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Food reinforcement and obesity: Psychological Moderators

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Abstract

The relative reinforcing value of food (RRV_{food}) provides an index of the motivation to eat. Research has shown that RRV_{food} is higher in obese individuals than their non-obese peers, is associated with greater energy intake, predicts weight gain and interacts with impulsivity to predict energy intake. This study was designed to determine whether dietary restraint, dietary disinhibition or hunger moderate the effect of RRV_{food} on the weight status and energy intake in 273 adults of various body mass index (BMI) levels. Hierarchical regression was used to assess the independent effects of RRV_{food} on BMI and energy intake, controlling for age, sex, income, education, minority status and RRV_{reading} . Results showed that greater RRV_{food} , but not RRV_{reading} , was associated with greater BMI and energy intake. Dietary disinhibition and dietary restraint moderated the relationship between RRV_{food} and BMI, with dietary disinhibition being a stronger moderator of this relationship ($r^2 = 0.20$) than dietary restraint ($r^2 = 0.095$). In addition, dietary disinhibition moderated the effect of RRV_{food} on energy intake. These results replicate the importance of RRV_{food} as a predictor of obesity, and show that psychological factors moderate the effect of food reinforcement on body weight and energy intake. This trial was registered at clinicaltrials.gov as NCT00962117.

Keywords

reinforcement; obesity; disinhibition; restraint

Food is a powerful motivator of behavior, and individual differences in the reinforcing value of food may help explain the excess energy intake responsible for obesity (Epstein, Leddy, Temple, & Faith, 2007). Obese individuals find food more reinforcing than lean individuals (Epstein, Temple et al., 2007; Saelens & Epstein, 1996; Temple, Legierski, Giacomelli, Salvy, & Epstein, 2008), and food reinforcement is related to energy intake in laboratory ad libitum eating tasks (Epstein, Temple et al., 2007; Epstein et al., 2004).

While food reinforcement is an important determinant of eating, it may interact with other psychological variables to influence intake. For example, based on similar models in drug

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abuse, Appelhans has presented a provocative theory (Appelhans, 2009) that high food reinforcement in combination with high impulsivity as assessed by delay discounting predicts eating. Recent research from our laboratory has supported this hypothesis (Rollins, Dearing, & Epstein, 2010), but there may be other psychological constructs that moderate the effect of food reinforcement on obesity. Impulsivity assessed by delay discounting is related to dietary disinhibition, with disinhibited eaters also being more impulsive (Yeomans, Leitch, & Mobini, 2008), so it is possible that food reinforcement interacts with dietary disinhibition to influence obesity. A consistent body of research has shown that dietary disinhibition is related to BMI (Dykes, Brunner, Martikainen, & Wardle, 2004; Lawson et al., 1995; Lindroos et al., 1997; Provencher, Drapeau, Tremblay, Despres, & Lemieux, 2003; Williamson et al., 1995), and disinhibition is more reliably related to BMI than dietary restraint (Bryant, King, & Blundell, 2008), though research does suggest that disinhibition and restraint may interact to influence obesity (Bryant et al., 2008; Williamson et al., 1995). In addition, restrained individuals find food more reinforcing than non-restrained individuals (Giesen, Havermans, Nederkoorn, Strafaci, & Jansen, 2009), and BMI has been shown to moderate the relationship between dietary restraint and the reinforcing value of food (Goldfield & Lumb, 2009). Thus, the current paper examined whether dietary restraint and/or disinhibition moderate the relationships amongst the reinforcing value of food, BMI and energy intake in obese as well as non-obese men and women.

