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Neural Predictors of Giving in to Temptation in Daily Life

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Abstract
The ability to control desires, whether for food, sex, or drugs, enables people to function successfully within society. Yet, in tempting situations, strong impulses often result in self-control failure. Although many triggers of self-control failure have been identified, the question remains as to why some individuals are more likely than others to give in to temptation. In this study, we combined functional neuroimaging and experience sampling to determine if there are brain markers that predict whether people act on their food desires in daily life. We examined food-cue-related activity in the nucleus accumbens (NAcc), as well as activity associated with response inhibition in the inferior frontal gyrus (IFG). Greater NAcc activity was associated with greater likelihood of self-control failures, whereas IFG activity supported successful resistance to temptations. These findings demonstrate an important role for the neural mechanisms underlying desire and self-control in people's real-world experiences of temptations.

Keywords
self-control, neuroimaging, individual differences

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The inability to curb desires and control impulses has far-reaching implications and costs for individuals and society at large (Baumeister, Heatherton, & Tice, 1994; Schroeder, 2007). Indeed, it has been estimated that up to 40% of deaths in the United States every year are attributable to self-control failures (Mokdad, Marks, Stroup, & Gerberding, 2004; Schroeder, 2007). Many models portray self-control as the outcome of a balance between the strength of impulses (e.g., desires and cravings) and the exertion of self-control (Hare, Camerer, & Rangel, 2009; Heatherton & Wagner, 2011; Hofmann, Friese, & Strack, 2009; Metcalfe & Mischel, 1999). These models predict that whenever this balance is tipped in favor of impulses, a person is especially prone to self-control failure (Heatherton & Wagner, 2011). Although previous research has identified some predictors of self-control failure, such as negative affect and resource depletion (for a review, see Wagner & Heatherton, in press), it is still unclear why certain people generally succeed at regulating their impulses and behaviors whereas others consistently fail. Indeed, identifying those individuals who are most likely to give in to temptation in the short term may help health practitioners develop programs that prevent this behavioral tendency from turning into a chronic, unhealthy lifestyle in the long run (e.g., overeating and obesity in the food domain).

To address this question, we set out to model how successful (and unsuccessful) people are in controlling their desires to eat on a daily basis. Although merely asking people to report their past eating behaviors might adequately capture differences in self-control, there are several reasons why retrospective self-report can be problematic. For instance, people's recall of their past behavior may be subject to memory biases (Gorin & Stone, 2001; Schwarz, 1999) and could lead them to misreport how often they have succumbed to a temptation. People also tend to grossly underestimate the number of eating decisions they make on a daily basis (Wansink, 2007). Thus, testing hypotheses about why some people

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are better than others at self-control may be difficult using retrospective measures that rely on participants having perfect recall of past events. As a result, it can be difficult to tease apart competing accounts of diet failure, such as those positing that excessive appetite and desire strength are critical factors (Hofmann & Dillen, 2012) and those proposing that compromised willpower is to blame (Mischel, Cantor, & Feldman, 1996).

One way to get around the inherent response biases in self-reports is to assess underlying neural correlates. We took this approach in the present study by pairing functional neuroimaging with subsequent smartphone experience-sampling technology to test whether neural reward activity in response to viewing appetizing food cues (Demos, Heatherton, & Kelley, 2012; Kelley, 2004; Wagner, Boswell, Kelley, & Heatherton, 2012) predicts the strength of everyday food desires, and whether activity in brain regions previously implicated in response inhibition (Berkman, Falk, & Lieberman, 2011; Menon, Adleman, White, Glover, & Reiss, 2001) predicts successful resistance of those desires. To elicit food-cue-specific reward activity, we used a cue-reactivity paradigm adapted from previous work in our lab (e.g., see Demos et al., 2012), and to examine the role of regions related to self-control, we administered a go/no-go response-inhibition task (Casey et al., 1997).

