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Women who are motivated to eat and discount the future are more obese

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Abstract

Objective—Food reinforcement and delay discounting (DD) predict Body Mass Index (BMI), but there is no research studying whether these variables interact to improve prediction of BMI.

Design and Methods—BMI, the relative reinforcing value of high ($PMAX_{HED}$) and low ($PMAX_{LED}$) energy dense food, and DD for $10 and $100 future rewards ($DD_{10}$, DD_{100}) were measured in 199 adult females.

Results—$PMAX_{HED}$ ($p = 0.017$), DD_{10} ($p = 0.003$) and DD_{100} ($p = 0.003$) were independent predictors of BMI. The interaction of $PMAX_{LED}$ X DD_{10} ($p = 0.033$) and DD_{100} ($p = 0.039$), and $PMAX_{HED}$ X DD_{10} ($p = 0.041$) and DD_{100} ($p = 0.045$) increased the variance accounted for predicting BMI beyond the base model controlling for age, education, minority status,

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Conflict of Interest
None of the authors have any conflict of interest.

disinhibition and dietary restraint. Based on the regression model, BMI differed by about 2 BMI units for low versus high food reinforcement, by about 3 BMI units for low versus high DD, and by about 4 BMI units for those high in $PMAX_{HED}$ but low in DD versus high in $PMAX_{HED}$ and high in DD.

Conclusions—Reducing DD may help prevent obesity and improve treatment of obesity in those who are high in food reinforcement.

Keywords

Obesity; food reinforcement; delay discounting; reinforcement pathology

Positive energy balance from overeating is one cause for obesity. A factor that influences the motivation to eat is food reinforcement, assessed by how hard someone will work for food when different types of food or food versus another commodity are concurrently available (1). Food reinforcement is cross-sectionally related to obesity (2–4) and prospectively related to weight gain (5, 6). Energy intake mediates the relationship between food reinforcement and Body Mass Index (BMI) (7). High food reinforcement in the absence of strong alternative reinforcers predicts poor weight loss in children participating in behavioral treatment (8).

Decision making involves not only concurrent choices assessed in food reinforcement, but also intertemporal choices, such as the choice about whether to choose a small immediate reward or a larger, delayed reward. This type of choice is thought of as delay of gratification or delay discounting, which has also been cross-sectionally related to obesity (9–11) and prospectively related to weight gain (12, 13). The ability to delay gratification is also a predictor of child weight loss in structured treatment programs (8).

High food reinforcement and the inability to delay gratification interact to influence eating (14) as well as weight loss (8), such that those who are high in food reinforcement and cannot delay gratification consume more calories and lose less weight than those who are high in food reinforcement but can delay gratification. The balance between the motivation to consume food and the inability to delay gratification is called reinforcement pathology (15, 16). One useful analogy to put this balance into perspective of everyday behaviors is to relate reinforcement pathology to driving a car, with the motivation to eat represented by the accelerator, and self-control represented by the brake. People with reinforcement pathology have too much gas to stimulate eating, and inadequate brakes when they try to stop or prevent eating. These two factors represent different aspects of brain activation, with reinforcement processes related to mesolimbic and mesocortical activation, while delay of gratification is related to frontal activation, and is considered to be part of executive function (17, 18). One important hypothesis that has not yet been tested is the relationship between reinforcement pathology and BMI. Based on the existing research, it is expected that food reinforcement and delay discounting would be related to BMI, with highest BMI for those with reinforcement pathology, who are high in food reinforcement and unable to delay gratification.

Methods

Participants

Participants were 199 females participants in an experimental online supermarket recruited from an existing family database, flyers posted around the University at Buffalo campuses and in the community, web based recruitment (e.g ads on Craig's list and on the department's website) and targeted direct mailings. Inclusion criteria included: 19 years of age or older and the primary grocery shopper for a household containing at least one child between the ages of 2 and 18; could purchase the majority of their groceries once a week; no dietary restrictions that could interfere with the experiments, including food allergies or religious or ethnic practices that limit food choice; medical conditions that could alter nutritional status or intestinal absorption (eg, inflammatory bowel disease); not currently pregnant; at least a moderate liking (≥3 on a 5-point Likert-type scale) of the food used in the study and willingness to consume the food; and no psychopathology or developmental disabilities (e.g. attention deficit hyperactivity disorder) that would limit participation. Participants were compensated $290, minus the cost of one week's worth of groceries they selected in the online grocery store, which they received at study completion. The study was approved by the University at Buffalo Social and Behavioral Sciences Institutional Review Board. Participant characteristics are shown in Table 1.

