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# Change in impulsivity is prospectively associated with treatment outcomes for binge-eating disorder

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## Original Article

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## Abstract

**Background.** Impulsivity may be a process underlying binge-eating disorder (BED) psychopathology and its treatment. This study examined change in impulsivity during cognitive-behavioral therapy (CBT) and/or pharmacological treatment for BED and associations with treatment outcomes.

**Methods.** In total, 108 patients with BED ($N_{FEMALE}$ = 84) in a randomized placebo-controlled clinical trial evaluating the efficacy of CBT and/or fluoxetine were assessed before treatment, monthly throughout treatment, at post-treatment (16 weeks), and at 12-month follow-up after completing treatment. Patients completed established measures of impulsivity, eating-disorder psychopathology, and depression, and were measured for height and weight [to calculate body mass index (BMI)] during repeated assessments by trained/monitored doctoral research-clinicians. Mixed-effects models using all available data examined changes in impulsivity and the association of rapid and overall changes in impulsivity on treatment outcomes. Exploratory analyses examined whether baseline impulsivity predicted/moderated outcomes.

**Results.** Impulsivity declined significantly throughout treatment and follow-up across treatment groups. Rapid change in impulsivity and overall change in impulsivity during treatment were significantly associated with reductions in eating-disorder psychopathology, depression scores, and BMI during treatment and at post-treatment. Overall change in impulsivity during treatment was associated with subsequent reductions in depression scores at 12-month follow-up. Baseline impulsivity did not moderate/predict eating-disorder outcomes or BMI but did predict change in depression scores.

**Conclusions.** Rapid and overall reductions in impulsivity during treatment were associated with improvements in specific eating-disorder psychopathology and associated general outcomes. These effects were found for both CBT and pharmacological treatment for BED. Change in impulsivity may be an important process prospectively related to treatment outcome.

Binge-eating disorder (BED), the most prevalent eating disorder among adults in the United States (prevalence rate: ~1.0%; Udo and Grilo, 2018), is characterized by recurrent episodes of binge eating (consuming an objectively large amount of food while experiencing a subjective sense of loss of control), with marked distress and without regular, extreme weight-compensatory behaviors (APA, 2013). BED is distributed across sex, ethnic/racial, and age groups and is associated strongly with obesity and with heightened risk for psychiatric/medical comorbidity (Udo & Grilo, 2018, 2019). Although psychological and pharmacological treatments for BED are effective at reducing binge eating (Grilo, 2017; Hilbert et al., 2019), approximately 50% of patients do not achieve abstinence (Linardon, 2018). Accordingly, research on predictors, moderators, and processes of treatment change is essential both to improve treatments and to refine conceptual models of the psychopathology of BED (Kraemer, Wilson, Fairburn, & Agras, 2002).

A candidate process that may influence treatment outcomes for BED is impulsivity. Impulsivity is a multi-dimensional construct reflecting poor reward-related decision-making and a propensity to engage in rash or reward-seeking behavior (Dawe & Loxton, 2004). Impulse-control difficulties have transdiagnostic neurobiological correlates (Bari & Robbins, 2013) and are related to severity of psychopathology (Berg, Latzman, Bliswise, & Lilenfeld, 2015), including eating psychopathology (Waxman, 2009). Individuals with BED have heightened impulsivity in the context of food (Giel, Teufel, Junne, Zipfel, & Schag, 2017) and in general (Boswell & Grilo, 2020; Boswell, Potenza, & Grilo, 2021; Kober & Boswell, 2018). To determine whether impulsivity might be a target for BED treatment, it will be important to establish whether impulsivity is impacted by treatments for BED and whether changes in impulsivity are associated with treatment outcomes.



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To date, very little research has examined the relationship between impulsivity and BED treatment outcomes. Greater impulsivity is associated with poorer treatment outcomes for substance use disorders (Hershberger, Um, & Cyders, 2017), gambling (Mallorqui-Bague et al., 2018), and bariatric surgery (Yeo et al., 2020); findings to date for BED are mixed. In two small pilot studies, baseline self-reported behavioral and brain-based measures of impulsivity were associated with poorer response to BED treatment (Balodis et al., 2014; Manasse et al., 2016). Two other studies, however, reported null associations between baseline impulsivity and treatment outcomes (Anderson et al., 2020; Castellini et al., 2012). Overall, the limited research to date has identified very few reliable patient predictors of outcome and even fewer findings about moderators (Linardon, de la Piedad Garcia, & Brennan, 2017). Identifying patient characteristics at baseline that predict or moderate treatment responses (e.g. for whom specific treatments work better) could inform rational decision-making about prescribing treatments (Grilo, 2017; Wilson, Grilo, & Vitousek, 2007).

