nature publishing group
Open

Usual Energy Intake Mediates the Relationship Between Food Reinforcement and BMI

Leonard H. Epstein[1], Katelyn A. Carr[1], Henry Lin[1], Kelly D. Fletcher[1] and James N. Roemmich[1]

The relative reinforcing value of food (RRV_{food}) is positively associated with energy consumed and overweight status. One hypothesis relating these variables is that food reinforcement is related to BMI through usual energy intake. Using a sample of two hundred fifty-two adults of varying weight and BMI levels, results showed that usual energy intake mediated the relationship between RRV_{food} and BMI (estimated indirect effect = 0.0027, bootstrapped 95% confidence intervals (CIs) 0.0002–0.0068, effect ratio = 0.34), controlling for age, sex, minority status, education, and reinforcing value of reading ($RRV_{reading}$). Laboratory and usual energy intake were correlated (r = 0.24, P < 0.001), indicating that laboratory energy intake could provide an index of eating behavior in the natural environment. The mediational relationship observed suggests that increasing or decreasing food reinforcement could influence body weight by altering food consumption. Research is needed to develop methods of modifying RRV_{food} to determine experimentally whether manipulating food reinforcement would result in changes in body weight.

Obesity (2012) **20,** 1815–1819. doi:10.1038/oby.2012.2

INTRODUCTION

Food is a powerful primary reinforcer that motivates people to eat (1). The reinforcing value (or reinforcing efficacy) of food (RRV_{food}) is assessed by how hard participants will work to obtain food. Based on procedures used to establish the reinforcing efficacy of drugs of abuse (2–4), the work requirements to obtain food are progressively increased and the schedule breakpoint, or the highest level of responding met by the subject, provides an index of food reinforcement. Using these methods, RRV_{food} has been cross-sectionally (5,6) and prospectively (7) related to obesity. RRV_{food} has further been associated with energy intake, as persons who find food more reinforcing eat more food during *ad libitum* eating tasks (8,9).

One way in which RRV_{food} is associated with obesity may be through energy intake, as people gain weight because they are in positive energy balance and obese individuals often consume more energy than leaner counterparts. The primary goal of this study was to assess whether usual energy intake, as assessed by a validated food frequency questionnaire (10,11) mediates the relationship between RRV_{food} and BMI. A secondary goal was to assess the relationship between laboratory energy and macronutrient intake in comparison to usual energy and macronutrient intake. Research has shown that RRV_{food} is related to energy intake in the laboratory (8,9) as well as usual energy intake assessed by repeated 24-h dietary recalls (12).

The most commonly used method of mediation analysis is the causal steps approach described by Baron and Kenny (13,14). In this model, the role of the mediating variable (M) in the relationship between the dependent variable (X) and the independent variable (Y) is confirmed if (i): all three variables are significantly correlated and (ii) if the significant relationship between X and Y is reduced to a nonsignificant level by addition of M into the regression model. However, the causal steps approach has been criticized for low statistical power, for drawing conclusions based on the inference rather than the quantification of the indirect (mediating) effect and for inherent susceptibility to both type I and type II error (15). Several methodologists have recommended newer tests of mediation (15–17). We test whether usual energy intake mediates the relationship between food reinforcement and BMI using the approach proposed by Preacher and Hayes (18) based on the (i) existence of a total effect to be mediated (e.g., a significant relationship between RRV_{food} and BMI) and (ii) a statistically significant indirect effect in the direction predicted, determined by the product of the regression coefficients of the RRV_{food} → increased energy intake (X→M) and increased energy intake → increased BMI (M→Y) paths with confidence intervals (CIs) generated by nonparametric bootstrapping procedures.

