ORIGINAL ARTICLE

Prediction of Daily Food Intake as a Function of Measurement Modality and Restriction Status

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ABSTRACT

Objective: Research on eating relies on various indices (e.g., stable, momentary, neural) to accurately reflect food-related reactivity (e.g., disinhibition) and regulation (e.g., restraint) outside the laboratory. The degree to which they differentially predict real-world consumption remains unclear. Further, the predictive validity of these indices might vary depending on whether an individual is actively restricting intake.

Methods: We assessed food craving reactivity and regulation in 46 healthy participants (30 women, 18–30 years) using standard measurements in three modalities: a) self-reported (stable) traits using surveys popular in the eating literature, and b) momentary craving ratings and c) neural activation using aggregated functional magnetic resonance imaging data gathered during a food reactivity-and-regulation task. We then used these data to predict variance in real-world consumption of craved energy-dense "target" foods across 2 weeks among normal-weight participants randomly assigned to restrict or monitor target food intake.

Results: The predictive validity of four indices varied significantly by restriction. When participants were not restricting intake, momentary (B = 0.21, standard error [SE] = 0.05) and neural (B = 0.08, SE = 0.04) reactivity positively predicted consumption, and stable (B = −0.22, SE = 0.05) and momentary (B = −0.24, SE = 0.05) regulation negatively predicted consumption. When restricting, stable (B = 0.36, SE = 0.12) and neural (B = 0.51, SE = 0.12) regulation positively predicted consumption.

Conclusions: Commonly-used indices of regulation and reactivity differentially relate to an ecologically-valid eating measurement, depending on the presence of restriction goals, and thus have strong implications for predicting real-world behaviors.

Key words: eating, food consumption, prediction, restriction, fMRI.

INTRODUCTION

Research on eating and dieting often relies on laboratory indices to accurately reflect food-related reactivity (i.e., tendency toward disinhibited eating) and regulation (i.e., ability/tendency toward restrained eating) outside the laboratory. Typically, standard, validated self-report questionnaires or laboratory tasks are used to investigate stable and momentary food reactivity and regulation in normal-weight, overweight, and obese populations (1,2). The laboratory tasks are increasingly being adapted for the neuroimaging environment to enable investigation of the neural mechanisms underlying food-related reactivity and regulation (3,4).

Whether these separate indices of food reactivity and regulation each account for unique variance in actual food consumption above and beyond the others is largely unknown, partly because studies often do not gather data in multiple response domains. In normal-weight, nondieting individuals, stable indices of reactivity (e.g., self-reported trait dietary disinhibition) predict greater food craving (5), more consumption of energy-dense (ED) foods (6–8), weight gain (9), and higher body mass index (BMI) (10). Momentary indices of food craving (e.g., laboratory task-based ratings of food liking and wanting) predict actual food choice (11,12), are associated with impaired control

BMI = body mass index, **DEBQ** = Dutch Eating Behavior Questionnaire, **ED** = energy dense, **LC** = look craved, **LN** = look neutral, **LNC** = look not craved, **RC** = regulate craved, **RNC** = regulate not craved, **TFEQ** = Three Factor Eating Questionnaire

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over sweet food consumption specifically (13), and positively correlate with energy intake more generally (10). Furthermore, cortical and subcortical brain activity in response to viewing food pictures positively correlates with BMI (3,14) and predicts subsequent weight gain (15). Evidence on the relationship between regulation and food consumption in normal-weight populations is mixed: stable dietary regulation (e.g., self-reported trait dietary restraint) is not related to consumption generally (7–9,16,17), but is associated with decreases in fat intake four years later in Type 2 diabetes samples (18). Neurally, regions underlying food-specific inhibitory control (e.g., ventrolateral prefrontal cortex) have been found to negatively correlate with BMI (14).