Although historically conceptualized as a trait measure and thought to be relatively stable over time, impulsivity changes in both prospective naturalistic studies (McGlashan et al., 2005) and during treatment, including for substance use disorders and obesity (Littlefield et al., 2015; Ross et al., 2020). Because greater impulsivity may interfere with treatment outcomes, reductions in impulsivity during treatment could signal better prognosis. To date, in the sole study with BED that has examined change in impulsivity during pharmacological treatment, lisdexamfetamine dimesylate, a pro-drug-stimulant medication that improves executive functioning, was associated with reduced self-reported impulsivity (McElroy et al., 2016). In contrast, a pilot study of an impulsivity-focused cognitive-behavioral therapy (CBT) for BED did not produce significant changes in impulsivity (Schag et al., 2019). Despite interest in the role of impulsivity in maintaining eating disorders (Waxman, 2009), no prior research has examined whether *changes* in impulsivity during treatment predict outcomes for BED (or any eating disorders).

Timing of reductions in impulsivity during treatment might also influence outcomes. Rapid response to treatment is the strongest and most well-established mediator of treatment outcomes across eating disorders (Linardon, Brennan, & de la Piedad Garcia, 2016). In BED, rapid reduction in binge eating (i.e. during the first month of treatment) is reliably associated with better outcomes across psychological and pharmacological treatments (Grilo & Masheb, 2007; Grilo, Masheb, & Wilson, 2006; Grilo, White, Masheb, & Gueorguieva, 2015; Grilo, White, Wilson, Gueorguieva, & Masheb, 2012; Masheb & Grilo, 2007). Prior BED research focused exclusively on rapid reductions in binge eating rather than early/rapid changes in other potentially clinically relevant variables such as impulsivity. Recent research with bulimia nervosa found that rapid improvements in emotion regulation predicted outcomes (MacDonald & Trottier, 2019; MacDonald, Trottier, & Olmsted, 2017; Peterson et al., 2017).

Examining change in impulsivity as a prospective indicator of treatment outcomes could highlight a transdiagnostic process underlying BED psychopathology and its treatment. This study examined changes in impulsivity during and after treatment with CBT and fluoxetine, alone and together, for BED. Examining changes in impulsivity in these treatments seems indicated for the following reasons. CBT, which has well-established effectiveness for BED (Grilo, 2017), includes structured treatment components (e.g. self-monitoring, regular eating, and cognitive reappraisal) that target/reduce binge eating. These CBT–BED-based skills, which require practice in planning, problem-solving, and cognitive flexibility, may indirectly generalize to broader behavioral and cognitive aspects of impulsivity. Fluoxetine modulates serotonergic systems implicated in impulsive responding, which is hypothesized to be broadly related to general psychopathology (Carver, Johnson, & Joormann, 2008; Coccaro & Kavoussi, 1997; Walderhaug et al., 2002). However, although fluoxetine has demonstrated effectiveness for depression and bulimia nervosa (Hagan & Walsh, 2021), its effects on impulsivity are less certain (Boswell et al., 2021).

Thus, the specific aims were: (1) to determine whether impulsivity changes during treatment for BED and during follow-up; and (2) to determine whether changes in impulsivity are related to treatment outcomes for BED, including: (a) does rapid change in impulsivity prospectively influence treatment outcomes?; and (b) does overall change in impulsivity during treatment influence outcomes? In order to examine the specificity of the effects of changes in impulsivity and include secondary outcomes from existing BED randomized controlled trials (RCTs), we characterized CBT and fluoxetine treatment outcomes broadly by including eating-disorder symptoms and features (i.e. binge-eating episodes and global eating-disorder psychopathology), depression scores, and body mass index (BMI) as outcome variables. In doing so, we addressed an additional specific aim: (3) to examine whether the association of change in impulsivity during treatment for BED is specific to eating psychopathology or also related to associated clinical outcomes. Finally, we examined an exploratory aim: (4) is baseline impulsivity a predictor or moderator of treatment outcomes (Kraemer et al., 2002)?

## Methods

### Participants

Participants were 108 adult patients who met *DSM-IV*-defined BED (APA, 1994), were ⩾100% ideal weight, and were consecutively enrolled in a randomized double-blind, placebo-controlled study examining fluoxetine and CBT, alone and in combination. Detailed study descriptions and outcomes at post-treatment (Grilo, Masheb, & Wilson, 2005) and 12-month follow-up (Grilo, Crosby, Wilson, & Masheb, 2012) have been reported and are therefore summarized briefly below. Study was approved by the Yale Institutional Review Board and all participants provided written informed consent.

Exclusion criteria included any concurrent treatment for eating, weight, or psychiatric problems, current medical conditions that influence eating/weight, current severe psychiatric conditions precluding accurate assessment or requiring alternative treatment, or current pregnancy/breastfeeding. Participants' mean BMI was 36.3 (S.D. = 7.9) and mean age was 44.0 (range: 21–59, S.D. = 8.6). The participant group was 89% White, 78% female, and 87% attended/finished college.