METHODS AND PROCEDURES

Participants

Full details of the study design and participant recruitment have been reported elsewhere and are summarized below (12). Two-hundred fifty-two participants (71 nonobese females, 70 nonobese males, 51 obese females, 60 obese males) visited the laboratory for two sessions: an *ad libitum* snack-eating task and a food reinforcement task. The sample

[1]Department of Pediatrics, University at Buffalo School of Medicine and Biomedical Sciences, Buffalo, New York, USA. Correspondence: Leonard H. Epstein (LHENET@acsu.buffalo.edu)

Received 9 June 2011; accepted 22 December 2011; advance online publication 23 February 2012. doi:10.1038/oby.2012.2

included 252 of 273 participants who were studied, as participants who did not complete the Block Food Frequency Questionnaire (n = 9) or reported implausible energy intakes (<800 or >6,000 kcal/day, n = 12) (19) were excluded from the analyses. Exclusionary criteria included taking medications associated with loss of appetite, smoking, diabetes, diagnosis of an eating or psychiatric disorder, allergic to study foods, were currently dieting, and 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. 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: an *ad libitum* snack-eating task and a food reinforcement task scheduled 2–3 weeks apart. After the first session, participants completed the self-administered version of the 2005 Block Food Frequency Questionnaire (11) at home and returned it before the second session. Both sessions were scheduled between the hours of 2:00 and 5:00 PM, during a normal period that individuals would consume additional calories outside of meal time (20). Participants were asked to refrain from consuming food or drinking beverages other than water for at least 3 h and to not consume the experimental foods at least 24 h before the test session. Upon initial arrival at the laboratory, participants gave their written informed consent and completed dietary recalls to ensure adherence to study protocol. Before each session, participants were then provided a choice of three isocaloric energy bar preloads (Clif Bar & Company; Berkeley, CA; 42 g, 150 kcal, 4 g fat, 23 g carbohydrates, 7 g protein) to minimize the effects of hunger on energy intake and food reinforcement. The preload was provided in both sessions to keep the experimental conditions across days as similar as possible, and inclusion of a standard preload increases the ability to show individual differences in food reinforcement (21). Demographic information, height and weight measurements and three dietary habits questionnaires were administered at the end of the *ad libitum* eating session.

Table 1 Participant characteristics

Characteristics	
N	252
Age	34.7 ± 10.7
BMI	29.7 ± 7.2
Hunger[a]	5.6 ± 2.1
Liking of study foods[b]	7.1 ± 0.9
Liking of food reinforcer	8.2 ± 1.0
Sex (M/F)	130/122
Education	
Some high school or completed high school	37
Some college/vocational training	76
Completed college	109
Completed graduate/professional school	30
Minority status	
Minority (non-white)	64
Non-minority (white)	188
RRV_{food}	61.8 ± 132.0
$RRV_{reading}$	81.6 ± 116.9
Laboratory energy intake (kcal)	593.0 ± 316.4
Usual energy intake (kcal)	1,945.2 ± 853.0

Plus-minus values are means ± s.d.
RRV_{food}, relative reinforcing value of food; $RRV_{reading}$, relative reinforcing value of reading.
[a]Post-preload hunger scores at the start of the *ad libitum* eating session. [b]Post-preload liking scores for all foods presented during the *ad libitum* eating session.

Ad Libitum eating task. The ad libitum food consumption task was presented as a taste test (12). 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); plain M&M's (60 g, 5.0);Twix (48 g, 5.0); Kit Kat (42 g, 5.0); and Butterfinger (57 g, 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 on pleasantness, sweetness, blandness, flavorfulness, and bitterness using 9-point Likert-type scales. Afterwards, participants were given several dietary habits questionnaires to complete. They were also told that the food would be discarded after the session and so they were free to continue eating. When participants indicated that they were finished, they were asked to identify their favorite food from among the six available and told that this would be the food used in the food reinforcement test session. The primary dependent variables were laboratory energy and macronutrient intake.

