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Does impulsivity predict outcome in treatment for binge eating disorder? A multimodal investigation

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Abstract

Multiple dimensions of impulsivity (e.g., affect-driven impulsivity, impulsive inhibition – both general and food-specific, and impulsive decision-making) are associated with binge eating pathology cross-sectionally, yet the literature on whether impulsivity predicts treatment outcome is limited. The present pilot study explored impulsivity-related predictors of 20-week outcome in a small open trial (n=17) of a novel treatment for binge eating disorder. Overall, dimensions of impulsivity related to emotions (i.e., negative urgency) and food cues emerged as predictors of treatment outcomes (i.e., binge eating frequency and global eating pathology as measured by the Eating Disorders Examination), while more general measures of impulsivity were statistically unrelated to global eating pathology or binge frequency. Specifically, those with higher levels of negative urgency at baseline experienced slower and less pronounced benefit from treatment, and those with higher food-specific impulsivity had more severe global eating pathology at baseline that was consistent at post-treatment and follow-up. These preliminary findings suggest that patients high in negative urgency and with poor response inhibition to food cues may benefit from augmentation of existing treatments to achieve optimal outcomes. Future research will benefit from replication with a larger sample, parsing out the role of different dimensions of impulsivity in treatment outcome for eating disorders, and identifying how treatment can be improved to accommodate higher levels of baseline impulsivity.

Binge eating disorder (BED), characterized by recurrent episodes of overeating accompanied by a sense of loss of control (1), is associated with increased risk for obesity and associated medical comorbidities, psychiatric comorbidity, impairment in role functioning, and reduced quality of life (2). While extant treatments for BED produce large improvements in binge eating symptomology, as well as high (i.e., ≥ 50%) rates of remission at follow-up (3), a significant subset of patients remain symptomatic, suggesting a need to identify predictors of poor treatment outcome. A recent review conducted by Vall & Wade (4) identified several robust predictors of response to treatment for BED (e.g., age of onset,

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duration of illness). However, few of these predictors are viable treatment targets (e.g., they reflect historical factors that cannot be changed). Assessing additional individual factors that are associated with the maintenance of binge eating pathology and are potentially amenable to treatment, such as impulsivity, might be one promising avenue for improving treatment response.

Impulsivity and binge eating

In recent years, research investigating impulsivity as a potential risk and/or maintenance factor for binge eating has proliferated. Impulsivity, defined as engagement in behavior with little forethought, is a multidimensional, higher-order construct comprised of several domains. Research has primarily focused on *affect-driven impulsivity*, *impulsive inhibition* (i.e., late-stage inhibition of a prepotent response), and *impulsive decision-making* (i.e., deliberate choice of a smaller short-term over a larger, long-term reward; (5)) as potential maintenance factors of binge eating.

Negative urgency (the tendency towards rash action when emotionally distressed) is one type of *affect-driven impulsivity* that has emerged as a potentially key maintenance factor for bulimia nervosa (6). Negative urgency is prospectively (7, 8), and cross-sectionally (9) associated with binge eating frequency.

A domain of *impulsive inhibition* that has received attention as a potential binge eating maintenance factor is response inhibition (the ability to withhold an already-initiated response). The research examining response inhibition and binge eating is mixed, with several studies detecting response inhibition deficits in those with binge eating (10–12), but others failing to detect such differences (13, 14). However, emerging neuropsychological and neuroimaging evidence suggests that inhibition deficits among BED patients could be food-specific (10, 12, 15).

Delay discounting (the tendency to delay a smaller short-term reward in favor of a larger long-term reward), is a domain of *impulsive decision-making* that has also been proposed as a potential maintenance factor for binge eating. Elevated discounting has been observed in individuals with BED (16), though evidence is mixed when comparing overweight/obese adults with and without BED (11, 16). In summary, although additional research is needed, these results together support a relationship between binge eating and impulsivity.