### Randomization and treatment

Participants were randomized to one of four treatment conditions in a balanced 2 × 2 factorial design for 16 weeks: (a) fluoxetine (60 mg/day), (b) placebo, (c) CBT + fluoxetine (60 mg/day), and (d) CBT + placebo. Randomization followed the consecutive order in which participants completed assessments and were deemed eligible to enroll. Computer-generated randomization schedule was created

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without stratification in blocks of eight to yield approximately equal allocations across conditions; the schedule was implemented by a research pharmacist separate from the blinded investigators/clinicians. The study medications were given in identical appearing capsules and delivered with minimal clinical management by faculty-level psychiatrists focused on medication regimen and adherence/safety without additional psychotherapeutic interventions. CBT was delivered following manualized protocols in weekly individual sessions by doctoral research-clinicians monitored for adherence. First stage of CBT (seven sessions) focused on psychoeducation about binge eating and specific behavioral strategies (e.g. self-monitoring, problem-solving, and regular-eating patterning) to help reduce binge-eating while normalizing eating. Middle stage (six sessions) included cognitive restructuring (of eating and weight/shape thoughts that maintain binge eating) while continuing to normalize eating patterns. Final stage (three sessions) focused on maintenance and relapse prevention. Independent (blinded) assessments were performed at baseline, monthly throughout treatment, post-treatment, and 12-month follow-up after completing and discontinuing treatments. Patients in placebo-only condition were not followed after posttreatment because they were offered treatment at that time (Grilo, Crosby, et al., 2012).

## Measures

### Diagnostic assessment

BED was determined via Structured Clinical Interview for *DSM-IV*-Axis I Disorders (SCID-I/P; First, Gibbon, Spitzer, Williams, & Benjamin, 1996) and confirmed with Eating Disorder Examination interview (EDE; Fairburn & Cooper, 1993). These diagnostic interviews were administered by doctoral-level research clinicians; interrater reliability was excellent (EDE: Spearman rho = 0.87–0.99; SCID: kappa for BED = 1.0).

### Baseline and repeated measures

Measures were administered at baseline, monthly during treatment, end of treatment (post-treatment), and 12-month follow-up after completing treatments.

*Impulsivity Control Scale* (ICS; Grosz et al., 1994; Plutchik & van Praag, 1989). ICS is a 15-item self-report measure of impulsivity that conceptually is related to UPPS-P dimensions of positive/negative urgency and sensation-seeking (Verdejo-Garcia, Lozano, Moya, Alcazar, & Perez-Garcia, 2010). ICS has well-established psychometric properties (Grosz et al., 1994); in patients with BED, ICS shows concurrent validity with diagnostic interview-based assessment of impulsivity (Boswell & Grilo, 2020). In this study, ICS had good internal consistency [alpha coefficient ($\alpha$) = 0.75].

*Eating Disorder Examination-Questionnaire* (EDE-Q; Fairburn & Beglin, 1993). EDE-Q, the self-report version of the EDE interview, assesses the behavioral/cognitive features of eating-disorder psychopathology. EDE-Q evaluates the frequency of binge-eating episodes and generates a global total score reflecting overall eating-disorder psychopathology severity. EDE-Q converges well with the EDE interview (Grilo, Masheb, & Wilson, 2001a, 2001b) and has excellent reliability in BED samples (Berg, Peterson, Frazier, & Crow, 2012; Reas, Grilo, & Masheb, 2006). In this study, EDE-Q global scale had good internal consistency ($\alpha = 0.80$).

*Beck Depression Inventory* (BDI; Beck, Steer, & Garbin, 1998). BDI is a 21-item measure of depression symptoms that is psychometrically well-established (Beck et al., 1998). In this study, the BDI had excellent internal consistency ($\alpha = 0.90$).

*Body mass index*. BMI (kg/m$^2$) was calculated from in-person measured height (at baseline) and weight at baseline and every session throughout the study 12-month follow-up.

## Statistical analyses

Mixed effects models were fit (SAS 9.4) to evaluate changes in impulsivity and associations of those changes in impulsivity with outcomes during treatment and follow-up. Analyses included all participants randomized to treatments with all repeated measures for each individual included in mixed-effects models.

### Predictor and outcome variables

Descriptive statistics and distributions were evaluated prior to analyses; no transformations were applied. Predictor (i.e. impulsivity) and outcome variables (i.e. binge-eating episodes, global eating-disorder psychopathology, depression scores, and BMI) were measured continuously. Rapid change in impulsivity was measured via the ICS change score following 1 month of treatment (baseline–month 1). In the case of missing month 1 ICS data, month 2 data were used ($N = 1$). Overall change in impulsivity was measured via the ICS change score during treatment (baseline–post). ICS predictor variables were mean-centered. If post data were missing, post value was imputed with last observation carried forward (20% imputed). No imputation was conducted for outcome variables because there was minimal missing outcome data and because mixed models provide unbiased and efficient estimates when data are missing at random. All available data from all available timepoints were included in analyses.