Methods

Participants

The participants were 273 adult subjects (79 non-obese females, 72 non-obese males, 60 obese females, 62 obese males) who participated in a study of genetic factors associated with food reinforcement. Participants were recruited from an existing family database, newspaper ads, flyers posted around the University at Buffalo campuses and in community settings, web-based recruitment (e.g. ads on Craigslist and on the departmental website) and direct mailings targeted to community residents between the ages of 18-50. Participants were not eligible for the study if they were taking medications associated with loss of appetite, were smokers, had diabetes, had a current or past history of an eating disorder or a psychiatric disorder (e.g. anxiety, depression, attention-deficit/hyperactivity disorder), were allergic to the ingredients in the study foods, were currently dieting, were pregnant or did not rate at least a moderate liking (<4 on a 9 point Likert type scale) for five out of the six study foods. Participants received a \$50 gift certificate to local stores for completing the study. Recruitment began in January 2008 and continued throughout June 2010. The study was approved by the University at Buffalo Health Sciences Institutional Review Board. Participant characteristics are shown in Table 1.

Procedures

Participants visited the laboratory for two sessions, the ad libitum snack-eating task and the food reinforcement task. Both experimental sessions were scheduled between the hours of 2PM and 5PM, during a normal period that individuals would consume additional calories outside of meal time (Popkin & Duffey, 2010). Participants were asked to refrain from consuming food or drinking beverages other than water for at least 3 hours and to not consume the experimental foods for at least 24 hours prior to the test session. Upon initial arrival at the laboratory, participants read and signed consent forms, completed a same-day and 24-hour food recall, and completed hunger questionnaires. Participants were provided a choice of two isocaloric energy bar preloads (Clif Bar & Company; Berkeley, CA, 42g, 150kcal, 4g fat, 23g carbohydrates, 7g protein) after the collection of the baseline hunger questionnaire measure to minimize the effects of hunger on energy intake and food reinforcement. The inclusion of a standard preload increases the ability to show individual

differences in food reinforcement (Reiss & Haverkamp, 1996). Demographic information, height and weight measurements and three dietary habits questionnaires were administered at the end of the ad libitum eating session.

Ad Libitum eating task—The ad libitum food consumption task was presented as a taste test. Participants were provided 210-305 kcal (42 – 60 g) servings of six palatable, high-energy-dense snack foods (amount of food presented (g) and energy density (kcal/g) shown in parentheses): Wavy Lay's Potato Chips (57 g, 5.4); Cooler Ranch Doritos (56 g, 5.4); M&M's (60 g, 5.0); Twix (48 g, 5.0); Kit Kat (42 g, 5.0); and Butterfinger (57g, 4.5). Water was provided ad libitum. Participants were told that they could consume as much or as little of the food that they wanted as long as they tasted each food so that they could accurately rate the food's characteristics. Participants rated each food on a number of different characteristics including pleurability, sweetness, blandness, flavorfulness and bitterness using 9-point Likert-type scales. Participants were then given the Three Factor Eating Questionnaire (Stunkard & Messick, 1985) to complete. Food from the taste test was left in the room and participants were told that the food would be discarded after the session and they could continue eating if they choose to do so. When participants indicated that they were finished, they were asked to identify their favorite food from among the six available and told that this was the food that would be used in the food reinforcement test session. Once the questionnaires were completed, participants' height and weight measurements were taken, and they were given a reminder for the food reinforcement visit. This visit was scheduled 2-3 weeks after the ad libitum eating session.

Food reinforcement task— RRV_{food} was measured by determining the number of responses participants made for food or food alternatives on progressive ratio schedules of reinforcement. The experimental environment included two computer stations that participants could go back and forth between. At one station, participants could earn points toward food and at the other station they could earn points for time to spend reading *Time* and *Newsweek* magazines. This alternative activity was provided to reduce the likelihood that participants would engage in responding out of boredom. Participants were instructed on how to use the computer task and given a practice session. Following the instructions for the task, the experimenter left the room. An intercom and a closed-circuit video system were present in the room so the experimenter could observe the participant and the participant could communicate with the experimenter.

The reinforcement task is similar to a slot machine with shapes that rotate on the screen and a point is earned each time the three shapes match in shape and color. For every five points earned, the subject received a 70-101 kcal (14 - 20 g) portion of his or her preferred snack food selected during the ad libitum eating session or 2 minutes of reading time, depending on which reward they were working for. The programmed reinforcement schedules for food and reading were progressive fixed-ratio schedules with response requirements of 4, 8, 16, 32, 64, 128, 256, 512 and so forth for each point. Participants were instructed to perform one activity at a time (i.e. play the computer game, eat or read), and that the session would end when they no longer wished to earn points for access to food or time to spend reading. Water was provided ad libitum.