These different ways of measuring food reactivity and regulation in the laboratory are regularly used as sole indices of consumption; few studies have compared or combined across these indices to predict food intake. Finlayson et al. (12) found that the Disinhibition subscale of the Three Factor Eating Questionnaire (TFEQ) was positively associated with laboratory ratings of explicit liking and implicit wanting of high-fat sweet foods, and negatively predicted satiation efficiency. French et al. (10) directly compared TFEQ and laboratory measures of explicit liking and wanting on BMI and energy intake, and found that although TFEQ Disinhibition was associated with BMI, explicit liking and wanting predicted energy intake independent of TFEQ. Both of the indices used in these studies, the TFEQ (stable) and laboratory ratings of liking and wanting (momentary), are different forms of self-report, which may be vulnerable to biases when there are strong self-presentational concerns (19) or when introspection upon specific cognitive processes (e.g., self-regulation) is difficult or impossible (20). Therefore, it may be beneficial to directly compare the predictive validity of stable and momentary indices of food reactivity and regulation with measures that do not rely on introspection, such as neuroimaging (21).

In addition, it remains unknown whether the predictive validity of these indices varies depending on whether an individual is actively restricting intake. This is of critical interest given the frequency with which people engage in dietary restriction (22). Furthermore, research on dietary restriction typically involves overweight or obese participants who are on weight loss diets, or compares individuals who chronically restrict their intake with those who do not. The effect of restriction in these populations is confounded by weight status and dieting history, respectively, both of which have known effects on food reactivity and regulation (23,24). Therefore, a more robust understanding of how standard indices of food-related reactivity and regulation relate to actual intake requires random assignment to restriction in a nondieting, nonoverweight, or obese population. Given its effects on other intake-related processes, restriction likely alters the meaning of standard measures of food-related reactivity and regulation. For example, attempting not to eat a favorite food while being exposed to it can actually increase its consumption (25–27). Individuals high on both reactivity (disinhibition) and regulation (restraint) eat more after eating prohibition than do inhibited or low restrainers (25). Together, research suggests that various indices of food reactivity and regulation differentially predict food consumption depending on the presence of restriction, but researchers most often compare chronically restrained to unrestrained eaters. A direct manipulation of restraint is still needed.

In the present study, we compared three kinds of laboratory measures that are commonly used to assess individual differences in food reactivity and regulation. These indices were chosen to be representative of how food-related reactivity and regulation are typically measured in the eating literature, and not necessarily because we hypothesized they would accurately predict food intake. Stable, trait indices were gathered using two of the most widely used questionnaire measures: the TFEQ and the Dutch Eating Behavior Questionnaire (DEBQ). Momentary ratings of food craving and the ability to reduce these cravings using cognitive self-regulation were gathered using a validated food reactivity-and-regulation task. This task was performed while participants were undergoing functional magnetic resonance imaging (MRI), which allowed us to characterize the neural indices of food reactivity and regulation. Data from these three indices were used to predict

TABLE 1. Demographics by Group

Demographics	Control (n = 23)	Restrict (n = 23)
Age, y, M (SD)	21.78 (2.72)	21.72 (1.9)
Sex	15 female, 8 male	15 female, 8 male
BMI, kg/m^2, M (SD)	21.2 (2.13)	22.1 (3.51)
DEBQ restraint, M (SD)	2.42 (0.85)	2.32 (0.82)
DEBQ external, M (SD)	3.29 (0.72)	3.34 (0.46)
TFEQ restraint, M (SD)	0.42 (0.23)	0.4 (0.2)
TFEQ disinhibition, M (SD)	0.37 (0.24)	0.35 (0.21)

The differences between the control and restrict conditions were not significant (all p values > .3).

unique variance in real-world consumption of craved ED foods across 2 weeks among normal-weight, nondieting participants who were randomly assigned to restrict or monitor these foods. Based on past work using the three indices individually, we hypothesized that all three would be generally predictive of food intake. However, given the substantial knowledge gaps reviewed about their unique predictive ability and how that ability might fluctuate with restriction, we did not hypothesize specific differences among the indices or whether they would predict intake more strongly under restriction versus ad lib eating.

