Hedonic hunger: A new dimension of appetite?

Michael R. Lowe *, Meghan L. Butryn

Department of Psychology, Drexel University, Philadelphia, PA 19102, USA

Abstract

An increasing proportion of human food consumption appears to be driven by pleasure, not just by the need for calories. In addition to its effects on body mass and health, the food environment in affluent societies may be creating an appetitive counterpart to the psychological effects of other hedonically-driven activities such as drug use and compulsive gambling. This phenomenon is referred to here as "hedonic hunger." Animal literature is reviewed indicating that brain-based homeostatic and hedonic eating motives overlap but are nonetheless dissociable. In humans there is evidence that obese individuals prefer and consume high palatability foods more than those of normal weight. Among normal weight individuals it has long been assumed that the appetitive anomalies associated with restrained eating are due to diet-induced challenges to the homeostatic system, but we review evidence suggesting that they more likely stem from hedonic hunger (i.e., eating less than wanted rather than less than needed). Finally, a recently-developed measure (the Power of Food Scale; PFS) of individual differences in appetitive responsiveness to rewarding properties of the food environment is described. Preliminary evidence indicates that the PFS is reliable and valid and is related to clinically-relevant variables such as food cravings and binge eating. This measure, combined with environmental manipulations of food availability and palatability, may constitute a useful approach to studying hedonic hunger.
© 2007 Elsevier Inc. All rights reserved.

Keywords: Hunger; Hedonic; Reward; Appetite; Food intake; Eating; Addiction

I pursue pleasure, but stingily, suspiciously. Mason Cooley

Do not bite at the bait of pleasure, till you know there is no hook beneath it. Thomas Jefferson

1. Introduction

These aphorisms suggest that the attainment of pleasure is both desirable and dangerous. For the vast majority of human history and prehistory, the primary objective of seeking food was survival through the maintenance of energy homeostasis and the avoidance of starvation. In modern times, among well-nourished populations, most food consumption occurs for reasons other than acute energy deprivation [1]. As the growing prevalence of global obesity suggests, an increasing proportion of human food consumption appears to be driven by pleasure, not just by the need for calories.

The purpose of this paper is to propose and justify a distinction between homeostatic and hedonic eating and to discuss the potential consequences of what, from an historical perspective, represents an unprecedented societal phenomenon: the constant availability and frequent consumption of highly palatable foods. We agree with other observers [1] that this phenomenon is contributing to escalating obesity and its physical comorbidities (diabetes, heart disease, etc.). However, we also propose that the food environment may be creating an appetitive counterpart to the psychological effects of other hedonically-driven activities such as drug use and compulsive gambling. Just as compulsive gamblers or drug-dependent individuals are preoccupied with their habit even when they are not engaging in it, so may some individuals experience frequent thoughts, feelings and urges about food in the absence of any short- or long-term energy deficit. These experiences may or may not be prompted by exposure to food-related cues but by definition they do not occur in response to prolonged food deprivation (i.e., homeostatic hunger). We refer to this tendency here as "hedonic hunger".

It is important to emphasize that the term hedonic hunger, like the traditional concept of hunger, is meant to refer to a subjective

* Corresponding author. 245 N. 15th St., MS 626, Philadelphia, PA 19102-1192, USA. Tel.: +1 215 762 4948; fax: +1 215 762 7441.
E-mail address: lowe@drexel.edu (M.R. Lowe).

state (and to physiological mechanisms that may mediate it), not to actual food intake. Research suggests that traditional self-reports of hunger are only weakly related to the amount of food that is subsequently consumed [2,3], and the same may be true for the concept of hedonic hunger. It also is possible that subjective hunger ratings are more reflective of a simple propensity towards eating, rather than serving as an index of how much food will be consumed. These observations do not mean that measures of hedonic hunger will not predict food intake, only that the term should be defined independently of actual food intake. In addition, the immediate food environment is presumed to play a much greater role in the generation of hedonic than of homeostatic hunger. The development of homeostatic hunger is based on the prolonged absence of energy intake. The palatability of the food that an individual is exposed to during and between eating bouts is assumed to be largely irrelevant to the generation of homeostatic hunger. The opposite appears to be true of hedonic hunger. That is, satiety has a relatively small effect on the pleasantness of foods (the pleasantness of a meal does not decline nearly as much as hunger as the meal is consumed). Rather, the availability and palatability of foods in the immediate environment has a major effect on whether they will be desired and consumed [4-6] and an individual's level of current caloric repletion is assumed to be relatively unimportant.

A further implication of these distinctions is a difference in the experimental methods one would use to study these two eating motives. Studies of homeostatic hunger would obviously involve the manipulation of hunger via differing lengths of food deprivation. Studying hedonic hunger appears to have two requirements. One is assuring that participants are in a state of short-term energy repletion — i.e., they are not in a state of impending or actual energy deficit (e.g., shortly before a meal). The other is that the experimental context examining hedonic hunger involves the introduction of some kind of highly palatable food stimulus. The reason for the second requirement is that the state of hedonic hunger is presumably a latent potentiality that can only be "called forth" by the introduction of highly-pleasurable food stimuli.