Impulsivity as a predictor of treatment outcome

Given that impulsivity is associated with binge eating pathology, evaluating whether baseline impulsivity predicts treatment outcome is an important next step. Higher levels of impulsivity may make it harder to implement skills taught in treatment, or to maintain gains when the structure of treatment ends. For example, greater levels of negative urgency may preclude the ability to implement effective problem-solving in the face of negative affect. It is also possible that existing treatments sometimes fail because they do not address impulsivity as a core maintenance factor of eating pathology. If this is the case, alterations to treatment for individuals higher in impulsivity may be warranted.

Although impulsivity predicts treatment outcome in other psychiatric disorders that share similar characteristics with BED, such as substance use disorders and pathological gambling (17), few studies have assessed the predictive value of impulsivity in samples with BED. Within cognitive behavioral treatments for bulimia nervosa, higher impulsivity has predicted treatment dropout (18) and poorer outcomes (19, 20). Of the primary studies that have assessed predictors of outcome in BED (e.g., (21–23)), only one has directly assessed impulsivity, finding that impulsivity was not significantly associated with treatment outcome (21). However, this study only evaluated general impulsivity with a single self-report measure. A separate study utilizing imaging procedures found that cortico-striatal hypofunctioning (a neural pathway associated with poor impulse control) was predictive of higher rates of binge eating after treatment for BED (24), suggesting neuropsychologically-measured impulsivity may be relevant to treatment outcome. More research is thus needed to determine whether impulsivity, as a broad construct or specific subcomponents thereof, is associated with treatment outcome in individuals with BED.

The current study

The present study is the first to explore several impulsivity-related predictors of outcome in a group treatment for BED. We assessed the three dimensions of impulsivity described above, specifically negative urgency (affect-driven impulsivity), response inhibition (impulsive inhibition) and delayed discounting (impulsive decision-making). We examined response inhibition with respect to both neutral (i.e., non-food) and food-specific stimuli, given that deficits in this area may be most pronounced in response to food-specific cues. We hypothesized that higher levels of all dimensions of impulsivity variables would be associated with more frequent binge episodes and higher levels of eating disorder psychopathology at baseline and across treatment. We also hypothesized that greater impulsivity across all domains would be associated with poorer treatment response, including slower and less pronounced reductions in eating disorder symptoms and binge frequency.

METHODS

The present study presents secondary outcomes from a pilot open trial of an acceptance-based group-treatment for BED (for primary outcomes, see Juarascio et al., under review (25)). This treatment combined core behavioral elements (e.g., self-monitoring of food intake, weekly self-weighing) of standard treatments for BED (e.g., (26)), with acceptance-based strategies drawn primarily from Acceptance and Commitment Therapy (ACT; (27)). The acceptance-based strategies included in the treatment sought to target the cognitive and affective maintenance factors of binge eating, and to increase adherence with key behavioral recommendations. The treatment consisted of 10 weekly sessions delivered to groups of five to seven participants across three treatment waves. Each group was led by two doctoral students supervised by a licensed psychologist; sessions were recorded and weekly supervision was provided to ensure treatment fidelity.

Participants

Adult female participants ($N = 19$) were recruited from a large metropolitan area in the United States. Recruitment methods included distribution of flyers to local universities and primary care facilities, targeted emails sent out to university faculty and students, and posting in online forums. A small number of participants ($n = 4$) were referred to the study after being excluded from behavioral weight loss treatment due to binge eating. Participants were eligible for the study if they were between the ages of 18 and 65, and endorsed at least 12 objective binge episodes in the past 3 months, consistent with DSM-5 criteria. Exclusion criteria for the primary outcomes study included severe psychiatric comorbidity (e.g., psychosis) and previous cognitive-behavioral or acceptance-based treatment for BED. For this series of analyses, an additional two participants were excluded due to history of head injury. All data reported below reflect only the 17 participants who were eligible for this series of analyses. Though males were considered eligible for the study, the sample consisted only of female participants, whose ages ranged from 20 to 63 years ($M = 39.59$; $SD = 14.48$). Participants were predominantly White ($n = 9$); the remainder of the sample identified as Hispanic ($n = 3$), Asian-American ($n = 2$), and Black ($n = 2$). BMI ranged from 21.20 to 50.10 kg/m^2 ($M = 33.24$; $SD = 9.29$).