Food reinforcement task. Specifics of the food reinforcement task have been previously described (12). The task was implemented as a computer program in which participants could choose to work for food (RRV_{food}) or reading ($RRV_{reading}$) on concurrent schedules of reinforcement. Subjects responded by clicking a mouse button. Participants earned a point by meeting the schedule requirement, and they 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 min of time to spend reading for every five points earned, depending on which reward they were working for. Progressive fixed-ratio schedules were programmed for food and reading, with response requirements of 4, 8, 16, 32, 64, 128, 256, 512 and so forth for each point. Water was provided *ad libitum*. The dependent measure that we used was the breakpoint, or P_{max} (2), which is the schedule (i.e., 4, 8, 16, 32, etc.) at which subjects last met response requirements for access to the food or non-food alternative. The test–retest reliability of RRV_{food} has been demonstrated (8), and RRV_{food} has been positively associated with energy intake in the *ad libitum* eating task (8,9) and with weight status (5,6,8).

Usual dietary intake. The self-administered version of the 2005 Block Food Frequency Questionnaire (Nutrition Quest, Berkeley, CA) was used to measure usual dietary intake. This 110-item questionnaire was designed to estimate usual and customary intake of a wide array of nutrients and food groups. Methodology for questionnaire development (10,22) and validation (11,23) are available. The food list for the 2005 revision was developed from NHANES 1999–2002 dietary recall data, and the nutrient database for that revision was developed from the USDA Food and Nutrient Database for Dietary Studies (FNDDS), version 1.0. Participants are asked to complete all questions including how frequently they consume specific foods and the usual portion size. The primary dependent variables were usual energy and macronutrient intake.

Height and weight. The participant's weight and height were measured using a digital scale (TANITA, Arlington Heights, IL) and a digital stadiometer (Measurement Concepts & Quick Medical, North Bend, WA). On the basis of height and weight data, BMI was calculated according to the following formula: BMI = kg/m^2.

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

Analytic plan

The goals of the analyses were to assess whether RRV_{food} is related to BMI, and whether usual energy intake mediates this relationship. Mediation models were established using multiple regression, controlling for age, sex, education, minority status, and $RRV_{reading}$ as covariates. The relationship between RRV_{food} and BMI was first evaluated. If there were a significant total effect of RRV_{food} on BMI, the size and significance of the indirect effect of RRV_{food} on BMI through energy intake was then estimated by the product of the regression coefficients of the predictive variables from the RRV_{food} → energy intake and the energy intake → BMI paths. CIs were constructed from 10,000 bootstrap resamples of the data (of the same size as the original study population, with replacement) and implemented *via* a macro developed by Preacher and Hayes (18). We present three ways to interpret the magnitude of the meditational effect. First, the mediator is considered to be significant if the indirect effect of RRV_{food} on BMI through energy intake is significantly different from 0 (the bootstrapped 95% CI does not contain 0). Second, the magnitude of the indirect effect reflects the change in the dependent variable (BMI) indirectly through the mediator variable (energy intake) per a unit change of the independent variable (RRV_{food}) (15). Third, the percent of the total effect of RRV_{food} on BMI explained by the indirect effect of RRV_{food} on BMI through energy intake was quantified by calculation of the effect ratio (indirect effect divided by the total effect) (24). Energy intake and dietary composition in the laboratory was compared to corresponding measures in the natural environment using Pearson product-moment correlations. Data were analyzed using SYSTAT 11 (Systat Software, Chicago, IL; 2004) and SAS 9.2 (SAS Institute, Cary, NC; 2008).

RESULTS

Characteristics of the sample are presented in **Table 1**. RRV_{food} was related to BMI, ($b = 0.0079$, $P = 0.031$, $n = 252$), indicating the presence of a significant total effect. The indirect effect of RRV_{food} on BMI through usual energy intake was significant (estimate = 0.0027, 95% CI = 0.0002, 0.0068), suggesting that usual energy intake is a mediator of the relationship between RRV_{food} and BMI. The size of the indirect effect on BMI mediated through usual energy intake can be estimated by comparing BMI at the average breakpoint of responding for food to BMI at a one-s.d. increase in the breakpoint of responding for food, which would predict an increase of 0.36 BMI units. Based on the effect ratio, usual energy intake explained 34% of the association between RRV_{food} and BMI.