A final introductory comment is needed based on the distinction that Berridge and Robinson have made between "wanting" and "liking" a substance. Although these two motivational drives normally go hand-in-hand, these researchers have shown that they are subserved by neurophysiological mechanisms that can be experimentally differentiated. We previously addressed the relation of this distinction to the distinction we are drawing between homeostatic and hedonic eating motives (see reference [7], pp. 800–801). We simply point out here that when discussing hedonic eating motives, such motives could be driven by the incentive salience of food stimuli (i.e., "wanting"), by the taste of food ("liking"), or by both.

2. Differentiating between homeostatic and hedonic hunger

The term "hunger" has historically been used to describe a biological state of acute energy deprivation or the subjective state presumably reflecting an actual or impending state of energy deprivation. However, although the use of a period (e.g., at least 8 h) of food deprivation creates an unambiguous state of homeostatic hunger, it is much less clear what self-report measures of subjective hunger level used in hundreds of past studies are actually measuring. For instance commonly-asked questions such as "How hungry do you feel right now?", "How strong is your desire to eat right now?" and "How much food do you think you could eat right now?" [8] were designed to assess what we are referring to as homeostatic hunger but, depending on the experimental context, could just as well be tapping what we refer to as hedonic hunger. For instance, if study participants consumed a normal dinner and were then provided with an array of their favorite deserts to sample ab lib, their answers to these questions would presumably reflect a fair degree of "hunger", despite having recently finished dinner. Additional evidence that self-reported hunger ratings often reflect some (unknown) mixture of homeostatic and hedonic eating motives comes from a study that examined pictorial measures of physical hunger (based on how many body areas were identified as reflecting current hunger levels) and traditional verbal measures of hunger repeatedly during a 22-hour fast [8]. The researchers found that the pictorial measure increased more rapidly than the verbal measure as the fast proceeded and also found that correlations between the verbal and pictorial measure were few in number and modest in size. These results suggest that verbal measures of hunger used in past studies are measuring something more than physical sensations of food deprivation. From an historical perspective there was no need to refer to hunger as "homeostatic" because this function of hunger was inherent in the term itself. However, since both food deprivation and the availability of palatable food (when energy replete) can produce strong motivations to eat, a priority for future research will be to differentiate between these two types of motivation to eat.

For most of the history of research on human and animal eating regulation it has been assumed that food intake is tightly regulated by homeostatic mechanisms and that deviations in energy intake in either direction will be corrected by behavioral and metabolic regulatory responses [9,10]. The obesity epidemic and research on topics such as the effects of portion size and food palatability on food intake have made it abundantly clear that upward deflections in energy intake are not well compensated for by reductions in subsequent intake [11]. There has also been a significant increase in research and commentary addressing the question of how and why the homeostatic system for eating regulation is being overridden [7,12,13].

One compelling question raised by this work is whether the concept of hunger is still relevant when discussing intake that supersedes energy needs. We suggest that it is still relevant but in a fundamentally different way. The reason is based on the paradoxical notion that both the absence and the presence of food can stimulate "hunger," albeit of different kinds. One illustration of this paradox is that hunger sometimes decreases over time when obese people are put on a very low-calorie diet and lose substantial weight [14]. Part of the reason for this may be that participants are exposed to and consume much less

conventional food while losing weight. If regular exposure to palatable food stimulates hunger, then a large reduction in such stimulation may reduce self-perceived hunger despite the existence of a significant energy deficit. Also consistent with the idea that exposure to palatable food can induce hunger is research showing that 1) relative to a bland food, consumption of a palatable food actually produces a small increase in hunger early in a meal, as well as a delay in the rate of decline of palatability as the meal progresses, and 2) self-rated appetite is increased by merely seeing a preferred food, 3) consumption of an equi-caloric palatable as opposed to bland preload produced less reduction in hunger in the former condition, and 4) consumption of palatable foods produce a more rapid recovery of hunger than less palatable foods up to 3 h following their consumption [13].

We do not mean to suggest that the line separating "homeostatic" and "hedonic" hunger is clear-cut. Part of the reason for this is that even consumption that is part of a "normal" eating pattern (e.g., three meals a day) does not appear to be strictly homeostatic since meals are usually initiated by the anticipation, not by the actual existence, of a short-term energy deficit [15]. There are two distinctions, however, that are helpful in differentiating homeostatic and hedonic hunger. The first is that while food palatability is relevant to both types of hunger, it appears to be essential to the definition of hedonic hunger. That is, at least part of the reason why some foods may still be desired and consumed when no energy deficit exists is assumed to be the rewarding properties of the food based on its gustatory properties rather than its energy content. Second, unlike homeostatic hunger, hedonic hunger can only be studied in the absence of a short-term energy deficit because otherwise the motivation behind appetitive behavior could be partially or totally based on the physical need for energy.

Some examples of the two types of hunger under discussion might also be helpful. For instance, the eating motivated by a fast of 12 or more hours is clearly homeostatic in nature, as is the chronic hunger a dieter might experience while losing weight. On the other hand, in a stable-weight person, desiring a dessert after a filling meal would be an example of hedonic hunger, as would a sudden craving for donut in the middle of the afternoon, after walking by a donut shop and smelling fresh donuts being made. These examples provide a sense of why hedonic hunger could become a powerful influence on food intake and, ultimately, body mass: The omnipresence of highly palatable food may be sufficient to induce hedonic hunger in its own right [5] and may also condition a variety of social (e.g., parties) and physiological (e.g., cephalic phase insulin responses) cues that become associated with hedonically-driven eating.