Procedures

Participants were studied across six weekly sessions. Prior to the first session, participants completed a basic demographics form and Three Factor Eating Questionnaire (TFEQ) (19, 20). Participants were asked to refrain from eating or drinking, other than water, for two hours prior to each appointment. Upon arrival to the laboratory, participants read and signed consent forms and a study agreement. Next, they completed a same-day food recall and hunger scale, and were then given a preload of a Nature Valley Oats 'n Honey or Nature Valley Oats 'n Dark Chocolate granola bar (General Mills; Minneapolis, MN, 42g, 190kcal, 7g fat, 29g carbohydrates, 4g protein). The amount given was based on their self-reported body weight during screening and was equivalent to approximately 2kcal for every kilogram of body weight. The inclusion of a preload is designed to assess hedonic rather than homeostatic hunger. During the rest of session one, participants completed response inhibition (data not reported), food reinforcement, and delay discounting tasks. Over the next five sessions, subjects selected their weekly household groceries in the online supermarket that included nearly 6,000 food items under varying price conditions to determine the influence of subsidizing healthy foods or taxing less healthy foods. The economic analyses from the shopping sessions will be reported elsewhere. After the completion of the purchasing sessions, participant's height and weight were taken and they were debriefed and compensated, which included receiving one week's worth of groceries they selected in the online grocery store.

Measures

Food reinforcement—The relative reinforcing value of low energy-dense ($PMAX_{LED}$) and high energy-dense ($PMAX_{HED}$) foods were assessed using a computer program where subjects responded by pressing a mouse button (3, 21). Participants earned a point by

meeting schedule requirements. The programmed reinforcement schedules for both types of food were progressive fixed ratio schedules with response requirements of 4, 8, 16, 32, 64, 128 … 2048 and so forth for each point. For every five points earned, they received a 30g portion of their preferred low (LED: ED ≤ 2.0) or high (HED: ED >2.0) energy dense snack food (LED foods, ED: carrots and dip, 0.76; celery and dip, 0.60; apple slices, 0.57; bananas, 0.86; applesauce, 0.41; low-fat strawberry yogurt, 1.00; mandarin oranges, 0.37; pineapple chunks; 0.60; HED foods, ED: Chips Ahoy!® Cookies, 4.80; Little Debbie® Zebra Cakes, 4.48; Reese's Peanut Butter Cups®, 5.41; M&M's®, 5.00; Hershey's® milk chocolate, 5.23; Mini Oreos®, 4.59; Doritos® Nacho Cheese, 5.39; and Pringles® Original), 4.95. The task was set up on two computer stations, one for LED and one for HED, so that participants could freely move between stations to earn as many portions as they wanted. Participants were instructed to perform one activity at a time (i.e. play the computer game or eat), and the session would end when they no longer wished to earn points toward portions of either type of food. Food portions were brought to the participant after they were earned and could be eaten right away or later during the task, however, the food could not be eaten once the task ended. Water was provided ad libitum.

The primary dependent measure was the relative reinforcing value for LED ($PMAX_{LED}$) or HED ($PMAX_{HED}$) foods operationalized as P_{max}, or the maximal reinforcement schedule that the participant completed for LED and HED foods. The relative reinforcing value of HED versus LED food (RRVHED) was established by the formula: $PMAX_{HED}/(PMAX_{HED} + PMAX_{LED})$.

