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Predictors of improvement in an open-trial multisite evaluation of emotion regulation group therapy

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ABSTRACT
Emotion regulation group therapy (ERGT) is a novel treatment specifically targeting deliberate non-suicidal self-harm (DSH) in individuals with borderline personality disorder (BPD). Identifying robust predictors of positive response to ERGT could aid clinicians in treatment selection; however, to date, only one such study has been conducted. Thus, we aimed to replicate previously identified predictors of treatment response to ERGT by investigating demographic, clinical, and diagnostic predictors in 95 women with BPD or subclinical BPD who had participated in an open-trial evaluation of ERGT. Outcomes evaluated were frequency of DSH and emotion dysregulation. Assessments were conducted at pretreatment, post-treatment, and 6-month follow-up. Multilevel mixed linear models and multilevel negative binomial generalized estimated equations were used to identify significant interactions between the predictors and outcomes.

We found that greater pretreatment DSH frequency was associated with greater improvements in DSH during treatment ($b = 0.998$, SE = 0.00, $p = 0.03$) and follow-up ($b = 0.997$, SE = 0.00, $p < 0.01$) and that greater BPD severity was associated with greater improvements in DSH during treatment ($b = 0.84$, SE = 0.06, $p = 0.02$) and in emotion dysregulation at follow-up ($b = -3.05$, SE = 1.47, $p = 0.04$). Co-occurring disorders were associated with poorer treatment response during follow-up. Results were generally consistent with a previous study of the predictors of response to ERGT. The findings provide further support for the utility of this treatment across a range of BPD patients, including patients with severe DSH and BPD.

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KEYWORDS
Emotion regulation group therapy; predictors of treatment response; borderline personality disorder; deliberate self-harm; non-suicidal self-injury; emotion regulation

Introduction
Borderline personality disorder (BPD) is a serious mental disorder characterized by emotional and behavioural dysregulation, suicidal and non-suicidal self-harm, and...
interpersonal difficulties (American Psychiatric Association, 2013). Emotion regulation group therapy (ERGT; Gratz & Gunderson, 2006; Gratz & Tull, 2011; Gratz, Tull, & Levy, 2014b) is a brief, acceptance-based behavioural group therapy and one of several efficacious treatments for BPD and related pathology that has been developed during the past decades (e.g. dialectical behaviour therapy [DBT]; Linehan, 1993, mentalization-based treatment [MBT]; Bateman & Fonagy, 1999, and schema-focused therapy [SFT]; Young, Klosko, & Weishaar, 2003). ERGT was developed specifically to target deliberate self-harm (DSH; defined as “the deliberate, direct destruction or alteration of body tissue without conscious suicidal intent, but resulting in injury severe enough for tissue damage [e.g., scarring] to occur”; Gratz, 2001, p. 253), a behaviour that is very common among individuals with BPD (Linehan, 1993; Sansone, Wiederman, & Sansone, 1998). This treatment was grounded in a growing body of research suggesting that DSH serves an emotion-regulating function (for a review, see Andover & Morris, 2014). ERGT aims to reduce DSH by increasing emotion regulation skills among self-harming individuals with BPD and has demonstrated efficacy in multiple trials (Gratz & Gunderson, 2006; Gratz et al., 2014b). However, few studies have examined predictors of treatment response to ERGT.

We previously evaluated ERGT in a multisite, uncontrolled, open-trial design of the implementation of this treatment in routine clinical care, finding significant improvements in DSH frequency, emotion dysregulation, general psychiatric symptoms, and social and vocational functioning at post-treatment and 6-month follow-up (for a comprehensive account of the results, see Sahlin et al., 2017). Based on these findings, we concluded that ERGT is a clinically feasible and useful treatment for DSH in BPD. Notably, however, not all patients responded to this treatment, and those who did not may have benefitted from a longer or more intensive treatment than ERGT, such as DBT or MBT. Identifying patients who are less likely to respond positively to ERGT could aid clinicians in selecting the best treatment for their patients. Indeed, the growing number of psychological treatments available for different disorders and maladaptive behaviours has led to a demand for researchers to identify not only whether a particular treatment is efficacious, but also for whom and under what conditions the treatment works (Kraemer, Wilson, Fairburn, & Agras, 2002).