There has been a strong trend in the literature to attribute eating beyond energy needs to a variety of psychological motives (e.g., escape from self-awareness, self-medication, emotional hunger). However there are now several studies showing that a variety of non-stressful cognitive activities increase food intake, particularly in restrained eaters [16]. Thus, when food is available, it may be that certain individuals are likely to increase their intake when engaged in any cognitively absorbing task even if the task does not generate stress or negative affect. It may

be that restrained eaters have a chronic, often-latent susceptibility to overconsume palatable foods and that cognitively absorbing or compelling environmental events – including, but not limited to stressful events – divert attention away from available food, allowing the latent susceptibility to manifest itself in increased eating.

Another intriguing explanation for the relation between stress and consumption of good-tasting foods was suggested by a recent study by Teegarden and Bale [53]. These investigators found that adding highly palatable foods to the all-chow diet of rats produced a reduction in a stress-related peptide (corticotrophin-releasing factor) relative to control animals who were maintained on chow. Furthermore they showed that after being on a diet that included highly-palatable food for 4 weeks, return to an all-chow diet produced an increase in several physiologic and behavioral indicators of stress. If these results can be generalized to humans, they suggest that palatable foods may indeed have anxiolytic effects and that ceasing consumption of highly palatable foods could increase stress and hasten a return to eating them. The potential parallel between this cycle and that involved in drug addiction and relapse is obvious. These results also potentially provide a whole new way of viewing the nature of "emotional eating" in humans.

Much of the evidence supporting a distinction between homeostatic and hedonic hunger has been generated with animal models. We therefore turn next to a summary of this research. We follow this with a discussion of research relevant to hedonic hunger in humans and then review our recent development of a new measure to assess the construct of hedonic hunger.

3. Animal research on hedonic eating motives

A large body of research indicates that there are neurophysiological substances (e.g., neurotransmitters, hormones) and pathways that operate within homeostatic and hedonic systems to help regulate eating behavior. Systems that control eating for energy homeostasis and for reward overlap but are also functionally dissociable [7]. Much of the research that supports the distinction between eating that is primarily motivated by energy deficits and by pleasure has been conducted with animal models.

3.1. Neurophysiological research relevant to hedonic hunger

The existence of distinguishable neural networks that motivate hunger- and hedonically-based eating has been documented in animal studies. Manipulating certain neuropeptides that affect hunger has been shown to have little or no effect on reward-based eating. For instance, administration of neuropeptide Y (NPY) induces a state that appears to resemble physiologically-based hunger, as reflected by the finding that sated rats will eat bland chow when NYP is administered [17,18]. Food-deprived rats also show increased gene expression of NPY in the arcuate nucleus of the hypothalamus [19]. When NPY is administered to non-deprived rats in an operant chamber, they will work for food (i.e., by lever pressing) in a

manner similar to rats who have been food-deprived for 36–48 h [20]. Another example of the distinction between these systems is that the drug D-fenfluramine, which affects serotonin circuitry, has been found to reduce hunger without altering ratings of the pleasantness of food [21].

By contrast, manipulating other peptides that affect reward-based eating have little effect on eating when hungry. A separate system of opioid peptides appears to regulate eating that is motivated by the rewarding properties of food [22]. When opioid receptors are blocked by administration of the opioid antagonist naloxone, it significantly reduces intake of preferred diets in rats deprived of food for 24 h, but has no effect on intake of a nonpreferred diet [23]. Similarly, when rats are restricted to a diet that reduces them to 85% of their body weight, administration of naloxone decreases intake of palatable food (i.e., a sucrose-based diet) but not of a less palatable cornstarch diet [24]. Results of taste reactivity tests, which document rat facial expressions that are associated with intake of palatable foods, indicate that naloxone decreases the hedonic properties of ingesting a sucrose solution [25]. When rats are fed a high-fat/sucrose diet or a less palatable cornstarch diet, each with equivalent calories, expression of dynorphin, an opioid peptide, increases only in the high-fat/sucrose diet [26,21]. This provides further evidence that opioid peptides are related to hedonic eating and more specifically to the pleasurable (as opposed to the energetic) property of good-tasting food. A possible human equivalent to this study would be comparing people fed small and equi-caloric amounts of a food high or low in palatability (e.g., pastry versus plain oatmeal). The strength of the desire for more pastry among pastry-consumers would likely be greater than the strength of the desire for more oatmeal among oatmeal consumers, a difference that would be based on a perceived deprivation of pleasure, not of calories.