### Change in impulsivity during treatment and follow-up

Change in impulsivity over time was investigated using two mixed models with measurements during treatment in one model (model 1A) and measurements during follow-up in another (model 1B). Fixed effects were time, treatment group, and the interaction. Because individuals in the placebo-only group did not complete follow-up assessments, there were four groups during treatment (fluoxetine, placebo, CBT + fluoxetine, and CBT + placebo) and three groups during follow-up (fluoxetine, CBT + fluoxetine, and CBT + placebo). The timepoints were baseline, monthly during treatment, and post-treatment (i.e. at 4 months). In the follow-up analyses, timepoints were baseline, post-treatment, and 12-month follow-up. For each model, the best-fitting variance–covariance structure was selected based on Akaike's information criterion (AIC). Least square means were compared in post-hoc tests to explain significant effects in the models.

### Rapid change in impulsivity and outcomes

The effects of rapid change in impulsivity on outcomes during treatment and follow-up were tested via two sets of mixed models (referred to as models 2A and 2B, respectively). Model 2A included during treatment timepoints/groups. Model 2B included follow-up timepoints (post and 12-month follow-up) and groups. Outcome variables were included as change scores from baseline by timepoint (baseline–subsequent timepoint). Fixed effects were ICS change score, time, treatment group, and all interactions. The same model-selection process as described above was used.

### Change in impulsivity during treatment and outcomes

The influence of change in impulsivity during treatment on outcomes was investigated using two separate types of models (model

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3A during treatment and model 3B during follow-up). For model 3A, change in impulsivity was computed via the overall ICS change score (baseline–post) rather than through repeated measures (i.e. effect of time) because of overlapping time-course for predictor and outcome variables. We used a linear model with the ICS change score, treatment group, and their interaction as predictors and change in the outcome from baseline to post as response. Model 3B was mixed models that investigated change in outcomes from baseline during follow-up (post and 12-month follow-up). Model 3B included fixed effects of ICS change score, time, treatment group, and all interactions. Model selection was based on the AIC.

#### *Exploratory analyses: baseline impulsivity*

The association of baseline impulsivity on treatment outcome was examined using two sets of mixed models (model 4A during treatment; model 4B during follow-up). Outcome variables were change scores from baseline by timepoint. Fixed effects were baseline impulsivity, time, treatment group, and interactions. The interaction between baseline impulsivity and treatment group was used to test potential moderation effects. Model selection was based on the AIC.

## Results

### *Treatment completion and primary outcomes*

Overall, 80% of randomized patients completed treatment (Grilo et al., 2005). Of patients who completed treatment (excluding placebo-only condition), 71.6% completed 12-month follow-up (Grilo, Crosby, et al., 2012). After treatment, intent-to-treat binge-eating remission rates were: 22% (fluoxetine), 26% (placebo), 50% (CBT + fluoxetine), and 61% (CBT + placebo; Grilo et al., 2005). At 12-month follow-up, remission rates were 3.7% for fluoxetine-only, 26.9% for CBT + fluoxetine, and 35.7% for CBT + placebo (Grilo, Crosby, et al., 2012).

### *Change in impulsivity during treatment and follow-up*

Figure 1 shows changes in impulsivity over time across the different treatment conditions. Mixed effects models revealed overall significant decreases from baseline to post-treatment across conditions (*Model 1A*: Time: $F_{(4,86.9)} = 9.45$, $p < 0.001$), but no significant differences between the specific treatments (Group: Group × Time: $ps > 0.34$). Analyses revealed a significant main effect of time during follow-up (*Model 1B*: Time: $F_{(2,59.2)} = 13.10$, $p < 0.001$), but no effects of treatment group or interactions (Group: Group × Time: $ps > 0.65$). Across groups, post-hoc analyses revealed significant decreases in impulsivity during treatment and significant increases during follow-up. After Tukey–Kramer adjustment, differences between baseline impulsivity and all subsequent timepoints remained significant (online Supplementary Table S1).

### *Rapid change in impulsivity and outcomes*

#### *Binge eating frequency*

Mixed effects models revealed a significant three-way interaction between impulsivity, group, and time (*Model 2A*: ICS × Group × Time: $F_{(9,208)} = 1.92$, $p = 0.05$) and a significant interaction between ICS and time (*Model 2A*: ICS × Time: $F_{(3,208)} = 3.07$, $p = 0.03$). However, none of the slopes describing the relationship between impulsivity and binge eating by treatment group and timepoint or by timepoint were statistically significant ($ps > 0.06$).