The literature on the predictors of outcome in treatments for BPD and related pathology has produced mixed and contradictory findings. Although this may be due to factors such as the wide variability in outcomes and predictors studied and the relatively small sample sizes, it may also be due to the heterogeneity of treatments for BPD (which make comparisons across treatments difficult). Nonetheless, a relatively recent review of predictors of response to treatments for BPD had two main findings: 1) demographic variables and co-occurring disorders did not influence treatment outcomes in a consistent way, and 2) pretreatment clinical severity and patient-rated treatment alliance showed a positive and significant influence across studies, such that patients with greater clinical severity or a strong treatment alliance evidenced better treatment response to BPD treatments (Barnicot et al., 2012). Notably, however, researchers have noted the need for replication of these and other findings in order to identify robust predictors of treatment response among those with BPD (Arntz, Stupar-Rutenfrans, Bloo, van Dyck, & Spinhoven, 2015).
To date, only one study has examined the predictors of response to ERGT (Gratz, Dixon-Gordon, & Tull, 2014a), and the results of this study are consistent with the aforementioned review (Barnicot et al., 2012). Specifically, whereas demographic variables had minimal impact on treatment response, several indices of greater clinical severity (i.e. higher baseline emotion dysregulation and BPD symptoms and lifetime and recent DSH frequency) predicted better responses during treatment and follow-up on the primary targets of treatment (Gratz et al., 2014a). However, whether these same factors predict response to ERGT in routine clinical care is unknown. Indeed, it is possible that differences in patient characteristics and therapist expertise and training across settings could influence both the utility of this treatment for a range of BPD patients and the patient characteristics that influence treatment response. Replication of these findings would advance research on the predictors of response to DSH and BPD treatments and have implications for the dissemination of ERGT to routine clinical care.

**Aim**

The current study investigated the predictors of response to ERGT in a multisite open trial of this treatment in routine clinical care. Our aim was to replicate previous findings of predictors of treatment response to ERGT. Specifically, we hypothesized that greater clinical severity (as evidenced by greater levels of emotion dysregulation, BPD symptoms, and DSH at baseline) would predict better response to treatment.

**Method**

**Design**

This study included 95 women with DSH and a threshold or sub-threshold diagnosis of BPD who had participated in a multisite uncontrolled evaluation of ERGT in routine clinical care (Sahlin et al., 2017). All participants were assessed at baseline, pretreatment (one week before the start of treatment), post-treatment, and 6-month follow-up. The current study examined baseline and pretreatment characteristics associated with responses at post-treatment and 6-month follow-up. The study was approved by the Regional Ethical Review Board in Stockholm (2013/1321–31/3) and was registered on Clinicaltrials.gov (Identifier NCT01986257).

**Participants**

Ninety-five participants were recruited and assessed by community-based healthcare professionals at 14 psychiatric outpatient clinics across Sweden. All participants were female, met at least three diagnostic criteria for BPD according to the DSM-IV (American Psychiatric Association, 2000), and had engaged in at least three episodes of DSH in the past six months. Procedures, recruitment, and description of eligibility criteria are detailed in the original report (Sahlin et al., 2017). Participant demographics are detailed in Table 1.
ERGT is a 14-session, acceptance-based behavioural group treatment developed to treat DSH among individuals with BPD by targeting its underlying mechanism of emotion dysregulation (Gratz & Gunderson, 2006). ERGT systematically teaches skills aimed at improving multiple dimensions of emotion regulation, including emotional awareness, understanding, and acceptance, as well as the use of non-avoidant emotion regulation strategies to modulate the intensity and/or duration of emotional responses (among others, see Gratz, 2007; Gratz et al., 2014b). ERGT has shown utility in reducing DSH, emotion dysregulation, and psychiatric symptoms (i.e. BPD, depression, and anxiety) both in efficacy studies (Gratz & Gunderson, 2006; Gratz & Tull, 2011; Gratz et al., 2014b) and when implemented in routine clinical care (Sahlin et al., 2017).

**Potential predictors**

**Demographic predictors**

Age and highest completed education level (on a three-level ordinal scale; 1: primary school; 2: high school/vocational school; and 3: university) were included as demographic predictors.
**Clinical predictors**

Information on medication use and treatment as usual in the community was collected by clinicians at the baseline assessment and self-reported by participants at the pre-treatment assessment.

Recent (i.e. past 4 months) DSH frequency was assessed via the Deliberate Self-Harm Inventory (DSHI; Gratz, 2001), a self-report questionnaire that assesses various aspects of DSH (including frequency) over specified time periods. Lifetime and past 6 month DSH frequency were assessed with an interview version of the DSHI (Gratz, 2001) administered at the baseline assessment. The DSHI has been found to have adequate test–retest reliability and construct, discriminant, and convergent validity.