Delay Discounting (DD) task—The DD task measures the degree to which people discount a hypothetical reward of $10 ($DD_{10}$) and $100 ($DD_{100}$) with an increasing time delay (22). In the discounting task, two sets of cards were placed on the table in front of the participant; one stack showed a dollar amount available now, and the other stack showed a dollar amount available after a time delay. For each trial, the participant was asked to choose between the two monetary amounts, and the "available now" cards were shifted so that the value of the immediate outcome changed after each trial. Participants experienced the task using both $100 and $10 as the hypothetical reward with the presentation of the hypothetical rewards counterbalanced. Values for the monetary reward consisted of 26 values ranging from 0.1 to 100% of the delayed reward to which they were compared. For each time delay (1 day, 2 days, 1 week, 2 weeks, 1 month, 6 months and 2 years), the immediate reward cards were titrated down then up for half of the subjects, and up then down for the other half of the subjects, the order of which was counterbalanced. Indifference points for each of the values of the delayed reward at each of the time delays were computed. Indifference points were determined for each time delay as the average of two determinations: the last immediate reward chosen when the participant switched preference from the immediate to the delayed reward when the immediate rewards were presented in descending order, and the first immediate reward value chosen when the participant switched preference from the delayed reward to the immediate reward when the immediate reward values were presented in ascending order. Area under the curve values for $10 rewards ($AUC_{10}$) and AUC for $100 rewards ($AUC_{100}$) were calculated from the indifference points for each delay (23). AUC values range from 0 (highest possible discounting) to 1 (no discounting).

Hunger and food liking—Subjective ratings of hunger were collected before consumption of the preload and before and after the RRV task using a 100 mm visual analogue scale anchored by not hungry (0) to extremely hungry (100). Food hedonics were measured before and after the RRV task using a 5-point Likert-type scale ranging from do not like at all (1) to like very much (5). Hunger refers to the hunger scores following consumption of the preload during session one.

Three Factor Eating Questionnaire (TFEQ)—The TFEQ is a validated instrument with subscales that assess dietary restraint, hunger and disinhibition (19, 20).

Anthropomorphic measurements—Height was measured with a digital stadiometer (Measurement Concepts & Quick Medical, North Bend, WA) and weight was assessed using a Tanita digital scale (Arlington Heights, IL). BMI was calculated using the formula $BMI=kg/m^2$.

Years of education—Years of highest parental education was used as an indicator of socioeconomic status because it may shape occupational status and income (24), and is more stable than occupational status or income beyond early adulthood (25).

Analytic plan

The analytic plan was designed to assess the interactive effects of low and high energy density food reinforcement and delay discounting of small and moderate rewards on BMI. First, zero order correlations were used to establish the relationships among the variables. Next, hierarchical linear regression was used, with the first step including different measures of food reinforcement and delay discounting as predictors of BMI, and the next step adding the interaction of food reinforcement and delay discounting on BMI. These models were run controlling for age, education, minority status and dietary disinhibition and dietary restraint. These variables were controlled since age (26) and education (27) can moderate the influence of delay discounting, and education (4), and dietary disinhibition and dietary restraint (28) can moderate the effect of food reinforcement on BMI. Minority status was included as a control variable since it was an independent predictor of BMI in this sample. Dietary hunger was also an independent predictor of BMI, but it was strongly related to dietary disinhibition ($r = 0.61$, $p < 0.001$), and both variables were not included in the model to reduce collinearity. The final set of models were to assess whether age, education, minority status, dietary disinhibition or restraint moderated the effects of reinforcement pathology on BMI. The effect of adding each additional variable was evaluated by determining whether it significantly increased variance accounted for using a F_{inc} test. The data were entered and analyzed using SYSTAT 11 (29).

Results

The zero order correlations showed that the RRVHED ($r = 0.197$, $p = 0.005$), $PMAX_{HED}$ ($r = 0.168$, $p = 0.017$), DD_{10} ($r = -0.206$, $p = 0.004$), DD_{100} ($r = -0.208$, $p = 0.003$), years of education ($r = -0.256$, $p < 0.001$), minority status ($r = 0.182$, $p = 0.01$) and the TFEQ disinhibition ($r = 0.366$, $p < 0.001$) and hunger scores ($r = 0.140$, $p = 0.049$) were correlated

with BMI. $PMAX_{LED}$ was not independently related to BMI (r = −0.042, p = 0.55). Years of education were related to RRV_{HED} (r = −0.164, p = 0.021) and DD_{10} (r = 0.184, p = 0.009).