#### *Eating-disorder psychopathology*

Mixed-model analyses revealed that rapid reduction in impulsivity was significantly associated with subsequent reduction in eating-disorder psychopathology during treatment (*Model 2A*: ICS: $F_{(1,92.3)} = 5.64$, $p = 0.02$), but not during follow-up (*Model 2B*: ICS: $F_{(1,62.3)} = 0.04$, $p = 0.84$; Fig. 2). During follow-up, analyses revealed an interaction between ICS and time, but the slopes were not significantly different from zero in post-hoc tests (*Model 2B*: ICS × Time: $F_{(1,51.1)} = 10.25$, $p = 0.002$; online Supplementary Table S2).

#### *Depression scores*

In mixed-model analyses, rapid reduction in impulsivity was significantly associated with subsequent reduction in depression scores during treatment; these relationships were non-significant during follow-up (*Model 2A*: ICS: $F_{(1,90.7)} = 11.13$, $p = 0.001$; *Model 2B:* ICS: $F_{(1,64)} = 3.81$, $p = 0.055$; Fig. 2). Analyses also showed significant interactions between ICS, group, and time during treatment but not follow-up (ICS × Group × Time: $F_{(9,77.5)} = 2.95$, $p = 0.005$; *Model 2B*: ICS × Group × Time: $F_{(2,51.2)} = 2.94$, $p = 0.06$; other $ps > 0.06$). Post-hoc tests revealed that the relationship between rapid reduction in impulsivity and subsequent reduction in depression scores was significant in the fluoxetine and CBT + fluoxetine groups. There were no significant differences between slopes by treatment condition ($ps > 0.17$; online Supplementary Table S3).

#### *BMI*

In mixed-model analyses, rapid reduction in impulsivity was associated with subsequent reduction in BMI during treatment (*Model 2A*: ICS: $F_{(1,88.2)} = 7.38$, $p = 0.008$) but not during follow-up (*Model 2B:* ICS: $F_{(1,59.3)} = 1.06$, $p = 0.31$; Fig. 2).

### *Change in impulsivity during treatment and outcomes*

#### *Binge-eating frequency*

There were no main or interactive effects of change in impulsivity during treatment on change in binge eating during treatment or follow-up (ICS: $ps > 0.45$).

#### *Eating-disorder psychopathology*

Reductions in impulsivity during treatment were significantly associated with reductions in eating-disorder psychopathology during treatment and follow-up (*Model 3A*: ICS: $b = 2.38$, $p = 0.03$; *Model 3B*: $F_{(1,63.5)} = 5.38$, $p = 0.02$; Fig. 3). The analyses revealed a significant interaction between ICS and time (*Model 3B*: ICS × Time: $F_{(1,54.4)} = 11.77$, $p = 0.001$), but no other interactions ($ps > 0.32$). This reflected a relationship between reduction in impulsivity during treatment and reductions in eating-disorder psychopathology at post-treatment, but not follow-up (post: $b = 2.51$, $t_{(62.8)} = 4.09$, $p < 0.001$; 12-month follow-up: $p = 0.79$).

#### *Depression scores*

Reductions in impulsivity during treatment were associated with reductions in depression scores at post-treatment and at 12-month follow-up (*Model 3A*: ICS: $b = 1.11$, $p = 0.004$; *Model 3B*: ICS: $F_{(1,65.1)} = 14.48$, $p < 0.001$; Fig. 3; online Supplementary Table S4).

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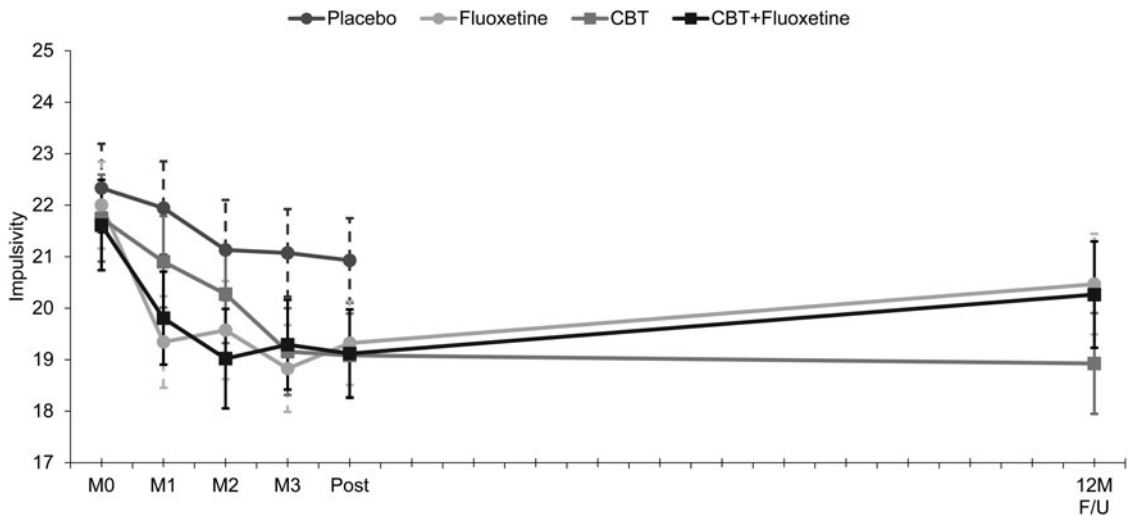