We then determined whether brain activity evoked by food cues or during a self-control task (i.e., go/no-go task) predicted participants' daily eating behaviors, as assessed via experience sampling. More specifically, we tested whether activity in the ventral striatum (specifically, the nucleus accumbens, NAcc) in response to images of food predicted desire for food, tendency to give in to desire, and amount of food consumed. We also tested whether activity in the inferior frontal gyrus (IFG) during the self-control task predicted successful resistance to desire for food and thereby decreased likelihood of eating. Overall, we aimed to determine whether, above and beyond self-report, these brain markers would be able to predict the self-regulatory outcomes of daily eating behaviors.

Method

Thirty-one female participants completed an initial functional MRI (fMRI) scanning session involving the cue-reactivity and response-inhibition tasks. At the end of the scanning session, we also collected two trait-level measures of interest, dieting status (Herman & Polivy, 1980) and sensitivity to external food cues (van Strien, Herman, & Anschutz, 2012). The fMRI session was followed by 1 week of experience sampling of participants' food desires and eating behaviors, in accordance with Hofmann, Baumeister, Förster, and Vohs's (2012) procedure. Specifically, all participants were provided with Blackberry smartphones and each day were signaled with seven short surveys (one randomly timed survey for each 2-hr interval) that prompted them to report on episodes of desire that might have occurred within the past half hour. Whenever participants indicated that they had experienced a food desire, they were asked to report on the strength of that desire, their resistance to it, whether or not they had given in to the desire and eaten (enactment), and if so, the amount they had eaten.

Participants

We recruited 31 females (mean age = 21.1 years, range = 18–28) from the Dartmouth College community to participate in the study. Sample size was determined on the basis of previous studies using the brain-as-predictor approach (see Berkman et al., 2011) and using our cue-reactivity paradigm (Wagner, Altman, Boswell, Kelley, & Heatherton, 2013). We sought to enroll 30 participants in the study, following the stop rule that eligible participants had to be able to complete all phases of the study by the end of the academic term in which they enrolled in the study. We included only female participants to be consistent with previous cue-reactivity studies that sampled from the same population (Demos et al., 2012), as well as to avoid the confound of gender effects on eating behaviors (Holm-Denoma, Joiner, Vohs, & Heatherton, 2008). All participants were right-handed, had normal or corrected-to-normal vision, and reported no history of psychiatric or neurological disorders. Because we were interested in capturing variability in eating behaviors in the general population, we did not recruit participants on the basis of dieting status per se, but in order to account for differences in dietary restraint, we had all participants complete the Restraint Scale (Heatherton, Peter, Polivy, King, & McGree, 1988; Herman & Polivy, 1980).

At the beginning of the study, we informed participants that it was about cognition and emotion in everyday life. Upon successful completion of the fMRI and experience-sampling portions of the study, participants were debriefed on its general goals. All participants gave their informed consent according to guidelines set by Dartmouth's Committee for the Protection of Human Subjects.

Stimuli

All images in the food-cue-reactivity task were adapted from previous work in our lab (Demos et al., 2012; Demos, Kelley, & Heatherton, 2011). In total, the images depicted 90 high-calorie foods, including 30 dessert items, 30 fast-food meals, and 30 snacks; these food images were intermixed with images of other types.

Stimuli in the go/no-go task were images taken from the cue-reactivity task (i.e., social scenes, food images), but we collapsed across image type to test whether domain-general response inhibition would encompass and be predictive of resistance to food impulses.

Imaging apparatus

All neuroimaging data were collected with a 3-T Philips Intera Achieva scanner equipped with a Philips 32-channel SENSE head coil (Philips Medical Systems, Bothell, WA). Stimuli were presented using SuperLab 4.0 (Cedrus Corp., San Pedro, CA) and projected to an Epson (Long Beach, CA) ELP-7000 LCD screen positioned at the end of the scanner bore. Participants were able to view the screen via a mirror mounted on the head coil. They made all responses with button presses on a Lumina LU-400 fMRI response pad (Cedrus Corp., San Pedro, CA).