Hierarchical regression models showed that adding the interaction of $PMAX_{HED}$ x DD_{10} (p = 0.034) increased variance accounted for predicting BMI from $r^2 = 0.285$ to $r^2 = 0.301$, ($F_{inc}(1,190) = 4.35$, p = 0.031). Similarly, the interaction of $PMAX_{HED}$ x DD_{100} (p = 0.05) increased r^2 accounted for from $r^2 = 0.284$ to $r^2 = 0.299$, $F_{inc}(1,190) = 4.07$, p = 0.045) (Figure 1). A similar pattern was observed for LED foods, as the interaction of $PMAX_{LED}$ x DD_{10} (p = 0.03) increased variance accounted for from to $r^2 = 0.285$ to $r^2 = 0.302$, ($F_{inc}(1,190) = 4.63$, p = 0.033). Finally, adding the interaction of $PMAX_{LED}$ x DD_{100} (p = 0.004) increased variance accounted for from $r^2 = 0.283$ to $r^2 = 0.299$, $F_{inc}(1,190) = 4.34$, p = 0.039 (Figure 2).

No significant improvement in variance accounted for was observed for the interaction of RRV_{HED} x DD_{10} or RRV_{HED} x DD_{100}. Moderator analyses did not show that age, education, minority status, TFEQ disinhibition or restraint moderated the interaction of $PMAX_{LED}$, $PMAX_{HED}$ or RRV_{HED} by DD_{10} or DD_{100} to predict BMI.

Discussion

We have shown for the first time that the balance between the motivation to eat and ability to delay gratification for both low and high energy dense foods predicts BMI. The pattern of the interaction between the reinforcing value of high-energy-dense foods and delay discounting is as predicted, with minimal effects of delay discounting when food reinforcement is low, but marked increases in BMI when food reinforcement is high. The lowest BMI is for women who find low energy dense foods reinforcing and who are able to delay gratification. Thus, those who find low energy dense foods reinforcing also benefit from the ability to delay gratification in regards to a lower BMI. The reinforcing value of low energy dense foods is rarely considered as a factor influencing weight, but it makes sense that being motivated to eat these foods may be a positive factor that influences lower weight. In the present study eating low energy dense foods may substitutes for eating higher energy dense foods.

The reinforcing value paradigm compared responding for concurrently available low and high energy dense foods, as opposed to high energy dense foods versus alternatives to food which is an alternative approach to studying food reinforcement (1). Comparing two types of food may not provide as large a contrast between reinforcers as food versus non-food alternatives, but both the proportion of responding for high versus low energy dense foods and responding for high energy dense foods predicted BMI, and responding for low and high energy dense foods interacted with delay discounting to predict BMI. Since the reinforcing value of any commodity depends on the alternatives available (1), differences between the characteristics of a food versus an alternative to eating are likely to be more dissimilar than if two foods are compared. Obese children find food more reinforcing than lean children, who find some alternatives to food more reinforcing than food (30), leading to the hypothesis that some obese people may find food more reinforcing than other activities due to lack of access to more reinforcing alternatives. This is consistent with the observation that

obese children lose less weight if they find food highly reinforcing and have decreased access to alternative reinforcers (8).

Knowledge of reinforcement pathology explains from 1.5 to 1.7 percent of the additional variance in BMI relative to base models, which is modest but may be important due to the multiple factors that influence obesity. Results suggest BMI is 2 units higher if participants were high in food reinforcement than if they were low in food reinforcement (28.80 vs 26.80, ± 1 SD based on the regression model). The effect of discounting the future is greater, as those high versus low in discounting the future have a difference of 3.01 BMI units (29.14 vs 26.13) for discounting $10 and 3.03 BMI units (29.15 vs 26.12) for discounting $100, suggesting that reducing future discounting would have a bigger impact on BMI than modifying food reinforcement. The biggest effects on BMI may come from modifying delay discounting in those who are high in food reinforcement. Previous research on reinforcement pathology and obesity has shown that if food reinforcement is high, then effects on eating (14) or weight loss (8) can be muted by the ability to delay gratification. The results of this study are consistent with these studies, as BMI was not elevated if food reinforcement was low, or if reinforcement for high energy density foods was elevated but the person did not discount the future. If people were high in food reinforcement and low versus high in discounting the future, they had a difference of 4.00 BMI units (26.17 vs 30.17 for discounting $10) or 4.08 (30.15 vs 26.07 for discounting $100) BMI units. The reduction in BMI when delay discounting is considered independently or interacting with food reinforcement suggests that modifying delay discounting represents a target for weight control.