Current, lifetime, and past 3 month suicidal ideation and behaviours were assessed using the Columbia Suicide Severity Rating Scale (C-SSRS; Posner et al., 2011). The C-SSRS has shown high convergent and divergent validity and moderate to strong internal consistency in clinical and research samples (Posner et al., 2011).

Pretreatment emotion dysregulation was assessed using the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004), a 36-item measure of clinically relevant emotion regulation difficulties across six domains (e.g. non-acceptance of negative emotions, difficulties controlling impulsive behaviours when distressed, lack of emotional awareness). Items are rated on a 5-point scale, from 1 (“almost never”) to 5 (“almost always”), with higher scores (min 36, max 180) indicating more severe emotion dysregulation. The DERS has shown high internal consistency (total score, Cronbach’s $\alpha = .90$ in this sample), good test–retest reliability, and good construct and predictive validity. BPD symptom severity was assessed via the number of criteria met according to the BPD module of the Structured Clinical Interview for DSM-IV Personality Disorders (SCID-II BPD; First, Gibbon, Spitzer, Williams, & Benjamin, 1997), administered at the baseline assessment.

**Diagnostic predictors**

Diagnostic predictors included in this study were DSM-IV Axis I disorders assessed with The MINI International Neuropsychiatric Interview version 6 (MINI 6; Sheehan et al., 1998) administered at the baseline assessment.

**Outcome measures**

The outcome measures of interest in this study were DSH frequency and emotion dysregulation at post-treatment and follow-up (assessed via the DSHI; Gratz, 2001 and DERS; Gratz & Roemer, 2004, respectively).

**Procedures**

Diagnostic interviews and measures of lifetime and past 6-month DSH frequency and suicidal ideation/behaviour were administered by clinicians at the baseline assessment (mean 20.7 days before treatment start, SD = 17.2, range 1–91). All demographic and clinical predictor variables were assessed via self-report measures administered online (a method with demonstrated validity; Hedman et al., 2010) at the pretreatment
assessments (1 week before treatment start). The outcome measures were administered at pretreatment, post-treatment, and 6-month follow-up.

**Statistical analyses**

Mixed-effect multilevel modelling was used to identify variables that influenced improvement between pretreatment and post-treatment, and post-treatment and 6-month follow-up. The models were built using the multilevel generalized estimated equations negative binomial regression for count data (i.e. DSHI) and general linear mixed-effect models for emotion dysregulation (DERS) in STATA version 14.1. The models included all available data at the three assessment points (pretreatment, post-treatment, and 6-month follow-up) for each outcome, thus making them intent-to-treat analyses. We estimated separate slopes for the change between the pre- and post-treatment assessments (T1) and the change between the post-treatment and 6-month follow-up assessments (T2). Random intercepts and random slopes were included in the linear models if they significantly improved model fit according to log-likelihood ratio tests.

Predictors were entered as interaction effects with T1 and T2, and significant interaction effect coefficients ($p < 0.05$) were interpreted as a linear relationship between the level of the predictor and the rate of improvement during (T1) and/or after (T2) treatment. If several predictors within the same cluster (demographic, diagnostic, or clinical) reached significance ($p < 0.05$), they were added into a multiple regression model, according to the two-step approach suggested by De Graaf, Hollon, & Huibers (2010). The predictor variables were centred before they were entered into the model. Continuous predictors were grand-mean centred, and binary predictors were centred by subtracting 0.5 from the variable value.

**Results**

**Sample characteristics**

Of the 95 participants who were included in this study (mean age 25.1, SD = 7.0, range 18–49), 21 dropped out of treatment (22%). See Table 1 for participant demographic, clinical, and diagnostic data at pretreatment.

**Treatment adherence and attrition**

Mean number of sessions attended for all included participants was 11 (SD = 5.2, min 0, max 16). Seventy-two participants (76%) attended ≥ 7 sessions and 47 (49%) attended 14 sessions. Post-treatment assessments were completed by 88 (93%) participants, and 6-month follow-up assessments were completed by 76 (82%) participants.

**Treatment outcome**

There was a significant general effect of time in ERGT on the rate of improvement of DSH frequency from pretreatment to post-treatment (T0 to T1; $b = 0.67$, SE = 0.06, $p < 0.001$) and
from post-treatment to 6-month follow-up (T1 to T2; \(b = 0.78, \text{SE} = 0.07, p < 0.01\)). There was also a significant general effect of time in ERGT on the rate of improvement of emotion dysregulation from pre- to post-treatment (\(b = -17.57, \text{SE} = 2.76, p < 0.001\)), albeit not from post-treatment to 6-month follow-up (\(b = -2.38, \text{SE} = 2.23, p = 0.29\)). Table 2 displays the results of all predictors on treatment response. Significant interactions are illustrated in the Supplemental material (see Supplemental Figures 1-5). When illustrating the interaction effects of continuous variables on DSH frequency or emotion dysregulation, participants with scores on the 75th percentile are compared to those with scores on the 25th percentile.