Imaging procedure

In the fMRI scanning session, all participants completed the cue-reactivity task first and then the go/no-go task. In the cue-reactivity task, we instructed participants to make simple perceptual judgments as to whether each image they viewed depicted an indoor or outdoor scene. All judgments were reported with a button press. Because the task incorporated images of multiple types, including images of people and nature scenes, participants were naive to the true purpose of the task. In total, there were 180 images displayed: 90 food images and 90 control images (people and nature scenes). In the go/no-go task, we asked participants to respond to a certain image type (go condition) by pressing a button and to withhold response to another image type (no-go condition) by refraining from pressing the button. Pairings of image and trial types were counterbalanced across participants, so there were four possible trial types (food or nonfood image paired with go or no-go cue).

Both tasks implemented a rapid event-related design. In the cue-reactivity task, each trial consisted of the presentation of an image (a food item, people, or a nature scene) for 2.5 s. We pseudorandomized the order of these image types. During interstimulus intervals (ISIs), a white fixation cross was displayed on a black background; the duration of the ISIs was randomly jittered (0–12.5 s) to create variable fixations that allowed for more accurate estimation of task effects. In each trial of the go/no-go task, a stimulus was presented on a black background for 500 ms (jittered ISIs ranged from 2 to 9.5 s). The go/no-go task consisted of a total of 108 go trials and 36 no-go trials.

For each task, data were collected in two functional runs. Each run of the cue-reactivity task consisted of 250 whole-brain volumes, and each run of the go/no-go task consisted of 266 volumes. The same acquisition parameters were used for the two tasks (36 axial slices per whole-brain volume, 3.5-mm thickness, 0.5-mm gap; 3- × 3-mm in-plane resolution).

Image preprocessing and analysis

The fMRI data were analyzed using Statistical Parametric Mapping software (SPM8; Wellcome Department of Cognitive Neurology, London, United Kingdom) in conjunction with a suite of tools for preprocessing and analysis (created by one of the authors and available at http://github.com/ddwagner/SPM8w). For each functional run, data were preprocessed to remove sources of noise and artifact and were corrected for differences in slice timing. Functional data were realigned within and across runs to correct for head movement and were unwarped to reduce residual movement-related image distortions that realignment may have failed to correct. Functional data were normalized into a standard stereotaxic space (3-mm isotropic voxels) based on the SPM8 Echo Planar Imaging template that follows the International Consortium for Brain Mapping 152 brain template space (Montreal Neurological Institute, MNI). To spatially smooth the normalized images, we applied a Gaussian kernel (6-mm full width at half maximum). For the go/no-go task, 3 participants' data were not included in the reported analyses (1 participant failed to complete the task, and 2 participants showed extreme motion-related artifact).

For each participant and for both tasks, we ran a general linear model (GLM) that included task effects (i.e., trial type) and covariates of no interest (instruction trials, error trials for the go/no-go task, a linear trend, and six motion parameters derived from realignment corrections). GLMs were convolved with a canonical hemodynamic response function and used to compute parameter estimates for comparisons at each voxel. For the cue-reactivity task, contrast images comparing food-image trials with trials involving all other stimuli (people and nature scenes) were entered into a second-level random-effects analysis, with the participant treated as the random effect. For the go/no-go task, go and no-go trials were modeled separately. Because we were interested in examining brain activity associated with response inhibition, for each subject we generated contrast images comparing no-go with go trials. These images were subsequently subjected to a second-level random-effects analysis, with the participant again treated as the random effect. Any trials in which participants made errors, whether omission or commission errors, were excluded from all analyses. This allowed for easier interpretation of response-inhibition-related activity in the no-go > go contrast.

To localize our NAcc region of interest (ROI) in the cue-reactivity task, we applied a functionally defined, spherical mask (4 mm) to the right NAcc (MNI coordinates: 12, 9, –3; based on previous work in our lab; Demos et al., 2011). In the present study, this ROI showed significant food-cue-specific activation, $t(30) = 2.87$, $p = .007$. We extracted the mean beta values from this region to be used in subsequent multilevel regression models predicting intensity and enactment of food desires and amount eaten.