Measures

Eating Disorder Examination Interview 16.0 (EDE)—The Eating Disorder Examination Interview (28) is the gold-standard, semi-structured diagnostic interview for eating disorders, which has strong validity and internal consistency (29). It is widely used for assessment of binge eating and yields a measure of overall symptom severity (Global score). Cronbach's alpha in our sample for the Global score was .82.

Wechsler Test of Adult Reading (WTAR)—We administered the WTAR (30) as an estimate of IQ given its strong association with full scale IQ (30).

UPPS Impulsive Behavior Scale, Negative Urgency Scale (UPPS-NU)—The UPPS (31) is a self-report measure that assesses negative urgency, or the tendency towards rash action in the context of negative affect. Higher scores indicate greater affect-driven impulsivity. The UPPS has good internal consistency (31). Cronbach's alpha for the UPPS-NU subscale in our sample was .81.

The Delis Kaplan Executive Functioning System Color-Word Interference Task (D-KEFS)—The Color-Word Interference task (32) is a modified Stroop task assessing response inhibition in the presence of distractors. Response inhibition (a domain of impulsive inhibition) was operationalized as the time to complete the inhibition trials, with performance on the third trial assessing inhibition-only ("inhibition") and performance on the fourth trial assessing inhibition with set-shifting ("inhibition-switch"). Performance on both the inhibition and inhibition-switch trials represented measures of impulsive inhibition.

Stop Signal Task (SST)—A modified version of a computerized SST also was used to measure impulsive inhibition, specifically inhibitory control in response to both neutral and food-specific stimuli. (See Manasse et al. (33) for more details). In this task, participants

categorize various stimuli on a screen with a keyboard press. The task included two blocks: neutral (e.g., staplers) and food (e.g., pizza) stimuli. In a subset of categorization trials, a stop signal is presented after the stimulus but before the response, which indicates to participants that they are to refrain from responding. As in previous studies, the outcome measure used for the current study was the stop signal reaction time (SSRT), which was calculated by subtracting the average reaction time on "go" trials from the average stop signal delay (34). The SSRT was calculated for each set of stimuli (i.e., neutral and food) for each subject. A smaller SSRT is indicative of greater inhibitory control and a larger SSRT reflects poorer inhibitory control.

Delayed Discounting Task (DDT)—Delayed discounting, a domain of impulsive decision-making, was assessed with the DDT (35), a commonly used computerized monetary discounting task. Participants were asked over several trials to choose between a hypothetical variable monetary amount that could be received immediately and a larger amount to be received after varying delays. Area-under the-curve was calculated from the points at which the subjective value of the delayed reward was equal to the amount of the immediate reward (36). Greater area-under-the-curve values indicated less discounting of delayed rewards.

Participant Assessment

Participants completed assessments at four time points: baseline, mid-treatment (5 weeks), end-of-treatment (10 weeks), and 3-month follow-up (22 weeks). The EDE was administered at all time points. The baseline assessment also consisted of the WTAR and all measures of impulsivity. For consistency of measurement across time points, all binge frequency values reported reflect the total number of objective binge episodes experienced in the four weeks prior to a given assessment point.

Statistical Analyses

Dependent variables were binge eating frequency and EDE Global Score (i.e., overall eating pathology). Inferential analyses were conducted in R (v.3.1.2) using the *lme4* packages for generalized linear mixed/multilevel modeling. Multilevel modeling was selected to allow for increased power, ability to detect nonlinear trends across multiple time points, and inclusion of all study participants, regardless of missing data at particular time points (37, 38). These models also allow for modeling of both fixed effects (interpreted as general trends averaged across participants) and random effects (interpreted as the extent to which each individual participant deviates from the average trend across time)(38, 39).