Because food reward and drug reward appear to have common neural pathways [27,28], addiction models have been used to investigate the hedonic properties of palatable food. In one study, researchers [29] deprived an experimental group of male rats for 12 h per day and then offered a 25% glucose solution in addition to chow for the next 12 h, so that periods of fasting and excessive sugar intake alternated. A control group was fed *ad libitum* chow. After 8 days, these rats were either administered naloxone or saline. Experimental rats who were injected with naloxone showed significantly more withdrawal symptoms than those who were injected with saline. Naloxone resulted in signs of anxiety and changes in the nucleus accumbens that are consistent with morphine withdrawal; these changes were not observed in control animals who were administered naloxone. The researchers also compared experimental and control rats after 24 h of food deprivation on Day 9. Teeth chatter, forepaw tremor, and head shaking were observed at significantly higher rates in the experimental group than the control group. Taken together, these results indicate that when fasting and excessive sugar intake are alternated, withdrawal subsequently can be induced either by administration of an opioid antagonist or by food deprivation. This study provided further evidence that dependence on naturally activated endogenous opioids may occur in the context of eating palatable foods.

Another study [30] of the potentially addictive properties of sugar involved repeatedly giving rats access to sugar for 12 h daily and then depriving them of sugar for 2 weeks. A comparison group was given access to sugar for 30 min daily before undergoing the 2 weeks of sugar deprivation. The former group showed an enhanced response to sugar (as measured by lever pressing), presumably because the greater prior exposure to sugar produced a stronger addictive response to its withdrawal. This pattern of response is similar to that observed in studies of addictive substances such as alcohol. This lends further support to the suggestion that palatable foods can produce effects similar to those observed in drug addiction.

3.2. Motivational effects on eating behavior

The effect of hedonic properties of food on a behavioral indicator of eating motivation has also been studied. Barbano and Cador [31] examined eating motivation in food-restricted and food-sated rats given access to either normal chow or highly palatable chocolate-flavored cereal. Running speed on a runway test was used to operationalize the strength of their appetitive motivation. (Of note, a preliminary experiment demonstrated that the effect of palatability could be dissociated from that of the chocolate cereal's novelty.) Food sated-rats ate large amounts of chocolate cereal. In the runway test, they also ran almost as fast for the cereal as food-deprived rats did for the chow. These results suggest that palatable food may motivate eating in excess of what is necessary for homeostasis and that the strength of this motivation may be indistinguishable for the homeostatic motive to obtain food energy under conditions of caloric deprivation.

4. Human research on hedonic eating motives

There is abundant evidence that BMI is related to preferences for and consumption of highly palatable (often, high-fat) foods [4,7]. However, as noted by Blundell and Finlayson [4], this relationship may not be linear in nature. For instance, they review evidence that overweight individuals score higher on a measure of reward sensitivity than obese individuals [32] and that the availability of the dopamine D2 receptor (a possible mediator of the rewarding property of palatable foods) was inversely related to BMI among obese individuals [33].

Regardless of the precise nature of this relationship, the evidence relating preferences for palatable foods to BMI is consistent with the conclusion that the widespread availability of energy-dense and highly palatable foods is a major contributor to weight gain and to the current obesity epidemic [4,7]. However, comparing normal weight and obese individuals' preferences for and consumption of highly palatable foods is of limited value in understanding the source of such differences because it is impossible to determine whether they preceded (and perhaps contributed to) or followed the development of the obese state. Comparisons of normal weight and reduced-obese individuals (who have reduced their weight from the obese to the normal weight range) partially solves this problem, but leaves open the possibility that characteristics of post-obese individuals stem from their previous obese state or

from their current weight suppressed state. This is especially the case because there is evidence that weight suppression – the existence of a large discrepancy between highest ever and current body weight – itself influences food intake and taste preferences [34].

There is reason to believe that normal weight restrained eaters may represent a group that has heightened susceptibility to the widespread availability of highly palatable food. Herman and Polivy [35] introduced the construct of restrained eating and conducted the first research demonstrating that restrained eaters are highly sensitive to food cues generally and to disinhibitory eating in particular [36]. These authors have long argued that individuals who were dieting or chronically restraining their intake (they use the terms interchangeably) are susceptible to the disinhibiting influence of eating diet-prohibited food or experiencing negative affect. They have assumed that the primary influence driving dieting is the desire to achieve a societally-endorsed but unrealistic level of thinness [37].

Herman and Polivy's boundary model [38] suggests that the eating behavior of non-dieters is largely regulated by biological cues of hunger and satiety but that restrained eaters impose a "diet boundary" on their eating to limit their food intake. When this self-imposed limit is exceeded or transgressed (e.g., by eating a "forbidden" food or experiencing emotional distress) restrained eaters become disinhibited and eat until they reach their (typically elevated) satiety boundary. Because disinhibitory eating is viewed as undermining and eventually reversing any weight loss produced by dieting, the developers of restraint theory assume that restrained eaters will exhibit wide fluctuations in their food intake and body weight but will lose little if any weight in absolute terms [39].