METHODS

Participants

Forty-six participants (30 women; mean [M; standard deviation {SD}] age = 21.75 [2.32] years; M [SD]BMI = 21.66 [2.9]) from the University of Oregon community completed the study. Inclusion criteria were as follows: right-handedness; age 18 to 30 years; native English speaking; no reports of dieting in the past 3 months or plans to diet in the future 3 months; and no current or past neurological/psychiatric disorder, head trauma, pregnancy, current psychoactive medication use, and MRI contraindications. Participants were randomly assigned to restrict (n = 23) or control (n = 23) groups (see Table 1 for demographics). Participants earned $30 total for laboratory sessions, $5/wk of consumption reporting, and an additional $5/wk for at least 90% response rate that week. The data were collected from February 2012 through February 2013. All participants provided written informed consent in accordance with University of Oregon's Institutional Review Board.

Procedure

As shown in Figure 1, participants came into the laboratory for a baseline session, in which they completed a food craving regulation task during functional MRI and surveys assessing stable food–related reactivity and regulation. Participants then chose a craved, ED "target food," were randomly assigned to restrict or simply monitor intake of the target food for 2 weeks, and learned to use the text message food craving and eating reporting system.

MRI Task for Momentary and Neural Indices of Craving

The food reactivity-and-regulation task is described fully in Giuliani et al. (4). Stimuli were images of low ED ("Neutral," e.g., broccoli) and high ED (e.g., donuts) palatable foods. The ED foods were selected for each participant based on their most craved ("Craved") and least craved ("Not Craved") food. Thus, "Neutral" images are low ED, "Not Craved" foods are high ED and not craved, and "Craved" foods are high ED and craved. Participants were instructed to Look ("focus on the food and imagine it is actually in front of you") and Regulate ("focus on the food, imagine it was in front of you, and think about it in a way that reduces your desire to eat it") each food. The task was event-related with 5 trial types with 20 trials for each condition: look neutral (LN), look craved (LC), look not craved (LNC), regulate craved (RC), and regulate not craved (RNC). Each trial contained instructions (Look or Regulate; 2 seconds), stimulus presentation (5 seconds), desirability rating (4 seconds), and a jittered intertrial interval (mean = 1 second). Desirability ("How much do you desire to eat this food?") was rated from 1 ("not at all") to 5 ("very much").

Task Data Analysis (Momentary)

Momentary assessments were calculated using the craving ratings made during the MRI task. To create a reliable measure aggregated across baseline stimuli, momentary reactivity to food cues was defined as change in self-reported desire to consume the food both between LN and LC (LC-LN) and between LNC and LC (LC-LNC). Similarly, regulation was defined as change in self-reported desire to consume the food both between LC and RC (LC-RC) and between LNC and RNC (LNC-RNC).

MRI Data Acquisition and Analysis (Neural)

Full details of MRI data acquisition and analysis are in Giuliani et al. (4). Briefly, data were acquired using a 3.0-Tesla Siemens Allegra head-only scanner at the University of Oregon's Robert and Beverly Lewis Center for Neuroimaging. Blood oxygen-level dependent echo-planar images were acquired with a T2*-weighted gradient echo sequence (repetition time = 2000 milliseconds, echo time = 30 milliseconds, flip angle = 80°, matrix size = 64 × 64, 32 contiguous axial slices with interleaved acquisition, field of view = 200 mm, slice thickness = 4 mm). Preprocessing was performed in SPM8 (Wellcome Department of Cognitive Neurology, London, UK; http://www.fil.ion.ucl.ac.uk/spm/) and included correction for field inhomogeneities, realignment and co-registration of functional images to each participant's own high-resolution structural image using a six-parameter rigid body transformation model, reorientation to the plane containing the anterior and posterior commissures, spatial normalization into space compatible with an MNI atlas, and smoothing using a 6-mm^3 full-width half-maximum Gaussian kernel. Whole-brain statistical analyses were implemented in SPM8, to which we applied a combined voxel-height and cluster-extent correction for multiple comparisons to guard against Type I error derived from AFNI's AlphaSim software (28). This determined that a voxel-wise threshold of $p < .001$ combined with a spatial extent