Consistent with methods from previous outcome studies (40), natural logarithmic transformation of binge frequency data was used to normalize the distribution. Prior to transformation, *z*-scores for skewness and kurtosis were significantly different from zero at the $p < .01$ level (i.e., $z \geq 2.38$) at both 10-week and 22-week assessment points, indicating substantial deviation from a normal distribution. After transformation, z-scores for skewness and kurtosis statistics for binge frequency at each respective time point fell within acceptable ranges (i.e., no longer significantly different from zero at the $p < .01$ level). For all models, observations of the outcome variables across time (Level 1) were nested within individual

participants (Level 2). Likelihood ratio tests (LRTs) were conducted to determine whether inclusion of linear and quadratic random effects significantly improved model fit, using the criterion $p \leq .05$ for the χ^2 statistic obtained from model comparison. Inclusion of random slopes improved model fit for binge frequency only (LRT test of random linear time effect on binge frequency: $p = .04$). EDE Global models include random intercepts and fixed effects of time, since addition of random slopes across time did not significantly improve model fit ($p = .47$). Linear, quadratic, and cubic fixed effects of time were tested for each of the outcome variables (binge frequency and EDE Global scores). Polynomial fixed effects of time were included in the respective models if they accounted for a significant amount of overall variance (i.e., $p < .05$ using the approximate normal z-distribution).

Separate models were used to assess the effects of each impulsivity measure on treatment outcome. Baseline impulsivity measures (negative urgency, delayed discounting, and measures of inhibition) were centered and added as fixed-effect covariates. To assess the effect of these measures on overall treatment response (i.e., rate of symptom reduction), interaction terms were added to each model. Interactions between impulsivity measures and both the linear and quadratic time terms were introduced in a stepwise manner, to assess whether impulsivity affects overall rate of treatment improvement (linear change over time), or rate of change in treatment response (e.g., earlier slowing of symptom reduction or difficulty maintaining gains; quadratic time). Estimated FSIQ based on performance on the WTAR was added as a covariate to models that included measures of neurocognitive performance.

RESULTS

Overall Treatment Response

As described more fully in Juarascio et al. (25), EDE Global scores and binge frequency were at clinical levels among participants at baseline. Participants generally experienced large reductions in binge frequency and eating pathology at post-treatment, which were largely maintained at follow-up (see Table 1).

Results from mixed effects linear regression indicated that symptom change was non-linear and fluctuated over time. A significant cubic effect of time was detected for binge frequency ($b = .002$, $SE_b = .001$, $p < .001$), but not for eating disorder psychopathology. This indicates that participants tended to experience slight, temporary increases in binge frequency after the rapid reductions made toward the beginning of treatment. However, the cubic effect also signifies that this slight post-treatment increase tapered off over time. Observed data indicate the increase in frequency occurred primarily at post-treatment and tapered off by follow-up (see Figure 1). Significant linear and quadratic fixed effects of time were detected for both binge frequency (linear: $b = -.80$, $SE_b = .11$, $p < .001$; quadratic: $b = .08$, $SE_b = .02$, $p < .001$) and EDE Global scores (linear: $b = -.16$, $SE_b = .03$, $p < .001$; quadratic: $b = .01$, $SE_b = 0.001$, $p < .001$). The presence of a quadratic effect of time as the highest-order effect on eating disorder psychopathology reflects the significant, initially rapid reductions in eating disorder symptoms that participants experienced, but also indicates that the rate of improvement slowed over time. Group means at each time point (described in Table 1) reflect the patterns observed in the linear models.