**Fig. 1.** Change in impulsivity during treatment and follow-up.
*Note*. Impulsivity changes from baseline throughout treatment and 12-month follow-up after treatment (effects of time; *Models 1A and 1B*). Least squares means and standard errors are represented graphically. CBT, cognitive-behavioral therapy; M0, baseline, M1, month 1; M2, month 2; M3, month 3; Post, month 4; 12M F/U, 12 month follow-up.

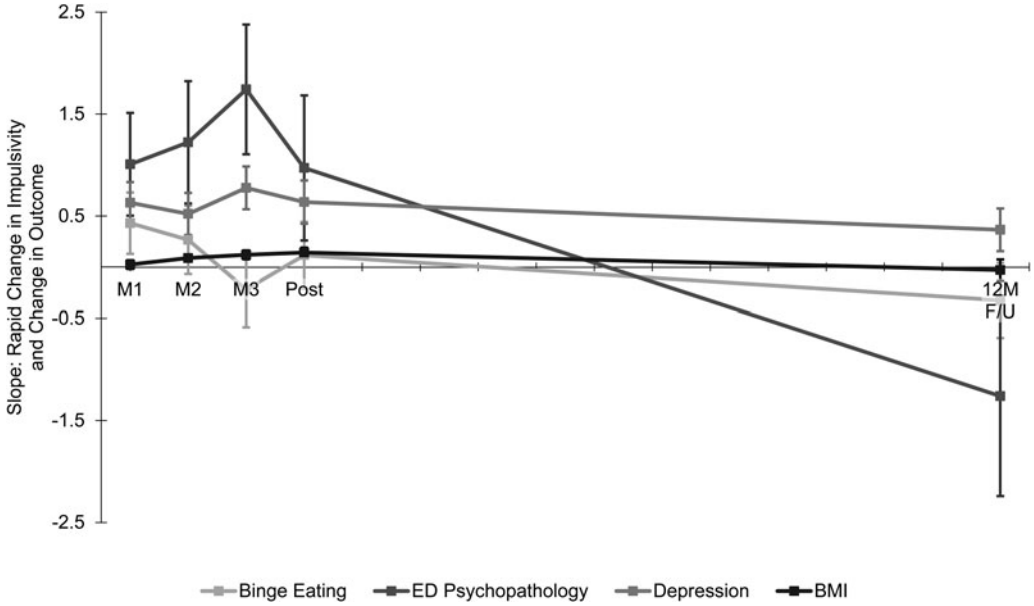


**Fig. 2.** Rapid change in impulsivity and treatment outcomes.
*Note*. Values represent slope of the association between rapid change in impulsivity (baseline – month 1) and change in outcome for the respective timepoint (*Models 2A and 2B*). Slope is expected change in outcome per unit change in impulsivity. Significant slopes are different than 0 (see online Supplementary Table S2). Error bars depict standard error. *Y*-axis: *b*-value representing slope of the effect of rapid change in impulsivity on change in outcome variables; *X*-axis: time. M1, month 1; M2, month 2; M3, month 3; Post, month 4; 12M F/U, 12 month follow-up; ED Psychopathology, eating disorder psychopathology.

### *BMI*

There was an interaction between ICS and time, reflecting a significant relationship between reduction in impulsivity during treatment and reduction in BMI at post-treatment but not during follow-up (*Model 3B*: ICS × Time: $F_{(1,47)} = 5.44$, $p = 0.02$; online Supplementary Tables S4 and S5).

## *Exploratory analyses: baseline impulsivity*

### *Binge-eating frequency*

There were neither main effects of baseline impulsivity nor interactions between baseline impulsivity and treatment group, for binge-eating outcomes (*Model 4A*: ICS: $F_{(1,107)} = 0.67$, $p = 0.41$; ICS × Group: $F_{(3,106)} = 2.51$, $p = 0.06$; *Model 4B*: ICS: $F_{(1,72)} = 1.67$, $p = 0.20$; ICS × Group: $F_{(2,71.4)} = 1.04$, $p = 0.36$; online Supplementary Table S5).