One approach to reducing delay discounting is to improve working memory. Working memory is related to discounting the future (31), and the better the working memory capacity the greater the opportunity for the person to hold information about current options and future opportunities (32). Reduced working memory capacity may relegate the person to focusing only on the current alternative, discounting the future (32). To support this hypothesis, research has shown that working memory training reduces delay discounting in stimulant addicts (31) and alcohol consumption in social drinkers (33). Working memory training has been used to complement obesity treatment to enhance short term treatment effects (34).

An alternative approach to reduce discounting of the future is to take advantage of prospection, or the ability to pre-experience the future (35). Since delay discounting is characterized by a preference for smaller immediate reward over larger delayed rewards, than the ability to consider the future may influence how individuals make these choices. Prospection allows the person to put themselves in the future (episodic future thinking) as they are considering whether to choose a small but very palatable immediate reward or a larger delayed reward. Episodic future thinking can reduce delay discounting in lean and overweight/obese women (36) and energy intake in overweight/obese women (37). Research is needed that incorporates episodic future thinking into obesity treatment programs to assess effects of episodic future thinking on delay discounting and weight control.

Food reinforcement pathology is not moderated by age, education, minority status, dietary disinhibition or dietary restraint. The absence of moderators suggests that the effect of reinforcement pathology on BMI is evident across all types of women. This is particularly relevant since education, dietary disinhibition and dietary restraint have been shown to moderate the effects of either food reinforcement or delay discounting on BMI. It is important to determine if these results can be extended from cross-sectional relationships to prospective relationships, such that women high in food reinforcement and low in the ability to delay gratification may be at risk for weight gain and obesity independent of their age, educational levels, minority status, or their degree of dietary disinhibition or dietary restraint.

These results add to the research on the importance of reinforcement pathology for eating, obesity and weight loss. Research has shown the inability to delay gratification is associated with obesity in males and females (11), but other studies suggest this relationship is greater in females than males (10). On the other hand, men may be more susceptible to the effects of food reinforcement on eating (38). Research is needed including both sexes to determine whether reinforcement pathology predicts BMI in both men and women.

This research needs to be extended to children. Research has shown that food reinforcement (6) and the inability to delay gratification (12, 13) are independent predictors of weight gain in children. No research has assessed whether reinforcement pathology in young children is a better predictor of weight gain than either of these risk factors separately. This warrants special consideration since children may be at greater risk for reinforcement pathology since their prefrontal cortex is not fully developed so they may not have the ability to delay gratification associated with strong food reinforcement (39, 40). Based on these developmental differences, it may be important to provide developmentally appropriate self-control strategies/training as children's self-control and prefrontal cortex matures. Finally, the additional variance accounted for by knowledge of reinforcement pathology ranges from .15

Reinforcement pathology represents the balance between motivation to eat and the ability to delay gratification of food. Using the analogy of driving, people with reinforcement pathology have a lead foot on the gas pedal to eating, and inadequate brakes when they try to stop. Research to improve this balance is needed to prevent obesity among those at risk, as well as facilitate weight loss among those whose reinforcement pathology has led to obesity.