Effects of Impulsivity on Binge Frequency

Consistent with hypotheses, negative urgency was found to significantly interact with the linear fixed effect of time ($b = .05$, $SE_b = .02$, $p = .03$), indicating that individuals with higher levels of urgency experienced more gradual reductions in binge frequency during and after treatment. The effect of urgency on baseline binge frequency was only marginally significant ($b = -.04$, $SE_b = .21$, $p = .05$), but was in the opposite direction of that expected. The negative coefficient indicated that participants with greater negative urgency had marginally fewer binge episodes at baseline. None of the additional, behavioral measures of impulsivity (delay discounting, inhibition, inhibition-switch, food-related inhibitory control, general inhibitory control) emerged as significant moderators (ps .31 – .68) of the influence of time and binge eating frequency. Similarly, none of the behavioral measures corresponded to greater baseline binge frequency (ps .12 – .99).

Effects of Impulsivity on Global Eating Pathology

No significant effect of negative urgency on baseline eating disorder pathology was detected ($b = .16$, $SE_b = .28$, $p = .55$), but the moderating effect was marginally significant ($b = .02$, $SE_b = .01$, $p = .09$), such that participants with higher levels of negative urgency experienced smaller reductions in EDE Global scores, and/or had more difficulty sustaining symptom improvement over time (see Figure 2).

The only dimension of response inhibition that emerged as a significant predictor of baseline global eating pathology was food-specific inhibitory control (measured by the SST; $b = .003$, $SE_b = .001$, $p = .02$; see Figure 3); however, this variable did not significantly impact symptom change over time ($b = .00007$, $SE_b = .00006$, $p = .27$). The remaining general dimensions of response inhibition (inhibition, inhibition-switch, and inhibitory control in response to neutral stimuli) were not significantly predictive of baseline pathology (ps .35 – .42), and none significantly moderated treatment response (ps .70 – .89). No significant effects of delay discounting on either baseline pathology or change in treatment response over time were detected (ps = .17 and .36 for baseline and moderating effect over time, respectively).

DISCUSSION

This small exploratory study is among the first to examine several indices of impulsivity (assessed through both self-report and behavioral tasks) as predictors of treatment outcome for BED. Baseline negative urgency significantly impacted symptom change over time, such that individuals with greater negative urgency experienced smaller reductions in binge eating frequency and global eating pathology during and after treatment. These findings are consistent with a robust literature linking negative urgency and binge eating pathology (41); however, to our knowledge, this is the first study to examine whether negative urgency is associated with treatment outcome. These findings indicate that, as expected, a greater perceived tendency to act rashly in response to negative affect at baseline is associated with more modest improvements during treatment.

No other dimension of impulsivity emerged as a statistically significant predictor of reduction in binge frequency or global eating pathology over time, although this lack of detection of effects should be considered in the context of a small sample. There was a main effect of baseline food-related response inhibition on greater global eating pathology throughout treatment, such that those with poorer food-specific inhibitory control displayed more global eating pathology at baseline, and that this increased severity was present at a consistent magnitude across the treatment and follow-up period. No other domains of response inhibition emerged as statistically significant predictors. This pattern of results is consistent with previous research indicating that food-specific inhibition deficits are more pronounced (and perhaps more detectable with limited power) in BED than are general ones (12).

Baseline delayed discounting did not significantly predict global eating pathology or binge frequency. Extant research is mixed in supporting the role of delayed discounting in the maintenance of BED (11, 16). If delayed discounting is not consistently related to the maintenance of binge eating pathology, it may not be predictive of treatment outcome. It is also possible that the monetary stimuli in the task may not be as relevant to individuals with BED as other stimuli (e.g., food), especially in the context of treatment outcome.