RRV_{food} was related to laboratory ($r = 0.31$, $P < 0.001$) as well as usual energy intake ($r = 0.35$, $P < 0.001$). Usual and laboratory energy intake were significantly related ($r = 0.24$, $P < 0.001$). Laboratory and usual consumption of protein ($r = 0.23$, $P < 0.001$), fat ($r = 0.25$, $P < 0.001$), and carbohydrates ($r = 0.20$, $P = 0.002$) were also significantly related.

DISCUSSION

This study provides support for the hypothesis that energy intake mediates the relationship between food reinforcement and BMI. These results integrate previous findings showing that food reinforcement is related to body weight (6,8) and that food reinforcement is related to energy intake (8,9) to provide insight into the relationship between behavioral phenotypes, dietary behaviors, and obesity. The relationship observed suggests that modifying food reinforcement could influence body weight by altering energy intake. This is very relevant for weight control, as obese persons may find food more reinforcing than other behaviors and thus overeat, contributing to positive energy balance (1). It is also possible that the relationship could be extended towards understanding how to increase body weight in malnourished individuals. One factor that may result in the low body weight of these people is the reduced motivation to eat, and thus increasing the RRV_{food} may be a reasonable goal for this population. There are a wide number of conditions in which increasing energy intake is important for recovery. For example, increasing food reinforcement may be relevant for children with cystic fibrosis, who need to increase their energy intake to gain weight or improve intake of nutrients. Similarly, patients experiencing cachexia from a variety of illnesses may benefit from interventions that increase food reinforcement to enhance eating. The majority of interest in food reinforcement has been focused on obesity and reducing the RRV_{food}, but interventions that increase the RRV_{food} might be useful as an adjunctive treatment for a variety of diseases.

Given that food reinforcement may play a central role in the regulation of body weight through energy intake, innovative methods of modifying the RRV_{food} are warranted. These approaches may broadly fall into two categories: (i) the direct modification of food reinforcement or (ii) the indirect modification of food reinforcement by increasing the efficacy of non-food reinforcers or putting constraints on access to food reinforcers (1). For example, research suggests that the RRV_{food} can be sensitized, or increased, just as the motivation to self-administer drugs can be increased over repeated presentations (25,26). Temple and colleagues have shown that the RRV_{food} can be sensitized based on the characteristics of the food, the amount of food consumed, and the pattern of food consumption. Sensitization of food reinforcement is related to weight gain (27), and these effects may be moderated by weight status, as obese participants were more likely to show sensitization of food reinforcement than their leaner peers (27–29). An alternative is to indirectly modify food reinforcement by providing strong alternative reinforcers or by increasing the constraints on access to food. In scenarios where people have to decide between eating or engaging in alternative non-eating behaviors, such as many snacking opportunities, providing an alternative that is reinforcing and incompatible with eating may shift choice away from food, reducing energy intake (5,30). Similarly, increasing constraints on access to food reinforcers may shift choice from food to alternatives (30,31). If access between alternatives is equal, people generally choose the more reinforcing alternative (1). However, if access varies, choice may shift towards the commodity that is easier to obtain (1). These strategies could further be integrative and specifically target certain types of food. Increasing the variety of fruits and vegetables while restricting the variety of less healthy snack foods may shift preferences toward the former. Subsidizing healthy foods while taxing less healthy foods may similarly influence food selection (32).

The relationship between laboratory energy intake and usual energy intake, as well as the relationship between laboratory macronutrient intake and usual macronutrient intake, suggest that the *ad libitum* eating session may provide an index of eating in the natural environment. This would be a very useful addition to the tools available to assess energy intake, given challenges in measuring usual energy intake. It is recognized that self-reports of energy intake are compromised by underestimation (33,34). Treatment studies may benefit from including a standardized eating task as part of the outcome assessment. It might be useful to consider combining multiple measures of dietary intake to develop the most valid measure of eating, which could include laboratory eating, food frequency questionnaires, and dietary recalls.