For the go/no-go task, we first ran Monte Carlo simulations using AFNI's AlphaSim (Ward, 2000) to calculate the minimum cluster size at an uncorrected threshold of $p < .005$ (required for a whole-brain correction of $p < .05$). We performed simulations (1,000 iterations) on the volume of the study-wide whole-brain mask. These simulations estimated a minimum cluster size of 180 voxels. On the basis of this estimation, we selected a spherical ROI (6 mm) in the left IFG centered on peak voxels (MNI coordinates: –36, 30, –3). Again, we extracted the mean beta values from this ROI to be used in our multilevel models.

Experience-sampling procedure

Following the fMRI scan, all participants underwent a short training session in which they received oral and written instructions on how to use the Blackberry smartphones. The experience-sampling protocol was administered on the smartphones via a customized Java ME software application that determined the assessment schedule, administered the questionnaire, and logged data.

Participants carried the smartphones on their person during the 1-week experience-sampling period. Each day, seven signals were distributed across a 14-hr time window, with each signal occurring randomly within a 2-hr time block, as per the recommendation of Hektner, Schmidt, and Csikszentmihalyi (2007). Any two signals were constrained to be at least 30 min apart. If the smartphone was turned off during the time of a signal, the program postponed the signal until later in the time block; if the time block passed without the smartphone being turned back on, the response was logged as missing. If the smartphone was turned on but the participant did not respond within 15 min of the signal's onset, the program turned off, and the response was categorized as missing. To ensure that we would have enough data-collection points per participant, we extended the experience-sampling period an additional day if a participant responded to fewer than five signals on any given day. In that case, a pop-up message appeared on the screen, asking the participant to carry the device an additional day. For each signal, participants reported the following: (a) desire strength (the experienced strength of food desire, on a scale from 0, *none at all*, to 6, *irresistible*); (b) resistance (how much they attempted to resist the desire, on a scale from 0, *not at all*, to 6, *very much*); (c) enactment (whether or not they had given in to the desire and already eaten; *yes/no*); and if they had eaten, (d) the amount eaten (on a scale from 1, *a tiny bit*, to 6, *much more than a regular portion/stuffed*).

None of the participants reported any difficulty or disruption associated with responding to the questionnaires, and the average response time per signal was only 2.65 min. Overall, there was high compliance, as participants completed responses to an average of 83% of all signals during the experience-sampling period and reported having food desires more than half of the time (54.4%).

Multilevel-analysis procedure

All multilevel regression models were run using the software package Hierarchical Linear Modeling (HLM; Raudenbush, 2004). Dependent variables were not transformed, but because enactment of food desires was a dichotomous variable, logistic multilevel regression was applied by specifying the Bernoulli model in HLM (Raudenbush, 2004). Level 1 predictors (the measurements of desire and resistance during the experience-sampling period) were person-mean centered, whereas Level 2 predictors were grand-mean centered. To incorporate neural data from the cue-reactivity and response-inhibition tasks, we included two Level 2 brain predictors in all models: signal-change beta values from (a) the NAcc ROI that showed significant activation in response to appetitive food images in the cue-reactivity task (hereafter, *NAcc activity*) and (b) the IFG ROI associated with successful response inhibition in the go/no-go task (hereafter, *IFG activity*; see Image Preprocessing and Analysis). In all models, we accounted for individual differences in dietary restraint by including scores from the Restraint Scale (Heatherton et al., 1988; Herman & Polivy, 1980) and scores from the External Eating subscale of the Dutch Eating Behavior Questionnaire (van Strien et al., 2012) as Level 2 predictors.

To answer our main questions, we ran several hierarchical linear regression models with situational variables at Level 1 and person-based variables (e.g., NAcc activity) at Level 2 to accommodate the nested structure of the experience-sampling data (i.e., observations within persons). All models incorporated Level 1 random intercepts, and the models predicting enactment and amount eaten included Level 1 random slopes for the relationship between resistance and the given outcome variable—as determined by variance components tests (all ps $< .005$; Hox, 2010).