It is somewhat counterintuitive that only negative urgency was associated with the rate of improvement in binge episodes over time. Although results should be interpreted in light of the small sample used, one might expect that behaviorally-measured impulsivity would relate most closely to overt behavioral outcomes (i.e., binge frequency) rather than to overall pathology. However, recent literature has suggested that illness severity does not necessarily correspond with binge eating size or frequency, but rather perceived loss of control (42). It is possible that impulsivity is associated with loss of control (regardless of frequency of the behavior), and thus in future research it may be beneficial to measure loss of control dimensionally. As an alternative explanation, the relation between self-report and behavioral measures of impulsivity is small, and it has thus been suggested that behavioral measures (such as the DDT and SST) may only capture and represent state (versus trait) tendencies that may be more related to current, rather than future, behavior (43). A second counterintuitive finding is the marginally significant negative association between negative urgency and binge eating at baseline. Two potential explanations for this finding are that (1) binge eating frequency (as opposed to another measure, such as dimensional loss of control) may not be a valid proxy for illness severity within a BED sample (42) and (2) that the relation between negative urgency and binge frequency may be different for those who are treatment-seeking. However, future research and replication is necessary to support these claims.

Clinical Implications

The results of the current study hold several potential clinical implications. Although our findings need replication with larger samples, it is possible that patients with greater negative urgency could benefit from greater explicit and repeated practice with responding to negative emotions in healthier ways during treatment to achieve optimum gains. Although the intervention employed in the current study attempted to improve emotion regulation

(along with several other primary treatment mechanisms, see Juarascio et al., under review (25)), perhaps an explicit focus on developing skills for withholding impulsive responses in the context of negative affect is necessary for those high in negative urgency. The current treatment was also short-term in nature; it may be that individuals high in negative urgency need a longer treatment approach to provide time to promote effective skills acquisition.

Although food-specific inhibitory control was not found to significantly impact symptom change over time in the current study, those with poorer food-specific inhibitory control displayed more global eating pathology at baseline, and this increased severity was present across the treatment and follow-up period. Future research and replication with larger samples is needed; however, findings suggest that individuals with poorer *food-specific* response inhibition may benefit from interventions that target the ability to withhold a prepotent response towards food. For example, recent research has suggested that training inhibitory control is a promising venue for treating eating disorders (44), and researchers have begun to develop paradigms for training food-specific inhibitory control (e.g., (45)).

Strengths and limitations

The current study featured several notable strengths. First, consistent with movements towards using multimodal measurement, we used several measures of impulsivity, a number of which were behavioral measures. Thus, the present study represents a novel addition to the extant literature. We also utilized statistical analysis techniques that maximized power given our small sample and that were appropriate for longitudinal data with multiple time points. Lastly, we utilized multiple assessment points, including a 3-month follow-up, which allowed our team to assess the influence of impulsivity beyond the treatment period.

Despite these strengths, several limitations should be considered. Most importantly, our small sample size may have limited our ability to detect additional effects, and may limit generalizability. For example, it is possible that other more general domains of impulsivity do impact treatment outcome, but we were unable to detect these effects. We were also unable to test predictors in the same model due to lack of power. In addition, utilizing small sample sizes in multilevel modeling analyses could result in unreliable model estimation. In the present study, it is therefore important to avoid overemphasizing the meaning of statistically significant versus null effects in the present study. Instead, we contend that the emergence of some statistically significant findings, despite limited power, provides cause for further investigation and future study.

The absence of a control group also limits our ability to attribute changes over time solely to treatment effects. Additionally, the treatment implemented in the current study was a treatment that incorporated novel components; thus, it is unclear whether the same patterns of results would hold with a different therapeutic approach (e.g., CBT-E). Finally, given the paucity of research in this area and the novelty of the present study's aims, we did not control for experiment-wise error. For all of the aforementioned reasons, caution is thus warranted when interpreting results.

Future directions

Future research should aim to replicate these initial findings with larger samples with adequate statistical power. Our sample was also limited to those with BED; research should be extended to transdiagnostic binge eating samples (e.g., bulimia). Additionally, future research should examine whether change in impulsivity mediates change in binge frequency and global eating pathology during treatment. Comparative trials should also examine whether the predictive and moderating effects of impulsivity are specific to certain treatments. Finally, given the interactive role of other factors (e.g., affectivity, food environment) with impulsivity in individuals with eating disorders (8), future research should examine whether such factors moderate the effect of impulsivity on treatment outcome. Such research could lead to the tailoring of interventions based on baseline characteristics, potentially improving outcomes.