**Demographic, clinical, and diagnostic predictors’ impact on outcome**

**Demographic predictors**

Table 2 displays the results of demographic predictors of treatment response. Both age and education level were significant predictors of response on the measure of DSH. Both younger age and lower education level were associated with higher levels of DSH at baseline and a more rapid decline in DSH frequency during treatment. After entering both predictors into a multiple regression, only age remained a significant predictor of improvements in DSH at T1 (\(p < 0.001\); see Supplemental Figure 1). There were no significant effects of age or education level on improvements in emotion dysregulation.

**Clinical predictors**

Several clinical predictors were associated with outcomes in this sample. BPD symptom severity, past 3-month suicidal behaviour, and past 6-month DSH frequency were associated with greater improvements in DSH during treatment, and past 6-month DSH frequency was associated with greater continued improvements in DSH during follow-up. Likewise, having ever attempted suicide, being on psychotropic medications, and having a Cognitive behavioural therapist (CBT) as treatment as usual (TAU) were all associated with greater reductions in DSH during follow-up. Contrary to expectations, however, greater emotion dysregulation at pretreatment was associated with an increase in DSH from post-treatment to follow-up. When all significant predictors of better treatment response on the measure of DSH were examined simultaneously in a multiple regression equation,\(^1\) all except having ever attempted suicide (\(p > 0.06\)) emerged as significant unique predictors (see Supplemental Figure 2). Specifically, BPD symptom severity demonstrated a significant interaction with both T1 and T2 (\(p = 0.05\) and \(p < 0.01\)), past 3-month suicidal behaviour demonstrated a significant interaction with T1 (\(p < 0.001\)), and past 6-month DSH, psychotropic medication use and having a CBT therapist as TAU demonstrated significant interactions with T2 (all \(p\)’s < 0.01).

As for predictors of improvements in emotion dysregulation, higher pretreatment levels of emotion dysregulation predicted greater improvements in emotion dysregulation during treatment, and greater BPD symptom severity predicted greater improvements in emotion dysregulation during follow-up. Both predictors remained significant when examined simultaneously in a multiple regression (BPD severity \(p = 0.005\), the DERS \(p = 0.01\); see Supplemental Figure 3).
Table 2. Effect of predictors on the rates of improvement in DSH and emotion dysregulation during treatment and follow-up.