The dependent measure that we used was the breakpoint, or P_{max} (Bickel, Marsch, & Carroll, 2000), which is the last schedule (i.e. 4, 8, 16 32, etc.) at which subjects met response requirements for access to the food or non food alternative. The test-retest reliability of RRV_{food} has been demonstrated (Epstein, Temple et al., 2007), and RRV_{food} has been positively associated with energy intake in the ad libitum eating task (Epstein, Temple et al., 2007; Epstein et al., 2004) and with weight status (Epstein, Temple et al., 2007; Saelens & Epstein, 1996; Temple et al., 2008).

Measurement

Dietary recalls—At the beginning of each session, participants were interviewed by the experimenter, using a multi-pass same-day and 24-hour food recall, to verify adherence to the study protocol.

Height and weight—The participant's weight and height were measured using a digital scale (TANITA Corporation of America Inc, Arlington Heights, IL) and a digital stadiometer (Measurement Concepts & Quick Medical, North Bend, WA). On the basis of height and weight data, body mass index (BMI) was calculated according to the following formula: $BMI = kg/m^2$. Individuals were considered obese if their BMI was at least $30 kg/m^2$ and non-obese if their BMI was less than $30 kg/m^2$ (NHLBI Obesity Education Initiative Expert Panel, 1998).

Dietary restraint and disinhibition—The Three Factor Eating Questionnaire (TFEQ) (Stunkard & Messick, 1985) contains 21 items assessing dietary restraint (the tendency to control food intake in order to maintain body image, e.g. “I deliberately take small helpings as a means of controlling my weight”) and 16 items assessing dietary disinhibition (loss of control over eating behavior, e.g. “Sometimes, I start eating and just cannot seem to stop.”), each with a True/False or multiple-choice response format. Items were scored according to published guidelines (Stunkard & Messick, 1985).

Screening for eating disorders—Participants were excluded if they had a current or past history of an eating disorder. This was determined in two stages. First, participants completed the Questionnaire of Eating and Weight Patterns (QEWP) (Spitzer et al., 1992) and the Binge Eating Scale (BES) (Gormally, Black, Daston, & Rardin, 1982). The QEWP and BES are used to assess binge-eating disorder. Participants were identified as potentially having binge-eating disorder if they scored higher than 27 on the BES or were indicated as having the disorder by the QEWP. Since diagnoses cannot be made on the basis of questionnaire scores, in a second stage, any participant identified as potentially having binge eating disorder was required to complete the Eating Disorders Examination (Cooper & Fairburn, 1987), administered by trained personnel in an additional session. No participants in this sample were excluded for eating disorders.

Food liking and hunger—Subjective ratings of hunger and food hedonics were collected before and after consumption of the preload and after both test sessions using Likert-type scales. For hunger, 1 indicated not hungry at all/not full at all and 10 indicated extremely hungry/extremely full. For hedonics, 1 indicated not liking at all and 9 indicated liking very much.

Analytic plan

Sample characteristics were analyzed using analysis of variance (ANOVA) for continuous variables and Chi-square for categorical variables. The goal of the primary analyses were to establish the independent effects of RRV_{food} , $RRV_{reading}$, and TFEQ measured dietary restraint, disinhibition and hunger on BMI and laboratory energy intake, as well as the interaction of dietary restraint, disinhibition and hunger with RRV_{food} on BMI and energy intake using hierarchical linear regression analyses. Since responding for food occurred in the presence of an alternative (reading), $RRV_{reading}$ was controlled for in all analyses. Age, sex, minority status, educational level and $RRV_{reading}$ were included as covariates in the base model, and the effects of adding additional variables was established by determining whether the addition of the variables was associated with a significant increase in variance accounted for using an F_{inc} test. Specifically, we investigated whether the interaction of dietary disinhibition or dietary restraint with RRV_{food} accounted for more variance in BMI

and energy intake than that predicted by RRV_{food} alone. The data were entered and analyzed using SYSTAT 11 (Systat Software, 2004).