Conclusion

Although a growing body of research highlights impulsivity as a maintenance factor in binge eating pathology, examination of the role of impulsivity in treatment outcomes has been limited. This study provides initial evidence that higher levels of impulsivity, specifically in the context of negative affect and food cues, may relate to levels of and changes in eating pathology across treatment. As such, future research will benefit from parsing out the role of different dimensions of impulsivity in outcome for eating disorders.

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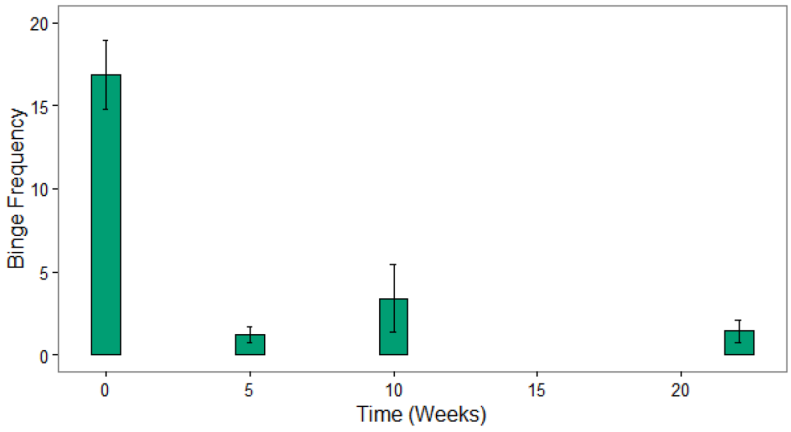

Figure 1.
Observed binge frequency at baseline, mid-treatment, end-of-treatment, and follow-up

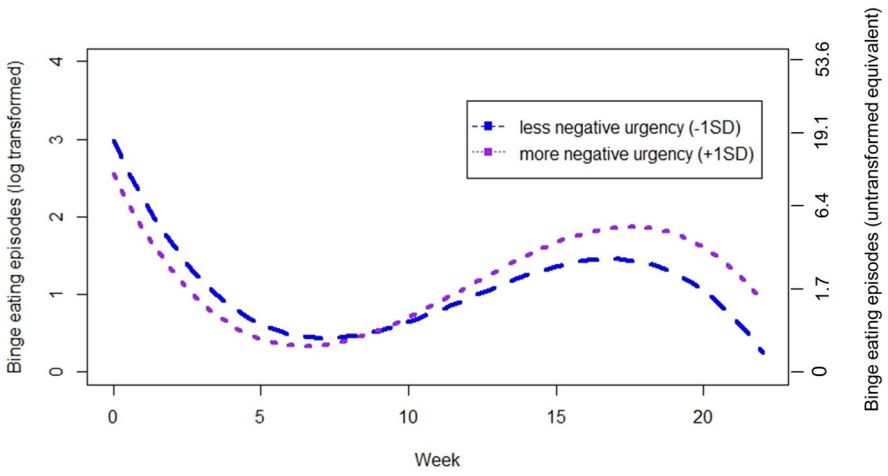

Figure 2. Predicted binge eating frequency at higher and lower levels of negative urgency

Note: Binge episodes were transformed onto a logarithmic scale, thus the y-axis on the right represents the conversion of the log-transformed values to the frequency of binge episodes equivalent.