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References

1. Epstein LH, Leddy JJ, Temple JL, Faith MS. Food reinforcement and eating: A multilevel analysis. Psychological bulletin. 2007; 133:884–906. [PubMed: 17723034]
2. Giesen JC, Havermans RC, Douven A, Tekelenburg M, Jansen A. Will work for snack food: the association of BMI and snack reinforcement. Obesity (Silver Spring). 2010; 18:966–70. [PubMed: 20150901]
3. Epstein LH, Temple JL, Neaderhiser BJ, Salis RJ, Erbe RW, Leddy JJ. Food reinforcement, the dopamine D2 receptor genotype, and energy intake in obese and nonobese humans. Behav Neurosci. 2007; 121:877–86. [PubMed: 17907820]
4. Lin H, Fletcher KD, Epstein LH. High food reinforcement mediates the relationship between socioeconomic status and obesity. Obesity (Silver Spring). In press.
5. Carr KA, Epstein LH. Food reinforcement predicts weight gain in non-obese adults. Obesity (Silver Spring). In press.
6. Hill C, Saxton J, Webber L, Blundell J, Wardle J. The relative reinforcing value of food predicts weight gain in a longitudinal study of 7--10-y-old children. Am J Clin Nutr. 2009; 90:276–81. [PubMed: 19535428]
7. Epstein LH, Carr KA, Lin H, Fletcher KD, Roemmich JN. Usual energy intake mediates the relationship between food reinforcement and BMI. Obesity. 2012; 20:1815–9. [PubMed: 22245983]
8. Best JR, Theim KR, Gredysa DM, et al. Behavioral economic predictors of overweight children's weight loss. J Consult Clin Psychol. 2012; 80:1086–96. [PubMed: 22924332]
9. Davis C, Patte K, Curtis C, Reid C. Immediate pleasures and future consequences. A neuropsychological study of binge eating and obesity. Appetite. 2010; 54:208–13. [PubMed: 19896515]
10. Weller RE, Cook EW 3rd, Avsar KB, Cox JE. Obese women show greater delay discounting than healthy-weight women. Appetite. 2008; 51:563–9. [PubMed: 18513828]
11. Rasmussen EB, Lawyer SR, Reilly W. Percent body fat is related to delay and probability discounting for food in humans. Behav Processes. 2010; 83:23–30. [PubMed: 19744547]
12. Francis LA, Susman EJ. Self-regulation and rapid weight gain in children from age 3 to 12 years. Arch Pediatr Adolesc Med. 2009; 163:297–302. [PubMed: 19349557]
13. Seeyave DM, Coleman S, Appugliese D, et al. Ability to delay gratification at age 4 years and risk of overweight at age 11 years. Arch Pediatr Adolesc Med. 2009; 163:303–8. [PubMed: 19349558]
14. Rollins BY, Dearing KK, Epstein LH. Delay discounting moderates the effect of food reinforcement on energy intake among non-obese women. Appetite. 2010; 55:420–5. [PubMed: 20678532]
15. Carr KA, Daniel TO, Lin H, Epstein LH. Reinforcement pathology and obesity. Curr Drug Abuse Rev. 2011; 4:190–6. [PubMed: 21999693]
16. Epstein LH, Salvy SJ, Carr KA, Dearing KK, Bickel WK. Food reinforcement, delay discounting and obesity. Physiol Behav. 2010; 100:438–45. [PubMed: 20435052]
17. Kalenscher T, Ohmann T, Gunturkun O. The neuroscience of impulsive and self-controlled decisions. Int J Psychophysiol. 2006; 62:203–11. [PubMed: 16828187]
18. Hofmann W, Schmeichel BJ, Baddeley AD. Executive functions and self-regulation. Trends Cogn Sci. 2012; 16:174–80. [PubMed: 22336729]
19. Allison DB, Kalinsky LB, Gorman BS. A comparison of the psychometric properties of three measures of dietary restraint. Psychological assessment. 1992; 4:391–8.
20. Laessle RG, Tuschl RJ, Kotthaus BC, Pirke KM. A comparison of the validity of three scales for the assessment of dietary restraint. J Abnorm Psychol. 1989; 98:504–7. [PubMed: 2592686]
21. Epstein LH, Carr KA, Lin H, Fletcher KD. Food reinforcement, energy intake, and macronutrient choice. Am J Clin Nutr. 2011; 94:12–8. [PubMed: 21543545]
22. Odum AL, Baumann AA, Rimington DD. Discounting of delayed hypothetical money and food: effects of amount. Behav Processes. 2006; 73:278–84. [PubMed: 16926071]
23. Myerson J, Green L, Warusawitharana M. Area under the curve as a measure of discounting. J Exp Anal Behav. 2001; 76:235–43. [PubMed: 11599641]