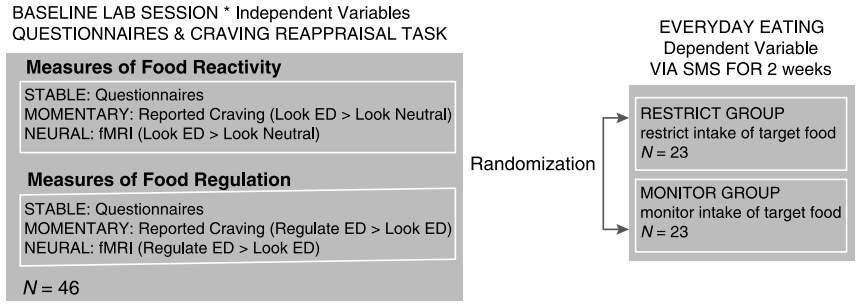

FIGURE 1. Experimental design. Independent variables were gathered in an initial laboratory session: stable, momentary, and neural indices of food reactivity and regulation. Participants were then randomly assigned to either restrict or monitor their intake of a craved target food. The primary dependent variable, food intake (in total number of target food servings consumed), was measured across 2 weeks of text messaging. ED = energy dense; fMRI = functional magnetic resonance imaging; SMS = short message service.

TABLE 2. Top Target Foods Chosen and the Number (Percentage) of Participants Who Chose That Food

Target Foods	Percent Chosen
Sweet foods	
Ice cream	9 (40.9%)
Chocolate	5 (22.7%)
Cake/Brownies	2 (9.1%)
Cookies	2 (9.1%)
Other	4 (18.2%)
Savory foods	
Chips	12 (50%)
Nuts	2 (8.3%)
Peanut butter	2 (8.3%)
Pizza	2 (8.3%)
Other	6 (25%)

threshold of 25 voxels corresponded to a family-wise error corrected false-positive probability of $p < .05$ across the whole brain.

For food-related reactivity, a whole-brain conjunction analysis of the two reactivity contrasts, LC > LN and LC > LNC, revealed common clusters of activity in left dorsal anterior cingulate cortex, left and right parahippocampal gyrus, bilateral and right posterior cingulate cortex, right inferior occipital gyrus, right anterior insula, and right postcentral gyrus. For food-related regulation, a whole-brain analysis of the main effect of regulation, (RC + RNC) > (LC + LNC), revealed active clusters in the posterior cingulate cortex, left parietal cortex, left prefrontal cortex, left and right middle temporal gyrus, thalamus, right middle frontal gyrus, and right insula (see Ref. (4) for complete information on clusters and data acquisition/processing). To reduce the dimensionality of the data, these clusters were combined into reactivity and regulation components based on a components analysis, which is described further later.

Self-Report (Stable)

After the scan, participants completed two commonly used questionnaires that index stable reactivity/regulation in the eating domain: TFEQ (29) (restraint M [SD] = 0.41 [0.22], $\alpha = .82$; disinhibition M [SD] = 0.36 [0.23], $\alpha = .75$) and DEBQ (30) (restrained M [SD] = 2.37 [0.83], $\alpha = .92$; external M [SD] = 3.32 [0.6], $\alpha = .80$). Participants reported their height, weight, and current hunger on a 1 ("very hungry") to 5 ("very full") scale.

Food Consumption

At the end of the baseline session, participants were randomly assigned to restrict or simply monitor their intake of an ED target food (high-calorie, readily available, often consumed when not hungry) for the next 14 days. The researcher and participant worked together to choose a target food and agree upon a reasonable serving size for that target food (e.g., one handful of chips). Serving sizes were standardized across participants who chose the same or similar foods (see Table 2). Participants identified their typical meal and bed times so prompts could occur approximately at the end of each meal and before bed. Participants were prompted four times a day for 14 days via automated text message (mProve Health; mprove.com). At each prompt, participants reported the number of servings of their target food they had consumed since the previous prompt (31). These responses were summed to calculate total target food consumption.