Results

Characteristics of the sample are presented in Table 1. The base model including age, sex, minority status, educational level, and RRV_{reading} accounted for 4.4% of the variance ($F(5,267) = 2.45$, $p = 0.034$) in BMI. Adding RRV_{food} to the base model increased r^2 to 0.06 ($F_{\text{inc}}(1,266) = 4.58$, $p = 0.033$). To graph the relationships between RRV_{food} , dietary disinhibition and BMI, values that maximized significant differences in BMI and sample size breakdown were assessed using one-way ANOVA. Results show differences at a progressive ratio schedule of FR32 ($p < 0.001$), as shown in Figure 1A. Similarly, adding dietary disinhibition to the base model increased $r^2 = 0.181$, $F_{\text{inc}}(1, 266) = 44.45$, $p < 0.0001$, with greater disinhibition associated with greater BMI ($\beta = 0.82$, $p < 0.0001$). Differences in BMI for those who scored at least 3 or greater on dietary disinhibition were observed, but to create a large enough sample of participants with high disinhibition scores, a value of 6 was used, with results for participants above and below 6 shown ($p < 0.0001$) in Figure 1B. The interaction of dietary disinhibition $\times$ RRV_{food} further increased the variance accounted to $r^2 = 0.20$, $F_{\text{inc}}(2, 264) = 23.73$, $p < 0.0001$, as shown in Figure 1C.

Adding dietary restraint to the base model did not significantly increase the variance accounted for ($F_{\text{inc}}(1,266) = 0.083$, $p = 0.77$). The interaction of $RRV_{\text{food}} \times$ dietary restraint increased the variance accounted for to $r^2 = 0.095$, $F_{\text{inc}}(2, 264) = 5.01$, $p = 0.0073$, but the variance accounted for by the interaction of $RRV_{\text{food}} \times$ dietary restraint was less than half that accounted for by the interaction of $RRV_{\text{food}} \times$ dietary disinhibition ($r^2 = 0.095$ vs. $r^2 = 0.20$). Sex did not interact with any variable to improve the prediction of BMI beyond knowledge of each predictor separately, and there was no interaction of dietary restraint $\times$ disinhibition, which has been observed in some studies (Bryant et al., 2008; Williamson et al., 1995).

The same base model was used to predict energy intake and accounted for $r^2 = 0.20$ of the variance ($F(5,267) = 13.76$, $p < 0.0001$). Adding RRV_{food} significantly increased r^2 to 0.28 ($F_{\text{inc}}(1,266) = 27.95$, $p < 0.0001$). To graph the relationships between RRV_{food} , dietary disinhibition and energy intake, values that maximized significant differences in BMI and sample size breakdown were assessed using one-way ANOVA. Again, results show differences at a progressive ratio schedule of FR32 ($p < 0.0001$), as shown in Figure 2A. Adding dietary disinhibition to the base model increased variance accounted for to $r^2 = 0.22$ ($F_{\text{inc}}(1, 266) = 5.71$, $p = 0.018$), as shown in Figure 2B. The interaction of dietary disinhibition $\times$ RRV_{food} further increased the variance accounted for to $r^2 = 0.30$, $F_{\text{inc}}(2, 264) = 3.52$, $p = 0.031$, as shown in Figure 2C. Adding dietary restraint to the model increased variance accounted for to $r^2 = 0.22$ ($F_{\text{inc}}(1, 266) = 3.97$, $p = 0.047$), but the interaction of dietary restraint $\times$ RRV_{food} did not account for significantly more variance. Sex interacted with RRV_{food} to predict eating, such that the effect of RRV_{food} on energy intake was more pronounced in males than in females ($r^2 = 0.30$, $F_{\text{inc}}(1, 265) = 7.25$, $p = 0.0075$). There was no interaction of dietary restraint $\times$ disinhibition ($F(2,264) = 2.22$, $p = 0.11$).