There are several aspects of the study methods that may limit the generalization of the results. Dieters were excluded, since they may not want to eat in the laboratory and thus would not work for food. Given the challenges of measuring food reinforcement and *ad libitum* consumption of people who do not want to eat in the laboratory, it is unclear whether the results can be generalized to obese people who are dieting. In addition, all subjects were provided a preload before the food reinforcement and *ad libitum* snack-eating session to focus the task on hedonic, rather than homeostatic hunger (35). However, many eating situations occur when people are energy-deprived and research is needed to assess whether results obtained after a preload can be generalized to eating while experiencing homeostatic hunger. Finally, the current mediation analysis is based on cross-sectional data. A stronger test of whether energy intake mediates the effect of food reinforcement on body weight is through experimental analysis (16).

Food reinforcement may play a central role in the regulation of energy balance and of body weight, but experimental research investigating whether energy intake mediates the relationship between food reinforcement and body weight is needed. For example, a study could be designed to randomly assign one group of participants to an intervention that reduces food reinforcement and another group of participants to a control condition while tracking changes in energy intake and body weight. If decreased food reinforcement mediates the reduction in body weight through negative energy balance, then it would be expected that the groups would differ in body weight changes. Moreover, changes in food reinforcement and energy intake would be correlated in the experimental group but not in the control group. Such experiments would facilitate the development of new methods of modifying food reinforcement as well as confirm the mediating role of energy intake in the relationship between food reinforcement and body weight.

ACKNOWLEDGMENTS

This trial was registered at http:www.clinicaltrials.gov as NCT00962117. Appreciation is expressed to Lora G. Roba, Vida Rostami, Lauren Angelucci, Nicole Gens, and Caitlin Hart for data collection and data entry and assisting in the implementation of protocol. This research was funded in part by a grant from the National Institute of Drug Abuse, R01DA024883 awarded to L.H.E.

DISCLOSURE

The authors declared no conflict of interest.