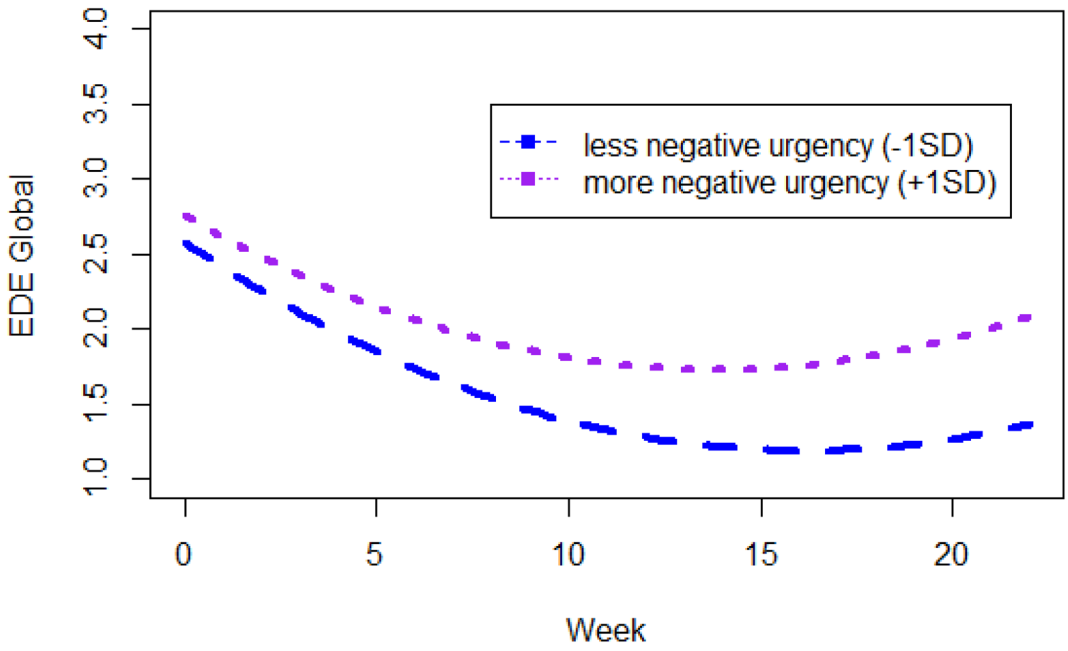

Figure 3.
Predicted Eating Disorder Examination Global values at higher and lower levels of negative urgency

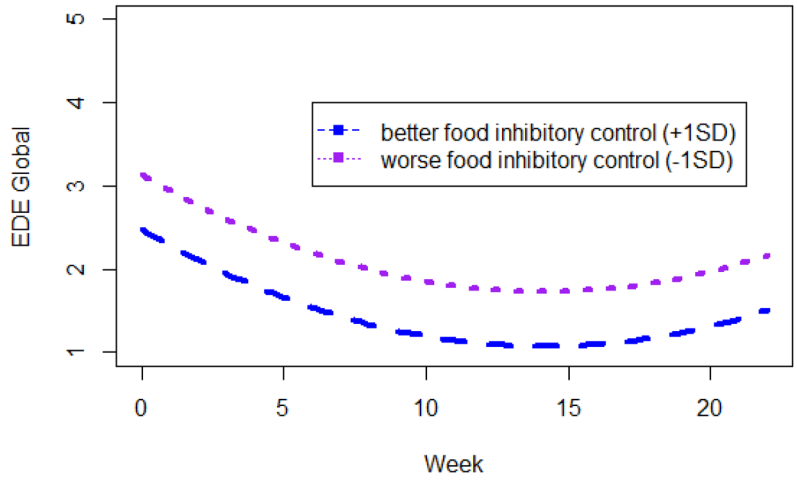

Figure 4.
Predicted Eating Disorder Examination Global values at higher and lower food-specific stop-signal reaction time

Table 1

Change in ED Psychopathology and Binge Frequency During Treatment

Outcome Measure	Baseline	Mid-Treatment	End-of-Treatment	3-Month Follow-Up
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
EDE Global	2.67 (0.81)	1.74 (0.53)	1.60 (0.63)	1.58 (0.98)
Binge Frequency[a]	16.82 (8.62)	1.35 (2.00)	3.29 (7.90)	1.33 (2.47)

Note. n's range from 15 to 17 due to occasional missing data.

[a]Over the past 4 weeks