cues" (p. 364).

Timmerman (96) recently suggested a term to describe the psychological state of eating less than one wants: perceived deprivation. This state may be similar to the concept of reward deprivation, a term introduced in a previous section to describe changes in opioid circuitry (after a high-fat/sucrose diet) that may be functionally similar to energy deprivation. Timmerman (97) had previously shown that, among obese women who regularly engaged in binge eating, caloric intake (mean = 2139) on the days prior to their highest calorie binge days did not reflect hypocaloric dieting. [In fact, because obese individuals substantially under-report their caloric intake (98), this figure is probably an underestimate of actual caloric intake.] In her 2003 study, Timmerman constructed a measure of perceived de-

privation, which was derived by averaging subjects' responses to two questions tapping the degree to which they ate as much as they wanted each day and the degree to which they ate the foods they wanted to eat. Scores on this measure of perceived deprivation, which were collected daily for 14 days, did not correlate with actual caloric intake on the same day, indicating that perceived deprivation did not stem from actual caloric deprivation. However, perceived deprivation scores averaged across 14 days did significantly correlate with Herman and Polivy's Restraint Scale. These results are consistent with the arguments presented above in that many obese individuals (and restrained eaters generally) may experience a sense of deprivation not because they are eating less than they need but because they are eating less than they want. If this interpretation is correct, then the best way of reducing the perception of chronic deprivation is not to try to convince such individuals that they are not really hungry (16) but to reduce their daily exposure to highly tempting foods that are likely to activate their hedonic motivation to eat (69).

Recent studies on an animal model of binge eating have produced results consistent with this viewpoint. Corwin and colleagues demonstrated that providing rats limited access to high-fat food produces a binge-like eating pattern even if the rats were never food deprived (99); that is, these animals had continuous access to chow. Indeed, such binge-like behavior occurs even in the absence of undereating on the day preceding binge-like episodes (100). Thus, this binge-like pattern appears to occur because the animals were restricted from eating as much high-fat food as they wanted, not because they were restricted from eating as much chow as they needed.

There is one final implication of the existence of brain motivational systems that underlie homeostatically and hedonically driven food intake. This is that the motivation to eat more than one needs is every bit as real, and, perhaps, every bit as powerful, as the motivation to eat when truly energy deprived. From this perspective, the most relevant question is not why so many people in developed countries are overweight but why everyone is not overweight. Against the background of a biologically based motivation to eat when palatable food is available and the unlimited opportunities to eat as much food as one desires, it appears that attempts to facilitate long-term weight control by bolstering self-regulatory skills is unlikely to succeed (75). A superior strategy may be to bring under control the availability, portion size, and composition of food at the individual and, eventually, at the population level.

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