REFERENCES

1. Epstein LH, Leddy JJ, Temple JL, Faith MS. Food reinforcement and eating: a multilevel analysis. *Psychol Bull* 2007;133:884–906.
2. Bickel WK, Marsch LA, Carroll ME. Deconstructing relative reinforcing efficacy and situating the measures of pharmacological reinforcement with behavioral economics: a theoretical proposal. *Psychopharmacology (Berl)* 2000;153:44–56.
3. Richardson NR, Roberts DC. Progressive ratio schedules in drug self-administration studies in rats: a method to evaluate reinforcing efficacy. *J Neurosci Methods* 1996;66:1–11.
4. Ator NA, Griffiths RR. Principles of drug abuse liability assessment in laboratory animals. *Drug Alcohol Depend* 2003;70:S55–S72.
5. 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;87:1121–1127.
6. Saelens BE, Epstein LH. Reinforcing value of food in obese and non-obese women. *Appetite* 1996;27:41–50.
7. 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;90:276–281.
8. Epstein LH, Temple JL, Neaderhiser BJ *et al*. Food reinforcement, the dopamine D2 receptor genotype, and energy intake in obese and nonobese humans. *Behav Neurosci* 2007;121:877–886.
9. Epstein LH, Wright SM, Paluch RA *et al*. Food hedonics and reinforcement as determinants of laboratory food intake in smokers. *Physiol Behav* 2004;81:511–517.
10. Block G, Hartman AM, Dresser CM *et al*. A data-based approach to diet questionnaire design and testing. *Am J Epidemiol* 1986;124:453–469.
11. Block G, Woods M, Potosky A, Clifford C. Validation of a self-administered diet history questionnaire using multiple diet records. *J Clin Epidemiol* 1990;43:1327–1335.
12. Epstein LH, Carr KA, Lin H, Fletcher KD. Food reinforcement, energy intake, and macronutrient choice. *Am J Clin Nutr* 2011;94:12–18.
13. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *J Pers Soc Psychol* 1986;51:1173–1182.
14. Lockwood CM, DeFrancesco CA, Elliot DL, Beresford SA, Toobert DJ. Mediation analyses: applications in nutrition research and reading the literature. *J Am Diet Assoc* 2010;110:753–762.
15. Preacher KJ, Kelley K. Effect size measures for mediation models: quantitative strategies for communicating indirect effects. *Psychol Methods* 2011;16:93–115.
16. Lockhart G, MacKinnon DP, Ohlrich V. Mediation analysis in psychosomatic medicine research. *Psychosom Med* 2011;73:29–43.
17. Hayes AF. Beyond Baron and Kenny: Statistical mediation analysis in the new millennium. *Commun Monogr* 2009;76:408–420.
18. Preacher KJ, Hayes AF. SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behav Res Methods Instrum Comput* 2004;36:717–731.
19. Schulze MB, Schulz M, Heidemann C *et al*. Fiber and magnesium intake and incidence of type 2 diabetes: a prospective study and meta-analysis. *Arch Intern Med* 2007;167:956–965.
20. Popkin BM, Duffey KJ. Does hunger and satiety drive eating anymore? Increasing eating occasions and decreasing time between eating occasions in the United States. *Am J Clin Nutr* 2010;91:1342–1347.
21. Reiss S, Havercamp S. The sensitivity theory of motivation: implications for psychopathology. *Behav Res Ther* 1996;34:621–632.
22. Block G, Coyle LM, Hartman AM, Scoppa SM. Revision of dietary analysis software for the Health Habits and History Questionnaire. *Am J Epidemiol* 1994;139:1190–1196.
23. Mares-Perlman JA, Klein BE, Klein R *et al*. A diet history questionnaire ranks nutrient intakes in middle-aged and older men and women similarly to multiple food records. *J Nutr* 1993;123:489–501.
24. Shrout PE, Bolger N. Mediation in experimental and nonexperimental studies: new procedures and recommendations. *Psychol Methods* 2002;7:422–445.
25. Robinson TE, Berridge KC. The neural basis of drug craving: an incentive-sensitization theory of addiction. *Brain Res Brain Res Rev* 1993;18:247–291.
26. Robinson TE, Berridge KC. The psychology and neurobiology of addiction: an incentive-sensitization view. *Addiction* 2000;95(Suppl 2):S91–117.

27. Temple JL, Epstein LH. Sensitization of food reinforcement is related to weight status and baseline food reinforcement. *Int J Obes (Lond)* 2011; e-pub ahead of print 1 Nov 2011.
28. Clark EN, Dewey AM, Temple JL. Effects of daily snack food intake on food reinforcement depend on body mass index and energy density. *Am J Clin Nutr* 2010;91:300–308.
29. Temple JL, Bulkley AM, Badawy RL *et al*. Differential effects of daily snack food intake on the reinforcing value of food in obese and nonobese women. *Am J Clin Nutr* 2009;90:304–313.
30. Goldfield GS, Epstein LH. Can fruits and vegetables and activities substitute for snack foods? *Health Psychol* 2002;21:299–303.
31. Lappalainen R, Epstein LH. A behavioral economics analysis of food choice in humans. *Appetite* 1990;14:81–93.
32. Epstein LH, Dearing KK, Roba LG, Finkelstein E. The influence of taxes and subsidies on energy purchased in an experimental purchasing study. *Psychol Sci* 2010;21:406–414.
33. Black AE, Goldberg GR, Jebb SA *et al*. Critical evaluation of energy intake data using fundamental principles of energy physiology: 2. Evaluating the results of published surveys. *Eur J Clin Nutr* 1991;45:583–599.
34. Goldberg GR, Black AE, Jebb SA *et al*. Critical evaluation of energy intake data using fundamental principles of energy physiology: 1. Derivation of cut-off limits to identify under-recording. *Eur J Clin Nutr* 1991;45:569–581.
35. Lowe MR, Butryn ML. Hedonic hunger: a new dimension of appetite? *Physiol Behav* 2007;91:432–439.