Discussion

This study replicates the observations that RRV_{food} is an independent predictor of BMI (Epstein, Temple et al., 2007; Saelens & Epstein, 1996; Temple et al., 2008) and energy intake (Epstein, Temple et al., 2007; Epstein et al., 2004). We also confirmed previous findings that dietary disinhibition is positively associated with BMI (Dykes et al., 2004;

Lawson et al., 1995; Lindroos et al., 1997; Provencher et al., 2003; Williamson et al., 1995) and food consumption (Bryant et al., 2008). Dietary restraint was not as strong a predictor of BMI as dietary disinhibition in this study, consistent with previous research (Williamson et al., 1995). To our knowledge, these results represent the first demonstration that food reinforcement interacts with dietary disinhibition to predict BMI and energy intake. Adults with high food reinforcement and high disinhibition scores were likely to have a higher BMI and to consume more calories during a laboratory ad libitum eating session. This finding is similar to the observation that food reinforcement interacts with impulsivity to influence energy intake in non-obese women (Rollins et al., 2010).

In a thorough review of the research on dietary disinhibition, Bryant, King and Blundell (Bryant et al., 2008) provide strong evidence that dietary disinhibition is related to obesity. They conceptualize dietary disinhibition in terms of a strong propensity to overeat in an obesogenic environment and argue that the descriptive terms “opportunistic eating”, or “readiness to eat” given the appropriate environment might be better labels than disinhibition for this factor on the TFEQ. More simply, dietary disinhibition may reflect an increased responsiveness to palatable food cues. Dietary disinhibition is also related to impulsivity (Yeomans et al., 2008), as those who are disinhibited are also impulsive.

Food deprivation (Epstein, Truesdale, Wojcik, Paluch, & Raynor, 2003; Raynor & Epstein, 2003) and palatable foods (Lappalainen & Epstein, 1990) can increase food reinforcement and energy intake, but to our knowledge, there is no research on how the environment or context cues can increase responding for food in those high in food reinforcement. Basic behavioral theory would argue that stimulus control for a food is developed after repeatedly eating a food in a specific context, and satiated subjects will eat after presentation of cues associated with food (Cornell, Rodin, & Weingarten, 1989; Weingarten, 1983). The concept of food craving increasing after exposure to cues associated with reward is central to elicitation of food cravings and wanting to eat (Berridge, 1996; Kelley & Berridge, 2002). This theoretical account suggests that the effect of food reinforcement on eating and obesity may depend on the environment as well as an individual's ability to delay gratification (Rollins et al., 2010).

In addition to the interaction of dietary disinhibition with food reinforcement to influence energy intake and BMI, we observed that dietary restraint moderated the effect of food reinforcement on BMI, such that restrained eaters who found food more reinforcing had greater BMI. While these findings are novel, other studies have investigated the relationships amongst dietary restraint, reinforcement processes and BMI. For example, dietary restraint may interact with BMI to predict food reinforcement (Goldfield & Lumb, 2009), and restrained non-dieters may find food more reinforcing than unrestrained non-dieters (Giesen et al., 2009). Consistent with these data, restrained eaters exhibit higher dopaminergic responsivity to food stimuli (Volkow et al., 2003) and are more reactive toward food cues (Brunstrom, Yates, & Witcomb, 2004; Lowe & Butryn, 2007). Maintaining dietary restraint in modern environments that are saturated with food cues may require substantial cognitive resources to inhibit prepotent responding (Hill & Peters, 1998; Hill, Wyatt, Reed, & Peters, 2003). Over time, restrained individuals who find food highly reinforcing and live in a toxic environment loaded with food cues may be more prone to lose control over their eating behavior, as executive processes have been demonstrated to be limited by cognitive resources and hence may fatigue (Persson, Welsh, Jonides, & Reuter-Lorenz, 2007). This interpretation would also explain how dietary restraint could be negatively related to energy intake but positively related to weight gain (Klesges, Isbell, & Klesges, 1992), and may account for the equivocal associations between dietary restraint and weight status in the literature (Konttinen, Haukkala, Sarlio-Lahteenkorva, Silventoinen, & Jousilahti, 2009). More research is needed to determine if dietary restraint leads to

increased food reinforcement and/or if increased food reinforcement is a predictor of unsuccessful dietary restraint.