24. Ross CE, Wu CL. The Links between education and health. Am Sociol Rev. 1995; 60:719–45.
25. Elo IT, Preston SH. Educational differentials in mortality: United States, 1979–85. Soc Sci Med. 1996; 42:47–57. [PubMed: 8745107]
26. Green L, Myerson J, Ostasqewski P. Discounting of delayed rewards across the life span: Age differences in individual discounting functions. Behav Proc. 1999; 46:89–96.
27. Jaroni JL, Wright SM, Lerman C, Epstein LH. Relationship between education and delay discounting in smokers. Addict Behav. 2004; 29:1171–5. [PubMed: 15236819]
28. Epstein LH, Lin H, Carr KA, Fletcher KD. Food reinforcement and obesity. Psychological moderators. Appetite. 2012; 58:157–62. [PubMed: 22005184]
29. Systat Software. Systat 11.0. SYSTAT Software, Inc; Richmond, CA: 2004.
30. Temple JL, Legierski CM, Giacomelli AM, Salvy SJ, Epstein LH. Overweight children find food more reinforcing and consume more energy than do nonoverweight children. Am J Clin Nutr. 2008; 87:1121–7. [PubMed: 18469229]
31. Bickel WK, Yi R, Landes RD, Hill PF, Baxter C. Remember the future: Working memory training decreases delay discounting among stimulant addicts. Biol Psychiatry. 2011; 69:260–5. [PubMed: 20965498]
32. Koffarnus MN, Jarmolowicz DP, Mueller ET, Bickel WK. Changing delay discounting in the light of the competing neurobehavioral decision systems theory: A review. J Exp Anal Behav. 2013; 99:32–57. [PubMed: 23344987]
33. Houben K, Wiers RW, Jansen A. Getting a Grip on Drinking Behavior: Training Working Memory to Reduce Alcohol Abuse. Psychol Sci. 2011
34. Verbeken S, Braet C, Goossens L, van der Oord S. Executive function training with game elements for obese children: A novel treatment to enhance self-regulatory abilities for weight-control. Behav Res Ther. 2013; 51:290–9. [PubMed: 23524063]
35. Gilbert DT, Wilson TD. Prospection: Experiencing the future. Science. 2007; 317:1351–4. [PubMed: 17823345]
36. Daniel TO, Epstein LH. The future is now: Comparing the effect of episodic future thinking on impulsivity in lean and obese individuals. Appetite. In press.
37. Daniel TO, Stanton CM, Epstein LH. The future is now: Reducing impulsivity and energy intake using episodic future thinking. Psych Sci. In Press.
38. Epstein LH, Wright SM, Paluch RA, et al. Food hedonics and reinforcement as determinants of laboratory food intake in smokers. Physiol Behav. 2004; 81:511–7. [PubMed: 15135024]
39. Best JR, Miller PH. A developmental perspective on executive function. Child Dev. 2010; 81:1641–60. [PubMed: 21077853]
40. Eigsti IM, Zayas V, Mischel W, et al. Predicting cognitive control from preschool to late adolescence and young adulthood. Psychol Sci. 2006; 17:478–84. [PubMed: 16771797]

What is already known about this subject

- Strong food reinforcement and discounting of the future are independently associated with obesity.
- Food reinforcement and delay discounting interact to predict energy intake, with highest intake for those who are high in food reinforcement but cannot delay gratification.
- Food reinforcement and delay discounting interact to predict child weight loss, with smallest weight loss for those who are high in food reinforcement but cannot delay gratification.

What this study adds

- The interaction of food reinforcement and delay discounting improves pediction of BMI over the independent predictors.
- Reinforcing value of high energy dense foods is a better predictor of BMI than reinforcing value of low energy dense foods.
- Being motivated to eat low energy dense foods, and not discounting the future may be protective against obesity.

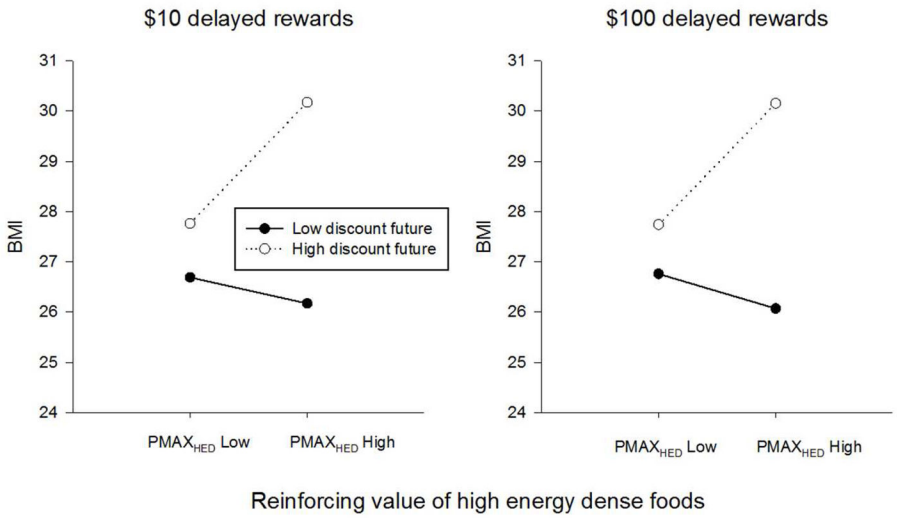

Figure 1.