However, there have been two developments in research on restrained eating that indicate that restrained eaters' hyper-responsiveness to food cues and susceptibility to disinhibitory eating is more likely part of a predisposition toward weight gain rather than a consequence of chronic dieting. One development is that dieting does not appear to be responsible for the negative appetitive or metabolic effects associated with restrained eating. Most restrained eaters are not currently dieting to lose weight and those who are on a diet — who according to restraint theory, should be most susceptible to disinhibition — show reduced, rather than enhanced, susceptibility to disinhibitory eating [34]. In a randomized controlled investigation, Presnell and Stice [40] found that women who were randomly assigned to lose weight on a reduced-calorie diet showed reduced rather than increased bulimic symptoms. Furthermore, although restrained eaters often report consuming fewer calories than unrestrained eaters on self-report measures of food intake, two multi-study papers (one of which – by Stice, Cooper, Schoeller, Tappe and Lowe – is unpublished) examining energy intake in everyday life (based on observations of food intake and on energy expenditure estimated by doubly-labeled water) have shown that the naturalistic intake of restrained and unrestrained eaters does not differ significantly [41]. At the same time, normal weight restrained eaters show several metabolic characteristics (e.g., low leptin and insulin levels) that should make them prone to weight gain [16]. Indeed, while there are no

studies suggesting that restrained eating predicts future weight loss, there are several studies showing that measures of restrained eating [42–44] and dieting [45] predict future weight gain. Taken together, these findings suggest that the metabolic and appetitive characteristics of restrained eaters are not due to low calorie dieting. Rather, these characteristics appear to make those we label as restrained eaters susceptible to weight gain and therefore to frequent dieting to reverse or prevent weight gain.

The second research development that suggests that Herman and Polivy's conceptualization of restraint is a proxy for susceptibility to weight gain involves the fact that the Restraint Scale consists of items measuring both dietary concerns and disinhibitory eating. If restrained eating was itself responsible for the various appetitive anomalies associated with this scale, then "purer" measures of restraint (which exclude items assessing overeating) should also predict disinhibitory eating. However neither the Restrained Eating scale from the Dutch Eating Behavior Questionnaire nor the Cognitive Restraint scale from the Three-Factor Eating Questionnaire predicts stress-induced or preload-induced disinhibitory eating [46–48]. There is evidence, however, that scoring high on both a pure measure of restraint and a measure of disinhibitory eating does predict the kind of counterregulatory eating previously associated only with the Restraint Scale [48]. These findings again suggest that restrained eating per se is not the culprit responsible for overeating in laboratory studies.

In sum, the foregoing arguments suggest that most normal weight restrained eaters are trying to control their food intake not to lose weight but to prevent overeating and weight gain. It is logical to expect that the combination of a susceptibility toward overeating and conscious efforts to avoid overeating would likely result in more frequent instances of "hedonic hunger". Thus the foregoing studies showing that naturalistic caloric intake of restrained eaters does not differ from that of unrestrained eaters could indicate that while restrained eaters are not in a state of deprivation-based hunger, they may be in a state of hedonic hunger. That is, if restrained eaters are chronically prone toward overeating and a positive energy balance (eventually leading to greater weight gain), then finding that restrained eaters eat no more than unrestrained eaters could mean that their restraint is helping prevent their underlying appetite from manifesting itself in increased caloric intake.

This reinterpretation of research on restrained eating is also relevant to the "boundary model" of eating proposed by Herman and Polivy [38] to account for restraint-related effects. In this model Herman and Polivy described the familiar influences that spur eating (i.e., experiencing sufficient food deprivation to cross the "hunger boundary") and that produce a cessation of eating (eating enough to cross the "satiety boundary"). Between these two boundaries Herman and Polivy posited the existence of a "zone of biological indifference" where social and psychological influences are thought to be most influential in determining what, when and how much food is eaten. However, the evidence reviewed in this paper suggests that it may be more accurate to call the area between the hunger and satiety boundaries the "zone of homeostatic indifference" rather than

the "zone of biological indifference." The body is still biologically motivated to eat within this zone but the motivation depends on external factors such as the availability of palatable food and permissive eating norms, rather than on an internal need for energy.

4.1. Preliminary research on hedonic hunger

In applying this model to restrained eaters, Lowe and Levine [7] suggested that the construct of restraint might reflect eating less than one wants, rather than less than one needs. They proposed that restrained eaters, though not consuming less than needed for energy balance, may nonetheless feel deprived because the "toxic" food environment creates frequent temptations to eat that they are constantly trying to resist.

Two studies have examined the relationship between hedonic hunger and restraint. The first [49] examined a construct called "perceived deprivation," which is similar to the hedonic hunger construct discussed in this paper. Participants included those reporting binge eating with no history of purging as well as obese individuals who reported current dieting with no history of bingeing. Perceived deprivation was measured at the end of each of 14 days of self-monitoring with the sum of visual analogue scale ratings for two items: *Did you eat enough food?* and *Did you eat what you wanted?* The findings indicated that perceived deprivation was significantly correlated with scores on the Restraint Scale but not with daily caloric intake, which was measured through 14-day food diaries that participants completed after receiving training in measuring and recording intake. The correlation with the Restraint Scale is consistent with our assumption that this scale measures relative deprivation (i.e., relative to what participants would like to have eaten) rather than absolute deprivation (relative to their energy needs for energy balance). The absence of a correlation with caloric intake is also consistent with the latter conclusion.