Data Analysis

All independent variables were tested for nonnormality and transformed accordingly if required. Variables were entered into principal components analyses, separately for reactivity and regulation, using Varimax rotations to create two components for each measurement modality: stable reactivity (TFEQ-disinhibition, DEBQ-external), stable regulation (TFEQ-restraint, DEBQ-restriction), momentary reactivity (LC-LN, LC-LNC), momentary regulation (LC-RC, LNC-RNC), neural reactivity (nine clusters from the reactivity conjunction), and neural regulation (eight clusters from

TABLE 3. Poisson Regression Results of Food Intake on Indices of (A) Reactivity and (B) Regulation

Indices	*B*	SE	Wald χ^2	*p*
(A) Reactivity				
Main effects				
Stable	0.046	0.0421	1.198	.27
Momentary	0.167	0.0446	14.02	<.001*
Neural	−0.059	0.0546	1.16	.28
Group	2.06	0.1338	236.82	<.001*
Interactions				
Stable by group	−0.168	0.1643	1.046	.31
Momentary by group	0.447	0.1567	8.138	.004*
Neural by group	−0.137	0.143	0.916	.34
Simple effects: control group				
Stable	0.063	0.0429	2.153	.14
Momentary	0.209	0.0458	20.722	<.001*
Neural	0.083	0.041	4.093	.043*
Simple effects: restrict group				
Stable	0.231	0.1597	2.09	.15
Momentary	−0.238	0.1502	2.519	.11
Neural	0.22	0.1375	2.556	.11
(B) Regulation				
Main effects				
Stable	−0.174	0.0458	14.396	<.001*
Momentary	−0.195	0.0432	20.25	<.001*
Neural	0.03	0.048	0.391	.53
Group	2.015	0.1332	228.805	<.001*
Interactions				
Stable by group	−0.588	0.1325	19.702	<.001*
Momentary by group	−0.398	0.1275	9.721	<.001*
Neural by group	−0.505	0.1355	13.888	<.001*
Simple effects: control group				
Stable	−0.224	0.0543	17.025	<.001*
Momentary	−0.241	0.0478	25.473	<.001*
Neural	0.005	0.0569	0.007	.94
Simple effects: restrict group				
Stable	0.364	0.1198	9.247	.002*
Momentary	0.157	0.1184	1.75	.19
Neural	0.51	0.1216	17.559	<.001*

* $p < .05$.