Results

In the first model, we regressed desire strength on the brain and trait predictors. Individuals with higher NAcc activity in response to appetitive food images during fMRI scanning experienced more intense food desires, $b = 0.27$, $p = .003$. Additionally, participants who reported being more sensitive to external food cues tended to have stronger desires, $b = 0.05$, $p = .023$. No other effects reached significance (see Table 1 for all estimated model parameters).

In the second model, our outcome variable was whether or not people gave in to their temptations to eat (i.e., enactment; see Table 2 for complete results). We replicated previous work (Hofmann et al., 2012) by showing that situational variation in the strength of food desire and resistance to food desire affected the likelihood of enactment; greater strength of desire predicted more frequent enactment, b_{log} (predicted log odds) = 0.32, $p < .001$, and higher resistance to desire predicted less frequent enactment, $b_{log} = -0.30$, $p = .004$. In addition to these effects, there was a main effect of NAcc activity on enactment, such that those participants who showed higher NAcc activity in the cue-reactivity task were more likely to give in to their temptations to eat, $b_{log} = 0.38$, $p = .014$. Greater sensitivity to external food cues also predicted enactment, $b_{log} = 0.05$, $p = .022$.

In this model, we also observed moderating effects of IFG activity associated with successful response inhibition in the go/no-go task. On average, individuals with higher IFG activity during the go/no-go task acted on their desires less frequently, as indicated by the significant negative interaction between IFG activity and desire strength, $b_{log} = -0.30$, $p = .003$, as well as the significant interaction between IFG activity and resistance, $b_{log} = -0.42$, $p = .019$. The interaction plot in Figure 1 shows that in particularly tempting situations (i.e., those characterized by active resistance to food desires), high-IFG individuals (IFG activity 1 *SD* above the mean) were considerably more successful at regulating their food consumption compared with low-IFG individuals (IFG activity 1 *SD* below the mean). Indeed, in these situations, low-IFG individuals were estimated to be 8.2 times more likely to give in to a food desire than were their high-IFG counterparts.

The last model we ran included the same predictor variables as the second model (predicting enactment), but with amount of food eaten as the outcome variable (see Table 3 for complete results). We found a pattern of effects similar to that in the second model, including main effects of NAcc activity, $b = 0.23$, $p = .025$, and food-cue sensitivity, $b = 0.05$, $p = .002$. IFG activity weakened the relationship between desire strength and amount eaten, $b = -0.19$, $p = .012$, such that those individuals with higher IFG activity ate less when faced with temptations to eat (i.e., experienced desire). Note that NAcc activity had consistent effects on the outcome measures across all three models described here (see Fig. 2 for a depiction of these effects).

Table 1. Multilevel Regression Results: Predicting Desire Strength From Trait and Brain Variables

Predictor	*b*	*SE*	*p*
Base predictor (Level 1)			
Intercept	4.20	0.09	< .001
Trait and brain predictors (Level 2)			
NAcc activity	0.27	0.08	.003
IFG activity	–0.13	0.21	.547
Dietary restraint	–0.02	0.02	.200
Food-cue sensitivity	0.05	0.02	.023

Note: IFG = inferior frontal gyrus; NAcc = nucleus accumbens.

Table 2. Multilevel Logistic Regression Results: Predicting Enactment From Desire, Resistance, and Trait and Brain Variables

Predictor	b_{log}	*SE*	*p*
Base predictors (Level 1)			
Intercept	–0.39	0.68	.010
Desire strength	0.32	0.09	< .001
Resistance	–0.30	0.09	.004
Trait and brain predictors (Level 2)			
NAcc activity	0.38	0.14	.014
IFG activity	0.14	0.23	.555
Dietary restraint	–0.03	0.02	.160
Food-cue sensitivity	0.05	0.02	.022
Interactions with desire strength			
NAcc Activity × Desire Strength	0.19	0.10	.069
IFG Activity × Desire Strength	–0.30	0.10	.003
Dietary Restraint × Desire Strength	–0.03	0.02	.178
Food-Cue Sensitivity × Desire Strength	< .001	0.01	.979
Interactions with resistance			
NAcc Activity × Resistance	–0.23	0.07	.004
IFG Activity × Resistance	–0.42	0.17	.019
Dietary Restraint × Resistance	–0.03	0.02	.276
Food-Cue Sensitivity × Resistance	–0.02	0.02	.398

Note: b_{log} = predicted log odds; IFG = inferior frontal gyrus; NAcc = nucleus accumbens.