The second study (Markowitz, Butryn, and Lowe, 2007, unpublished data) examined hedonic hunger in normal weight young women with no history of or current eating disorders. Hedonic hunger was assessed with a 7-item self-report measure, with items similar to those used by Timmerman and Gregg. Participants completed the measure by e-mail daily before they went to sleep at night for a one-week period. This study replicated the finding that perceived deprivation was significantly correlated with scores on the Restraint Scale. It also found that participants with higher levels of subclinical bulimia nervosa symptoms reported greater perceived deprivation. These two studies, along with evidence that restrained eaters are not in negative energy balance [41], provides support for the hypothesis that many restrained eaters may be in a state of *relative* deprivation (because they are avoiding eating as much as they'd like to eat) rather than in a state of *absolute* deprivation (i.e., eating less than they need to eat).

There are several directions for future research in this area. Refining the measurement of hedonic hunger is a challenging but important topic. It would be best to measure hedonic eating contemporaneously with its occurrence (e.g., using ecological momentary assessment), not later on in the day. The appropriate periods to assess it would be the 2–3 h following each bout of food intake. This would help ensure that reported food-related cognitions or motivations that occur would be hedonic rather than homeostatic in nature. Measurement in larger, more diverse samples also is desirable. Additional research efforts might focus on examining the relationship between hedonic hunger and craving and understanding the extent to which hedonic hunger is influenced by environmental versus internal factors or their interaction.

5. A new measure of appetitive responsiveness to the food environment

Despite living in an obesogenic environment, a substantial minority of individuals in the United States remain in the normal weight range their entire lives. Presumably, there is substantial variability in the extent to which individuals are affected by food or think about food even when eating is not imminent or underway. It is precisely during these time periods that hedonic hunger become potentially relevant. A measure that assessed individual differences in hedonic hunger could prove useful for both research and clinical purposes. Our research team (Lowe, Butryn, Didie, Annunziato, Crerand, Ochner, Coletta, Lucks, Wallaert, and Halford, 2007, unpublished data) developed such a measure during the past several years. This measure, the Power of Food Scale (PFS) is reviewed next (and can be obtained from the corresponding author).

The PFS was designed to measure individual differences in appetitive responsiveness. It is only appropriate for use in environments where the population is well-nourished and has a plentiful supply of food available. The PFS is not appropriate in cultures where this is not the case because the regular occurrence of significant deprivation-based (or homeostatic) hunger would likely explain the existence of frequent food-related thoughts or urges to eat.

The initial pool of items for the PFS was collected at pre-treatment from a group of obese women who had enrolled in a weight loss study. The final version consists of 21 items, each rated on a 5-point Likert scale (sample items: "I find myself thinking about food even when I'm not physically hungry." and "Just before I taste a favorite food, I feel intense anticipation."). In a sample of 563 respondents, the PFS was found to be internally consistent and temporally stable over a 4-month period. Factor analyses found it to be comprised of a single factor in both a mostly normal weight and in an overweight group enrolled in a weight loss program.

To test construct validity, the relationship between the PFS and other measures was examined. Additionally, because the Restraint Scale has itself been related to various measures of responsiveness of food cues, analyses were also conducted to determine if the PFS accounted for variance beyond that explained by the Restraint Scale.

There was a moderate correlation between the PFS and the Restraint Scale ($r = .30$). The PFS and the Restraint Scale were regressed on four self-report measures of overeating: the Three-Factor Eating Questionnaire (TFEQ) — Disinhibition scale, the TFEQ — Hunger scale, the Dutch Eating Behavior

Questionnaire (DEBQ) — External Eating scale, and the DEBQ — Emotional Eating scale. The PFS was independently related to all four whereas the Restraint Scale was independently related to two (TFEQ-Disinhibition and DEBQ-Emotional Eating).

Clinical data and analogue studies also provide support of the validity of the PFS. A group of obese participants with binge eating disorder scored much higher on the PFS than control groups of normal weight and obese participants. In an experiment in which undergraduate student participants were given boxes of chocolate to keep with them but not eat for 48 h, baseline PFS scores significantly predicted self-reported cravings for the chocolate as well as consumption of it [50].

Taken together, these findings indicate that the PFS reflects a generalized tendency toward preoccupation with food despite the absence of a short-term energy deficit. The PFS intentionally does not contain items describing actual food consumption or overeating for two reasons. One is that much of the food that people consume everyday is assumed to be driven by homeostatic more than by hedonic motives. Second, if the PFS contained items describing overeating and it was used to predict overeating then it would be subject to criterion confounding. The PFS may also reflect susceptibility to the rewarding properties of food, though this assumption must be tested in future research.

Of note, the PFS was designed to measure hedonic hunger as a trait (and its 4-month test–retest reliability of .79 is consistent with this assumption). However, it is likely that this trait interacts with exposure to food cues, which suggests that combining a measure like the PFS with exposure to food cues (either in laboratory settings or naturalistically) may represent a useful approach to studying hedonic hunger.