### *Eating-disorder psychopathology*

Baseline impulsivity did not predict or moderate treatment effects for eating-disorder psychopathology outcomes during treatment or follow-up (*Model 4A*: ICS: $F_{(1,101)} = 0.01$, $p = 0.91$; ICS × Group: $F_{(3,100)} = 0.61$, $p = 0.61$; *Model 4B*: ICS: $F_{(1,68.4)} = 0.00$, $p = 0.99$; ICS × Group: $F_{(2,67.6)} = 0.28$, $p = 0.75$).

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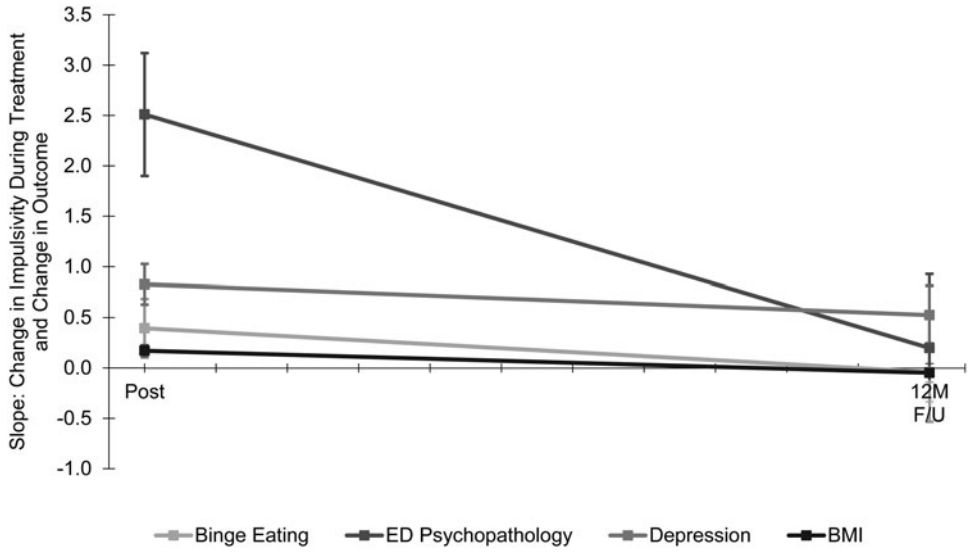


**Fig. 3.** Change in impulsivity during treatment and treatment outcomes.
*Note*. Values represent slopes of the association between change in impulsivity (baseline – post) and change in outcome for the respective timepoint (*Model 3B*). Positive values at post are greater than zero, indicating significant effects of decrease in impulsivity during treatment on outcomes (see online Supplementary Table S4). Slope is expected change in outcome per unit change in impulsivity. Error bars depict standard errors. *Y*-axis: *b*-value representing slope of the effect of change in impulsivity during treatment on change in outcome variables; *X*-axis: time. Post, month 4; 12M F/U, 12 month follow-up; ED Psychopathology, eating disorder psychopathology.

### *Depression scores*

Higher baseline impulsivity was associated with greater change in depression scores during treatment and follow-up (*Model 4A*: ICS: $F_{(1,98.4)} = 4.38$, $p = 0.04$; *Model 4B*: ICS: $F_{(1,67.8)} = 6.41$, $p = 0.01$). Additionally, there was a significant interaction between baseline impulsivity and treatment group during treatment and follow-up (*Model 4A*: ICS × Group: $F_{(3,98)} = 4.06$, $p = 0.009$; *Model 4B*: ICS × Group: $F_{(2,67.2)} = 3.13$, $p = 0.05$), explained by greater baseline impulsivity being significantly associated with greater change in depression scores in the CBT + fluoxetine group but not in the other groups (online Supplementary Table S6).

### *BMI*

Greater baseline impulsivity was not associated with change in BMI (*Model 4A*: ICS: $F_{(1,93)} = 3.79$, $p = 0.06$; *Model 4B*: ICS: $F_{(1,68)} = 0.02$, $p = 0.88$; other $p$s > 0.14).

## Discussion

The current study examined the effects of rapid and overall change in impulsivity on BED outcomes achieved with CBT and fluoxetine treatments. Across treatment conditions, impulsivity declined during treatment and remained at lower levels compared to baseline during 12 months of follow-up. Change in impulsivity was significantly associated with initial treatment outcomes. Rapid reductions in impulsivity were prospectively associated with reductions in eating-disorder psychopathology, depression scores, and BMI during treatment, but not with reductions in binge eating. Additionally, the relationship between change in impulsivity during treatment and depression scores persisted to 12-month follow-up. Baseline impulsivity neither predicted nor moderated treatment effects on eating-disorder outcomes or BMI. Collectively, these results suggest that change in impulsivity may be an important outcome and process underlying outcomes of CBT and fluoxetine treatments for BED, and that this process may be associated with longer-term psychological changes.