Discussion

Taken together, the results from the present study provide initial evidence for neural markers of everyday eating behaviors that can identify individuals who are more likely than others to give in to temptations to eat. Food-cue reactivity in the NAcc, a part of the mesolimbic

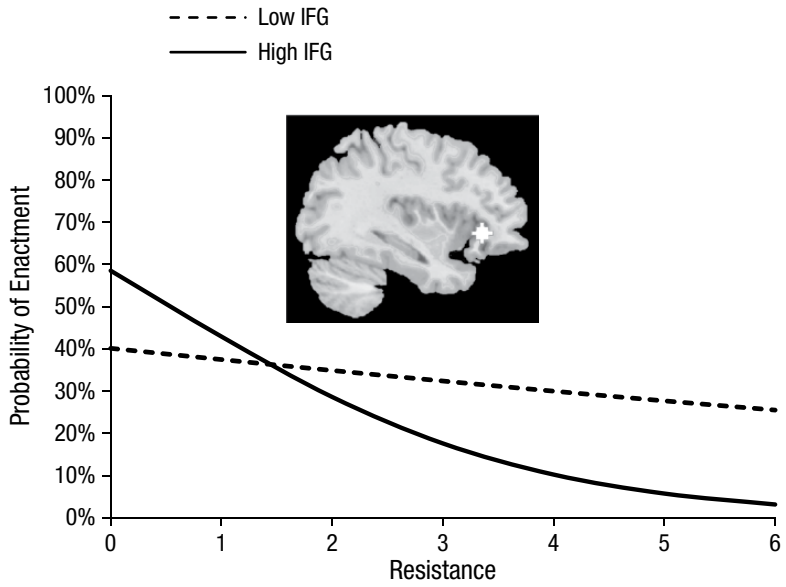

Fig. 1. Interaction plot depicting the effect of reported resistance to food desire on the probability of giving in to the temptation to eat (enactment) as moderated by individual differences in recruitment of the inferior frontal gyrus (IFG; see inset) during successful response inhibition in the go/no-go task (Montreal Neurological Institute coordinates for the IFG region of interest: −36, 30, −3). Predicted log-odds for individuals with low (1 *SD* below the mean) and high (1 *SD* above the mean) IFG recruitment have been transformed to probabilities.

dopamine system associated with reward processing (Schultz, 2006), significantly predicted strength of food desires, enactment of those desires, and even the amount eaten (Fig. 2). Additionally, the moderating effects of IFG activity suggest that the IFG is a critical brain region that can influence self-regulatory outcomes, especially when people are faced with strong temptations and self-control is required (Fig. 1). Those individuals who recruited the IFG more during the response-inhibition task tended to be less likely to succumb to temptations and also ate less.

Table 3. Multilevel Regression Results: Predicting Amount Eaten From Desire, Resistance, and Trait and Brain Variables

Predictor	*b*	*SE*	*p*
Base predictors (Level 1)			
Intercept	1.17	0.11	< .001
Desire strength	0.23	0.06	< .001
Resistance	–0.21	0.06	.004
Trait and brain predictors (Level 2)			
NAcc activity	0.23	0.10	.025
IFG activity	0.08	0.19	.667
Dietary restraint	< 0.01	0.02	.879
Food-cue sensitivity	0.05	0.01	.002
Interactions with desire strength (DS)			
NAcc Activity × Desire Strength	0.20	0.06	.001
IFG Activity × Desire Strength	–0.19	0.08	.012
Dietary Restraint × Desire Strength	–0.02	0.01	.187
Food-Cue Sensitivity × Desire Strength	0.02	0.01	.071
Interactions with resistance			
NAcc Activity × Resistance	–0.10	0.06	.089
IFG Activity × Resistance	–0.14	0.11	.224
Dietary Restraint × Resistance	–0.01	0.02	.459
Food-Cue Sensitivity × Resistance	–0.01	0.01	.562

Note: IFG = inferior frontal gyrus; NAcc = nucleus accumbens.