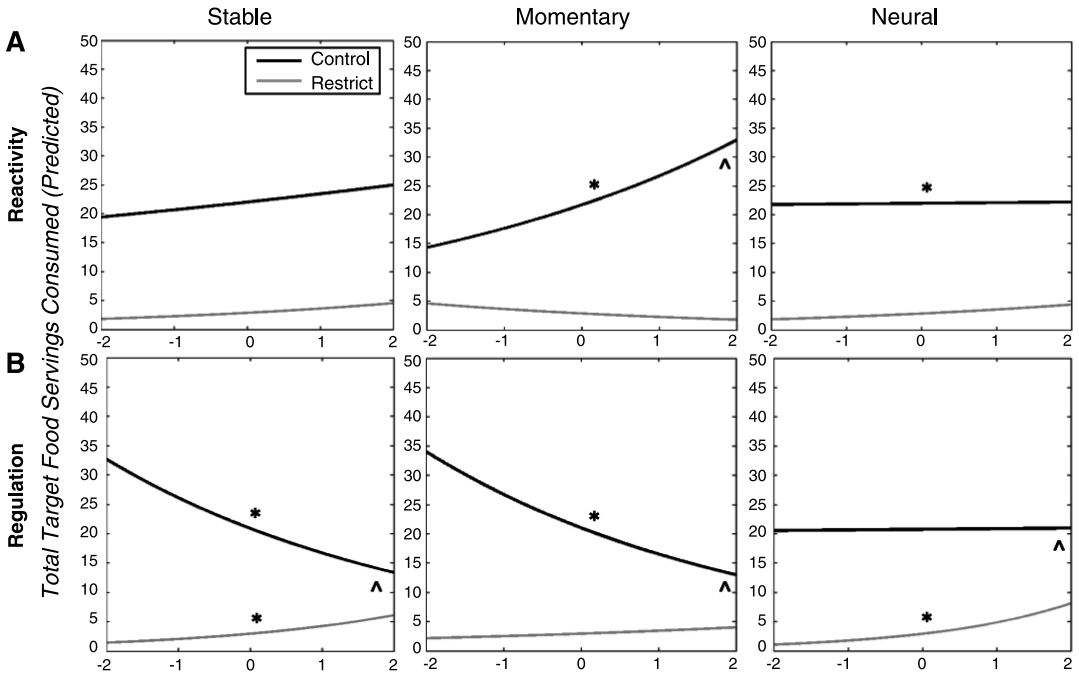

FIGURE 2. Graphs representing the relationship between stable, momentary, and neural reactivity (A) and regulation (B) and predicted total target food servings consumed. Participants instructed to restrict target food consumption are shown in gray, and participants instructed to simply monitor consumption (control) are shown in black. The *x*-axis scales represent the degree of reactivity (top row) or regulation (bottom row) in standard units and reflect a composite of the measures based on independent components analyses as described in the text; *y*-axis scales are the predicted total number of target food servings consumed across the 2-week sampling period. *Main effect $p < .05$; ^ interaction $p < .05$.

Regulate > Look). This procedure maximized the orthogonality of the stable, momentary, and neural components, thus allowing them to be entered simultaneously into regression analyses. The three components for reactivity and regulation were entered simultaneously in generalized linear models with Poisson distributions, which tested whether or not the indices uniquely predicted total servings of target food consumed. Because not all participants responded to every rating prompt, the number of valid responses was entered as a covariate of no interest for all models. All regression analyses were conducted using SPSS19.

RESULTS

Manipulation Check

A one-way analysis of variance was performed to test the efficacy of the restriction instructions on the total number of target food servings consumed across the 2-week period. Participants who were instructed to restrict their consumption reported eating significantly fewer servings of their target food (M [SD] = 3.48 [4.06]) than did those who were instructed to only monitor their intake (M [SD] = 20.83 [19.22]; $F(1,44) = 17.95$, $p < .001$).

Reactivity

Stable, momentary, and neural indices of reactivity were entered simultaneously as predictors of target food consumption with group and group by index interaction terms. As shown in Table 3A, the main effects of momentary reactivity and group assignment were significant (see the table for full statistics, including regression parameters and associated *p* values). The group interaction was significant for momentary reactivity. Simple effects within group revealed that momentary and neural reactivity indices positively predicted consumption among control participants, who were simply monitoring their intake of the target food, but none of the indices did so among participants who were restricting their intake of the target food (Fig. 2A). The correlation between the stable and momentary indices of reactivity was −0.19 ($p = .22$), that between the stable and neural indices was −0.08 ($p = .6$), and that between the momentary and neural indices was 0.03 ($p = .85$).

Regulation

As shown in Table 3B, the main effects of stable and momentary regulation were significant, as was the effect of group assignment. Significant group interactions emerged for all indices of regulation. Decomposing these, simple effects within group showed that stable and momentary indices of regulation negatively predicted consumption in participants simply monitoring their target food intake, and

stable and neural indices positively predicted consumption in those restricting their target food intake (Fig. 2B). The correlation between the stable and momentary indices of regulation was −0.13 (p = .39), that between the stable and neural indices was 0.09 (p = .56), and that between the momentary and neural indices was −0.09 (p = .55).

DISCUSSION

We compared how well stable, momentary, and neural indices of food reactivity and regulation separately predicted food consumption, and whether those indices did so differentially depending on whether or not normal-weight, nondieting participants had been assigned to restrict their intake of that food. Importantly, we found significant interactions between group assignment and indices of momentary reactivity and of stable, momentary, and neural regulation in predicting food intake: when the participants were not restricting, reactivity (momentary, neural) related positively and regulation (stable, momentary) related negatively to consumption; however, when the participants were restricting their intake, regulation (stable, neural) related positively to consumption.