The significant reductions in impulsivity following treatments for BED were sustained 12 months after treatment. CBT, fluoxetine, and their combination were associated with significant reductions in impulsivity from baseline that persisted through 12-month follow-up. One previous treatment study for BED reported reductions in impulsivity during 11 weeks of treatment with lisdexamfetamine dimesylate (McElroy et al., 2016). Our findings extend those to different interventions (CBT and fluoxetine alone and in combination) and suggest that the changes are largely durable, as they were maintained for 12 months after discontinuing the acute treatments.

Reductions in impulsivity during treatment were observed across all treatment conditions including placebo (although a non-significant trend *v.* placebo was observed). Such findings might suggest that psychological and pharmacological treatments that do not specifically target impulsivity, including even a non-specific placebo treatment, may result in reduced impulsivity. These findings are consistent with previously observed reductions in impulsivity following bariatric surgery (Sarwer et al., 2019) and varied treatments for other forms of psychopathology (Hershberger et al., 2017; Littlefield et al., 2015; Peckham, Forgeard, Hsu, Beard, & Bjorgvinsson, 2019; Reese, Conway, Anand, Bauer, & Daughters, 2019).

Our findings that changes in impulsivity were associated with broad treatment outcomes for BED echo recent research with patients being treated for substance use disorder that changes in distress tolerance were associated with outcomes (Reese et al., 2019). These findings perhaps clarify further the mixed findings from prior research on baseline impulsivity as a predictor of treatment outcomes for BED (Balodis et al., 2014; Manasse et al., 2016; cf. Anderson et al., 2020; Castellini et al., 2012) by suggesting that impulsivity can decrease and, in those cases, the decreases predict improvements associated with treatments for BED even when baseline impulsivity does not. Because impulsive behaviors

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share neurobiological correlates (Bari & Robbins, 2013) and are observed across forms of psychopathology (Berg et al., 2015), change in impulsivity could be a transdiagnostic indicator of improvement during treatment across disorders. Future research should establish the consistency, specificity, timeline, and neurobiological mechanisms of changes in impulsivity and their relationships with BED treatment and outcomes.

Analyses revealed potential relationships between impulsivity and secondary outcome variables for BED (i.e. depression scores and BMI). Although CBT conditions had the greatest reduction in depression scores during the treatment study (Grilo, Crosby, et al., 2012; Grilo et al., 2005), the present analyses revealed stronger relationships between change in impulsivity and depression scores in patients who received fluoxetine, which may indicate that fluoxetine reduced both impulsivity and depressive symptoms in some individuals. Exploratory analyses also showed stronger relationships between baseline impulsivity and depression scores in patients who received CBT + fluoxetine, suggesting that individuals with greater baseline impulsivity may derive this specific benefit from combination treatment. Furthermore, although these treatments did not produce significant overall reductions in BMI (Grilo, Crosby, et al., 2012; Grilo et al., 2005), a well-known challenge in most treatment outcome studies for BED (Grilo, 2017; Hilbert et al., 2020), the present analyses revealed that change in impulsivity was associated with change in BMI and future research should investigate this finding further.

Our findings should be considered in the context of potential study limitations/strengths. The participants were predominantly female, White, and well-educated, and findings may not generalize to groups with different characteristics or to those who seek treatment in different types of clinical settings. Study design did not allow for including participants from the placebo condition in the follow-up analyses, which reduced the overall follow-up sample-size and precluded analyses of longer-term changes in impulsivity in those who received placebo. Additionally, the predictor/moderator analyses were exploratory in nature and thus should be interpreted cautiously. Potential strengths include the rigorous RCT design, different treatments delivered through standardized protocols, psychometrically established assessments performed by independent assessors, and rigorous statistical analyses examining multiple aspects of change in impulsivity and outcomes through 12-month follow-ups.

With the study's limitations/strengths as context, we cautiously offer clinical implications. Change in impulsivity may be a non-specific process that is prospectively associated with changes in some clinical outcomes in those receiving treatment for BED. Even if non-specific, active monitoring of the rate of change in impulsivity during treatment could help to inform timely interventions. The observed trend toward an increase in impulsivity during follow-up suggests potential utility of clinical and research work targeting risk for relapse. Alternatively, if change in impulsivity is found to be directly related to clinical improvement, incorporating impulsivity into conceptualizations of BED could refine treatments (Balodis, Grilo, & Potenza, 2015; Boswell & Grilo, 2020; Boswell & Kober, 2016; Hutson, Balodis, & Potenza, 2018). Targeting impulsivity more specifically, including through specific pharmacological agents (McElroy et al., 2016), brief skills training (Boswell, Sun, Suzuki, & Kober, 2018), and impulsivity-focused augmented treatment (Schag et al., 2019) could help speed the rate at which impulsivity changes or improve the durability of these changes, and thereby enhance BED treatment outcomes.

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