Interaction of food reinforcement for high energy dense foods ($PMAX_{HED}$) and delay discounting for \$10 (DD_{10}, left graph) and \$100 (DD_{100}, right graph) delayed rewards on BMI. Hierarchical regression analyses revealed that the interaction of $PMAX_{HED}$ X DD_{10} ($F_{inc}(1,190) = 4.25$, $p = 0.041$) or $PMAX_{HED}$ X DD_{100} ($F_{inc}(1,190) = 4.07$, $p = 0.045$) increased variance accounted for beyond the base model that included DD_{10} or DD_{100}, age, education, minority status, dietary disinhibition and dietary restraint. BMI values were calculated from the regression models based on using ± 1 standard deviation values for PMAX and DD values.

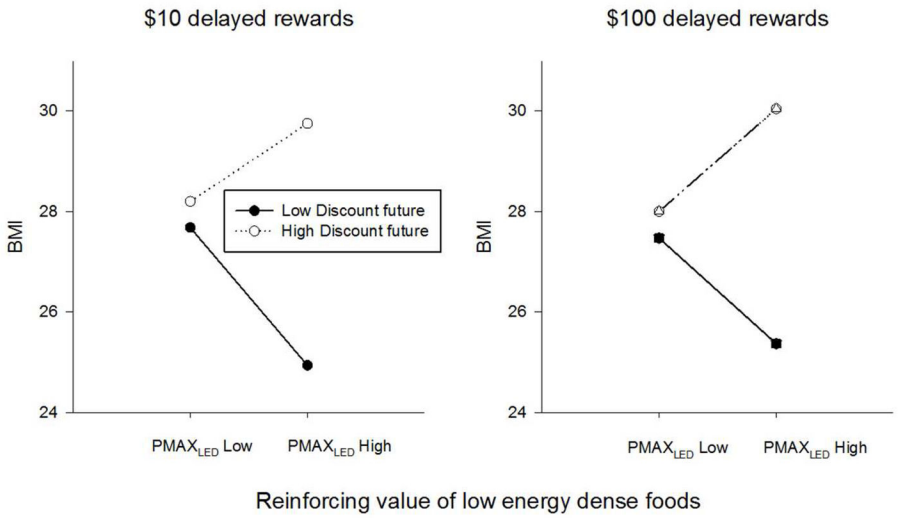

Figure 2.

Interaction of food reinforcement for low energy dense foods ($PMAX_{LED}$) and delay discounting for $10 ($DD_{10}$, left graph) and $100 ($DD_{100}$, right graph) delayed rewards on BMI. Hierarchical regression analyses revealed that the interaction of $PMAX_{LED}$ X DD_{10} ($F_{inc}(1,190) = 4.63$, $p = 0.033$) or $PMAX_{LED}$ X DD_{100} ($F_{inc}(1,190) = 4.34$, $p = 0.039$) increased variance accounted for beyond the base model that included DD_{10} or DD_{100}, age, education, minority status, dietary disinhibition and dietary restraint. BMI values were calculated from the regression models based on using ± 1 standard deviation values for PMAX and DD values.

Table 1

Participant characteristics (Mean ± SD)

N	199
Age	42.7 ± 7.3
Body Mass Index (kg/m^2)	27.6 ± 7.3
Years of education	16.0 ± 3.0
Self reported minority status	
Minority (Non-Caucasian)	46
Caucasian	153
RRV_{HED}	0.37 ± 0.20
$PMAX_{LED}$	24.2 ± 32.4
$PMAX_{HED}$	12.1 ± 17.8
DD_{10}	0.61 ± 0.28
DD_{100}	0.70 ± 0.25
Hunger (after preload)	35.4 ± 25.5
Liking LED foods	4.5 ± 0.7
Liking HED foods	4.6 ± 0.7

Note: $RRV_{HED} = PMAX_{HED}/(PMAX_{HED} + PMAX_{LED})$. N for RRV_{HED} = 197, as two people did not respond for either $PMAX_{LED}$ or $PMAX_{HED}$.