The present findings also demonstrate the importance of individual differences in how people experience and respond to temptation in their day-to-day lives. These differences appear to arise not only from how temptation is experienced in the moment (as measured by desire strength and resistance), but also from neural mechanisms associated with both reward processing (NAcc) and response inhibition (IFG). Indeed, the models we report here indicate that variation in these brain regions' activity predicts how well (or poorly) individuals exert self-control when confronted with temptations to eat. We observed these effects in models that accounted for variance captured by self-report, which suggests that neuroimaging can provide an independent means to validate different accounts of why certain people are prone to self-regulation failure.

Rather than supporting one account exclusively over another, our findings support multiple accounts of self-control failure. For example, cue exposure is a well-known threat to self-regulation (Heatherton & Wagner, 2011), and the NAcc effects we found demonstrate that

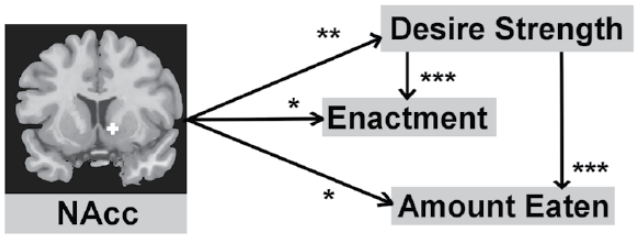

Fig. 2. Summary of effects of activity in the nucleus accumbens (NAcc) region of interest (Montreal Neurological Institute coordinates: 12, 9, −3) on multiple outcomes in the experience-sampling period (from all multilevel regression models). Asterisks indicate the significance of the effects (*$p < .05$, **$p < .01$, ***$p < .001$). See Tables 1 through 3 for all other predictors and effects.

higher reward-related activity during cue exposure is associated with greater likelihood of failure to resist temptations to eat. Other theories propose that the likelihood of self-regulatory failure increases whenever executive functions, supported by various regions of prefrontal cortex (e.g., IFG; Aron, Robbins, & Poldrack, 2004), are not engaged to modulate or dampen the reward value of a tempting stimulus (Heatherton & Wagner, 2011). In accordance with this account, we observed that individuals who showed lower IFG activity associated with response inhibition were prone to give in to their temptations, whereas those with higher IFG activity were more successful in resisting desires to eat.

To conclude, the brain-behavior relationships demonstrated in the present study support and extend previous research on everyday desires (Hofmann et al., 2012) and validate theories of self-control behaviors by incorporating neural markers of these behaviors. Related work has linked reward activity in the NAcc to long-term weight change (e.g., Demos et al., 2012), but the present study applied a brain-as-predictor approach (Berkman & Falk, 2013) to shed light on neural mechanisms of more proximal, short-term eating behaviors, which, over time, may give rise to chronic patterns of overeating and possibly weight gain. Future investigations should explore the extent to which brain systems associated with reward (e.g., NAcc) and self-control (e.g., IFG) can serve as neural markers of other appetitive and addictive behaviors—from binge drinking, to compulsive gambling, to risky sexual behaviors.

Author Contributions

R. B. Lopez, T. F. Heatherton, and W. Hofmann conceived and designed the study. W. Hofmann provided smartphones and software support for experience-sampling data collection and trained R. B. Lopez in all experience-sampling procedures. R. B. Lopez ran participants and gathered the neuroimaging data. R. B. Lopez analyzed the neuroimaging data under the supervision of D. D. Wagner, W. M. Kelley, and T. F. Heatherton. R. B. Lopez and W. Hofmann ran the multilevel models. R. B. Lopez wrote the manuscript, and all the other authors provided comments and edits.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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