The pattern of results suggests that BMI is related to food reinforcement moderated by disinhibition through increased energy intake. Research in children has shown that food reinforcement predicts weight gain (Hill, Saxton, Webber, Blundell, & Wardle, 2009), but this relationship should be systematically replicated in adults. In addition, based on the present results, a prospective design is needed to test whether those high in food reinforcement and high in disinhibition are at greatest risk for weight gain. Another limitation of this study was that energy intake was measured solely in the laboratory and that only snack foods were used to assess RRV_{food} . Whether this relationship could be generalized to the natural environment or to mealtime eating behavior remain open questions.

It would be interesting to evaluate the role of interventions that target disinhibition as a way to reduce the impact of food reinforcement on eating. Reductions in dietary disinhibition have been associated with success in behaviorally oriented weight control programs (Cuntz, Leibbrand, Ehrig, Shaw, & Fichter, 2001), but there is no research base to test different approaches to reducing dietary disinhibition. Given the relationship between disinhibition and impulsivity, it may be that methods to reduce impulsivity (Bickel, Yi, Landes, Hill, & Baxter, 2010) may also reduce disinhibition, and research is needed to test this hypothesis.

In summary, the cross-sectional relationship between BMI, high food reinforcement and disinhibition suggests that these factors may be important in the development of obesity. Research has shown that food reinforcement predicts weight gain in children (Hill et al., 2009), and that dietary disinhibition was correlated with weight gain in women (Hays & Roberts, 2008). Research is needed to test the predictive effect of high food reinforcement and high disinhibition phenotypes on weight gain in a prospective study.

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Highlights

- Food reinforcement, dietary restraint, disinhibition and hunger were assessed in 273 adults.
- Greater food reinforcement was associated with greater BMI
- Dietary disinhibition moderated the relationship between food reinforcement and BMI.
- Dietary disinhibition moderated the relationship between food reinforcement and energy intake.

Figure 1.

Hierarchical regression analyses showed RRV_{food} ($p = 0.028$), dietary disinhibition ($p < 0.0001$) and the interaction of $RRV_{food} \times$ dietary disinhibition ($p = 0.007$) predicted BMI. Figure 1 shows differences in BMI for those with a breakpoint of $\leq FR32$ or $>FR32$ (A), dietary disinhibition scores of <6 or ≥ 6 (B) and the interaction of $RRV_{food} \times$ dietary disinhibition (C).

Figure 2.

Hierarchical regression analyses showed RRV_{food} ($p < 0.0001$), dietary disinhibition ($p < 0.0001$) and the interaction of $RRV_{food} \times$ dietary disinhibition ($p = 0.029$) predicted energy intake. Figure 2 shows differences in energy intake for those with a breakpoint of $\leq FR32$ or $>FR32$ (A), dietary disinhibition scores of <6 or ≥ 6 (B) and the interaction of $RRV_{food} \times$ dietary disinhibition (C).

Table 1
Participant characteristics

	Overall	Lean	Obese	p
N	273	151	122	
Age	34.4 ± 10.7	33.3 ± 11.2	35.8 ± 9.9	NS
Body Mass Index	29.9 ± 7.4	24.6 ± 3.0	36.4 ± 5.9	<0.0001
Restraint	7.7 ± 4.7	7.6 ± 5.0	7.8 ± 4.3	NS
Disinhibition	6.3 ± 3.4	5.3 ± 3.1	7.6 ± 3.5	<0.0001
Hunger	5.2 ± 3.2	5.1 ± 3.3	5.5 ± 3.2	NS
Sex (M/F)	134/139	72/79	62/60	NS
Education				NS
Attended high school	6	3	3	
Completed high school	36	12	24	
Some college/vocational training	81	51	30	
Completed 2 year college	45	21	24	
Completed 4 year college	73	43	30	
Completed graduate/professional school	31	18	14	
Race/Ethnicity				NS
Caucasian	75	40	35	
Non-Caucasian	198	111	87	