6. What might be "new" about hedonic hunger?

We finally return to the title of this paper and specifically to the idea that there might be something "new" about the concept of hedonic hunger. We are of course referring not just to a new term but to the possibility that society-wide changes in both the physical and psychological availability of food has created a type of eating motive in whole populations that has never been seen before. By psychological availability we are referring to the types of norms and expectations governing eating in a particular cultural context. For instance, there was more than enough high-calorie food available to most Americans several decades ago so that Americans could have overeaten as much then as today if they were so motivated. Similarly, there is more than enough palatable food in a country like France so that most French people could consume all the food they might want to. Thus although the food environment in America several decades ago, and the food environment in France today are not as obesogenic as the present American environment, it may not just be changes in the physical availability of food that accounts for the very high prevalence of overweight and obesity in present-day America. Rather it may also be prevailing personal or social norms that it is acceptable, sometimes even desirable, to partake of the available food whenever, wherever,

and however one wants to [51,52]. This permissive norm may be part of what makes the omnipresence of food so compelling — it is not only easy to eat any time of the day or night, but there is often substantial encouragement to do so and few prohibitions against doing so. The combination of an environment filled with highly palatable foods, and cultural norms that make these foods "psychologically available" on an ad lib basis, may paradoxically be a perfect recipe for the generation of both epidemic obesity and widespread hedonic hunger.

Acknowledgements

References

[1] Brownell K, Horgen KB. Food fight: the inside story of the food industry, america's obesity crisis, and what we can do about it. 1st ed. New York: McGraw-Hill; 2003.

[2] Herman CP, Fitzgerald NE, Polivy J. The influence of social norms on hunger ratings and eating. Appetite 2003;41:15–20.

[3] Mattes R. Hunger ratings are not a valid proxy measure of reported food intake in humans. Appetite 1990;15:103–13.

[4] Blundell JE, Finlayson G. Is susceptibility to weight gain characterized by homeostatic or hedonic risk factors for overconsumption? Physiol Behav 2004;82:21–5.

[5] Painter JE, Wansink B, Hieggelke JB. How visibility and convenience influence candy consumption. Appetite 2002;38:237–8.

[6] Yeomans MR, Tovey HM, Tinley EM, Haynes CJ. Effects of manipulated palatability on appetite depend on restraint and disinhibition scores from the three-factor eating questionnaire. Int J Obes Relat Metab Disord 2004;28:144–51.

[7] Lowe MR, Levine AS. Eating motives and the controversy over dieting: eating less than needed versus less than wanted. Obes Res 2005;13:797–805.

[8] Lowe MR, Friedman MI, Mattes R, Kopyt D, Gayda C. Comparison of verbal and pictorial measures of hunger during fasting in normal weight and obese subjects. Obes Res 2000;8:566–74.

[9] Friedman MI, Stricker EM. The physiological psychology of hunger: a physiological perspective. Psychol Rev 1976;83:409–31.

[10] Keesey RE. A set point theory of obesity. In: Foreyt JP, editor. Handbook of eating disorders. New York: Basic books; 1986. p. 63–87.

[11] Levitsky DA. The non-regulation of food intake in humans: hope for reversing the epidemic of obesity. Physiol Behav 2005;86:623–32.

[12] Berthoud, H. Interactions between the "cognitive" and "metabolic" brain in the control of food intake. Physiol Behav in press.

[13] Yeomans MR, Blundell JE, Leshem M. Palatability: response to nutritional need or need-free stimulation of appetite? Br J Nutr 2004;92:S3–S14.

[14] Wadden TA, Stunkard AJ, Day SC, Gould RA, Rubin CJ. Less food, less hunger: Reports of appetite and symptoms in a controlled study of a protein-sparing modified fast. Int J Obes 1987;11:239–49.

[15] Woods SC. The eating paradox: how we tolerate food. Psychol Rev 1991;98:488–505.

[16] Lowe MR, Kral TVE. Stress-induced eating in restrained eaters may not be caused by stress or restraint. Appetite 2006;46:16–21.

[17] Clark JT, Kalra PS, Crowley WR, Kalra SP. Neuropeptide Y and human pancreatic polypeptide stimulates feeding behavior in rats. Endocrinology 1984;115:427–9.

[18] Levine AS, Morley JE. Neuropeptide Y: a potent inducer of consummatory behavior in rats. Peptides 1984;5:1025–9.

[19] Brady LS, Smith MA, Gold PW, Herkenham M. Altered expression of hypothalamic neuropeptide mRNA's in food-restricted and food-deprived rats. Neuroendocrinol 1990;52:441–7.

[20] Jewett DC, Cleary J, Levine AS, Schaal D, Thompson T. Effects of neuropeptide Y, insulin, 2-deoxyglucose, and food deprivation on food-motivated behavior. Psychopharmacol 1995;107:267–71.

[21] Blundell JE, Hill AJ. Serotoninergic modulation of the pattern of eating and the profile of hunger–satiety in humans. Int J Obes 1987;11:141–55.

[22] Levine AS. The animal model in food intake regulation: examples from the opioid literature. Physiol Behav 2006;89:92–6.

[23] Glass MJ, Grace M, Cleary JP, Billington CJ, Levine AS. Potency of naloxone's anorectic effect in rats is dependent on diet preference. Am J Physiol 1996;271:R217–21.

[24] Weldon DT, O'Hare E, Cleary J, Billington CJ, Levine AS. Effect of naloxone on intake of corn starch, sucrose, and polycose diets in restricted and non-restricted rats. Am J Physiol 1996;270:1183–8.