The present findings are consistent with research using momentary and neural indices in isolation to predict food intake. Our statistical models that included all three kinds of predictors revealed that momentary and neural indices accounted for unique variance in food intake above and beyond stable (self-report) measures. Contrary to studies showing that stable indices of reactivity predict greater consumption of ED foods (6–8), we did not find a significant association between self-reported stable reactivity and food consumption in either group. This indicates that either there is no relationship between self-reported trait food reactivity and consumption or the surveys used to create the stable reactivity composite may lack predictive validity in small samples. Regarding regulation, we found that momentary and stable regulation negatively predicted consumption in the participants not restricting intake, which is inconsistent with previous studies (e.g., Refs. (7–9,16,17)) that did not find a relationship between self-reported trait regulation and food intake or body weight. We also did not observe the relationship between neural correlates of food restriction and food intake seen in previous work. This may be due to the fact that we included all three indices in the same model, a procedure that isolates the unique variance in food consumption explained by each index.

Restriction (versus not restricting) altered the relationship between several of the indices and food intake. Specifically, restriction eliminated the relationships between intake and momentary reactivity (positive) and momentary regulation (negative) observed in the nonrestriction group. Restriction also apparently reversed the relationship between stable regulation and intake, from negative to positive, and created a positive relationship between the neural index of regulation and intake where there had been none in the nonrestricting group. This paradoxical pattern of greater stable and neural regulation predicting greater consumption supports the hypothesis that restriction can lead to greater consumption (25–27). An important feature of our randomized design lies in its ability to demonstrate this pattern among individuals who were *assigned* to restrict their intake of a single food, suggesting that these effects are not merely due to preexisting features of chronically restrained eaters.

The present study has several limitations. First, the measurement tools chosen to represent stable, momentary, and neural indices of food reactivity and regulation in the present study are only a subset of those that are commonly used to assess individual differences in these processes. However, our direct comparison of these indices represents a useful and important preliminary step toward a fuller understanding of the (separable) processes underlying food reactivity and regulation that nonetheless applies to hundreds of studies in the literature. For example, as of publication of this article, the TFEQ has been cited 2640 times and the DEBQ has been cited 1395 times. Second, food intake was measured using the number of servings participants reported consuming since the last text message prompt. Although self-reports of food intake can be unreliable, a considerable body of evidence supports the idea that measurement methods such as ecological momentary assessment that capture reports close in time to the experience at hand can dramatically reduce these biases (32,33). Text messaging allows for highly timely reporting of food intake compared with alternative methods (34). Nonetheless, this is still a potential source of variability, so future studies should investigate food intake using more objective measures such as the Remote Food Photography Method (35) or direct observation where possible. For example, Remote Food Photography Method estimates of food intake do not significantly differ from doubly labeled water estimates and do not show increased error among participants with larger body mass (36). Third, because the data are drawn from a relatively young, college-attending, and nonobese sample, it is not possible to know whether the results generalize to other groups. However, given that this is the first study to directly compare these three indices of reactivity/regulation in their ability to predict food intake, we intentionally recruited a relatively homogeneous sample to increase our power to detect effects. Research in our laboratory and others can now extend this line of work to other populations, and that subsequent research will benefit from the initial estimates of the effects provided here.

This study is the first to compare, directly and simultaneously, the predictive validity of three indices across different response modalities that are commonly used to assess food-related reactivity and regulation. Our results

demonstrate that these indices each account for unique variance in daily eating behavior, adding to past work comparing stable and momentary indices of reactivity and regulation (10,16). Thus, incorporating a fuller range of measurement tools into studies of real-world eating behavior will allow researchers to capture greater predictive power.

All authors conceived this study, N.R.G. collected the data, N.R.G. and E.T.B. analyzed and interpreted the data, and all authors were involved in writing the manuscript and had final approval of the submitted and published versions. We thank Lisa May for graphic design support. We also thank the participants in this study and the Lewis Center for Neuroimaging.

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