[25] Parker LA, Maier S, Rennie M, Crebolder J. Morphine- and naltrexone-induced modification of palatability: analysis by the taste reactivity test. Behav Neurosci 1992;106:999–1010.

[26] Welch CC, Kim E, Grace MK, Billington CJ, Levine AS. Palatability-induced hyperphagia increases hypothalamic dynorphin peptide and mRNA levels. Brain Res 1996;721:126–31.

[27] Kelley AE, Berridge KC. The neuroscience of natural rewards: relevance to addictive drugs. J Neurosci 2002;22:3306–11.

[28] Volkow ND, Wise RA. How can drug addiction help us understand obesity? Nat Neurosci 2005;8:555–60.

[29] Colantuoni C, Rada P, McCarthy J, et al. Evidence that intermittent, excessive sugar intake causes endogenous opioid dependence. Obes Res 2002;10:478–88.

[30] Avena NM, Long KA, Hoebel BG. Sugar-dependent rats show enhanced responding for sugar after abstinence: evidence of a sugar deprivation effect. Physiol Behav 2005;84:359–62.

[31] Barbano MF, Cador M. Various aspects of feeding behavior can be partially dissociated in the rat by the incentive properties of food and the physiological state.see comment. Behav Neurosci 2005;119:1244–53.

[32] Davis C, Strachan S, Berkson M. Sensitivity to reward: implications for overeating and overweight. Appetite 2004;42:131–8.

[33] Wang GJ, Volkow ND, Logan J, et al. Brain dopamine and obesity see comment. Lancet 2001;357:354–7.

[34] Lowe MR. The effects of dieting on eating behavior: A three-factor model. Psychol Bull 1993;114:100–21.

[35] Herman CP, Polivy J. Anxiety, restraint, and eating behavior. J Abnorm Psychol 1975;84:66–72.

[36] Herman CP, Polivy J. Restrained eating. In: Stunkard AJ, editor. Obesity. Philadelphia: Saunders; 1980. p. 208–25.

[37] Polivy J, Herman CP. Diagnosis and treatment of normal eating. J Consult Clin Psychol 1987;55:635–44.

[38] Herman CP, Polivy J. A boundary model for the regulation of eating. In: Stunkard AJ, Stellar E, editors. Eating and its disorders. New York: Raven Press; 1984. p. 141–56.

[39] Heatherton TF, Herman CP, Polivy J, King GA, McGree ST. The (mis) measurement of restraint: an analysis of conceptual and psychometric issues. J Abnorm Psychol 1988;97:19–28.

[40] Presnell K, Stice E. An experimental test of the effect of weight-loss dieting on bulimic pathology: tipping the scales in a different direction. J Abnorm Psychol 2003;112:166–70.

[41] Stice E, Fisher M, Lowe MR. Are dietary restraint scales valid measures of dietary restriction? unobtrusive observational data suggest not. Psychol Assess 2004;16:51–9.

[42] Klesges RC, Isbell TR, Klesges LM. Relationship between dietary restraint, energy intake, physical activity, and body weight: a prospective analysis. J Abnorm Psychol 1992;101:668–74.

[43] French SA, Jeffery RW, Forster JL, McGovern PG, Kelder SH, Baxter JE. Predictors of weight change over two years among a population of working adults: the healthy worker project. Int J Obes Relat Metab Disord 1994;18:145–54.

[44] Stice E, Cameron RP, Killen JD, Hayward C, Taylor CB. Naturalistic weight-reduction efforts prospectively predict growth in relative weight and onset of obesity among adolescent females. J Consult Clin Psychol 1999;67:967–74.

[45] Lowe MR, Annunziato RA, Markowitz JT, et al. Multiple types of dieting prospectively predict weight gain during the freshman year of college. Appetite 2006;47:83–90.

[46] Lowe MR, Maycock B. Restraint, disinhibition, hunger and negative affect eating. Addict Behav 1988;13:369–77.

[47] Ouwens MA, van Strien T, van der Staak CP. Tendency toward overeating and restraint as predictors of food consumption. Appetite 2003;40:291–8.

[48] Westenhoefer J, Broeckmann P, Munch AK, Pudel V. Cognitive control of eating behaviour and the disinhibition effect. Appetite 1994;23:27–41.

[49] Timmerman GM, Gregg EK. Dieting, perceived deprivation, and preoccupation with food. West J Nurs Res 2003;25:405–18.

[50] Forman EF, Hoffman KL, McGrath K, Herbert JH, Brandsma L, Lowe MR. A Comparison of Acceptance- and Control-based Strategies for Coping with Food Cravings: an Analog Study. Behav Res Ther in press.

[51] Herman CP, Polivy J. Normative influences on food intake. Physiol Behav 2005;86:762–72.

[52] Rozin P, Fischler C, Shields C, Masson E. Attitudes towards large numbers of choices in the food domain: a cross-cultural study of five countries in Europe and the USA. Appetite 2006;46:304–8.

[53] Teegarden SL, Bale TL. Decrease in dietary preferences produce increased emotionality and risk for dietary relapse. Biol Psychiatry 2007;61:1021–9.