<table>
<thead>
<tr>
<th>Outcome variables</th>
<th>Effect on rate of improvement</th>
<th>Self-harm frequency (DSHI)</th>
<th></th>
<th>Emotion dysregulation (DERS)</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Predictors</td>
<td>T1: PRE–POST</td>
<td>T2: POST TO FU6</td>
<td>T1: PRE–POST</td>
<td>T2: POST TO FU6</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Demographic predictors</td>
<td>Coefficient (SE) p-value</td>
<td>Coefficient (SE) p-value</td>
<td>Coefficient (SE) p-value</td>
<td>Coefficient (SE) p-value</td>
<td>Coefficient (SE) p-value</td>
</tr>
<tr>
<td>Age</td>
<td>1.06 (0.01) &lt; 0.001</td>
<td>1.00 (0.01) 0.84</td>
<td>0.29 (0.40) 0.47</td>
<td>0.30 (0.32) 0.34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education level</td>
<td>1.54 (0.20) 0.001</td>
<td>0.79 (0.11) 0.10</td>
<td>0.64 (4.62) 0.89</td>
<td>–1.58 (3.71) 0.67</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of BPD criteria</td>
<td>0.84 (0.06) 0.02</td>
<td>1.15 (0.09) 0.07</td>
<td>0.82 (1.81) 0.65</td>
<td>–3.05 (1.47) 0.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suicide attempt, lifetime (yes/no)</td>
<td>1.08 (0.21) 0.69</td>
<td>0.57 (0.12) &lt; 0.01</td>
<td>6.10 (5.69) 0.28</td>
<td>–2.32 (4.56) 0.61</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suicide attempt, past 3 months</td>
<td>0.31 (0.07) &lt; 0.001</td>
<td>0.65 (0.18) 0.11</td>
<td>2.54 (7.32) 0.73</td>
<td>–0.03 (6.53) 0.99</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DSH frequency, lifetime</td>
<td>1.00 (0.00) 0.12</td>
<td>1.00 (0.00) 0.81</td>
<td>–0.00 (0.00) 0.06</td>
<td>0.00 (0.00) 0.95</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DSH frequency past 6 months</td>
<td>0.99 (0.00) 0.03</td>
<td>0.99 (0.00) &lt; 0.01</td>
<td>0.04 (0.03) 0.23</td>
<td>–0.02 (0.03) 0.52</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotion dysregulation (DERS-36)</td>
<td>1.00 (0.00) 0.65</td>
<td>1.01 (0.00) 0.001</td>
<td>–0.32 (0.14) 0.02</td>
<td>–0.06 (0.08) 0.44</td>
<td></td>
<td></td>
</tr>
<tr>
<td>On psychotropic medication (yes/no)</td>
<td>1.11 (0.27) 0.66</td>
<td>0.59 (0.16) 0.05</td>
<td>5.49 (6.18) 0.37</td>
<td>7.14 (5.15) 0.17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cognitive behaviour therapy as TAU</td>
<td>0.96 (0.19) 0.84</td>
<td>0.63 (0.13) 0.02</td>
<td>–6.91 (5.87) 0.24</td>
<td>2.68 (4.66) 0.57</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnostic predictors (yes/no)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>0.64 (0.12) 0.02</td>
<td>4.06 (0.81) &lt; 0.001</td>
<td>6.48 (5.45) 0.23</td>
<td>3.38 (4.47) 0.45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Panic disorder</td>
<td>1.30 (0.23) 0.14</td>
<td>1.08 (0.17) 0.46</td>
<td>2.72 (5.68) 0.63</td>
<td>5.25 (4.66) 0.26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social anxiety disorder</td>
<td>1.12 (0.21) 0.54</td>
<td>1.66 (0.34) 0.01</td>
<td>3.10 (5.58) 0.58</td>
<td>13.44 (4.56) &lt; 0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-traumatic stress disorder</td>
<td>0.98 (0.22) 0.94</td>
<td>1.82 (0.44) 0.01</td>
<td>–3.81 (6.77) 0.57</td>
<td>0.92 (5.54) 0.87</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Generalized anxiety disorder</td>
<td>1.22 (0.21) 0.25</td>
<td>0.44 (0.08) &lt; 0.001</td>
<td>–3.99 (5.65) 0.48</td>
<td>4.46 (4.60) 0.33</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Substance-use disorders</td>
<td>2.08 (0.90) 0.09</td>
<td>0.26 (0.12) &lt; 0.01</td>
<td>16.04 (13.34) 0.23</td>
<td>–11.44 (10.04) 0.26</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: BPD, borderline personality disorder; DSHI, Deliberate Self-harm Inventory; DERS, Difficulties in emotion regulation scale; DSH, deliberate self-harm.
Diagnostic predictors

Only major depressive disorder (MDD) predicted response on the measure of DSH during treatment, such that the presence of MDD was associated with greater improvements in DSH during treatment. Conversely, with the exception of panic disorder, all examined co-occurring disorders predicted response on the measure of DSH frequency during follow-up. Specifically, whereas generalized anxiety disorder (GAD) and substance-use disorder (SUD) predicted greater improvements in DSH frequency during follow-up, MDD, social anxiety disorder (SAD), and post-traumatic stress disorder (PTSD) were associated with an increase in DSH during follow-up. When entered simultaneously into a multiple regression equation, MDD ($p < 0.001$), PTSD ($p = 0.01$), and SAD ($p < 0.001$) remained significant unique predictors of DSH during follow-up (see Supplemental Figure 4).

There were fewer diagnostic predictors of treatment response on the measure of emotion dysregulation. Specifically, only co-occurring SAD was associated with poorer response on the measure of emotion dysregulation during follow-up (see Supplemental Figure 5).

Discussion

This study examined the predictors of treatment outcome in a multisite open trial of ERGT in routine clinical care. Overall, many of the results were consistent with those obtained in the previous study examining the predictors of response to ERGT (Gratz et al., 2014a), replicating some of the main findings of that study. Specifically, and consistent with Gratz et al. (2014a), there were very few significant predictors of response on the measure of emotion dysregulation, including no impact of any of the demographic, TAU, or diagnostic characteristics on treatment response during treatment, and only one of these characteristics (i.e. the presence of co-occurring SAD) predicting poorer response on this measure during follow-up. These results provide further support for the utility of this treatment for emotion dysregulation across a range of self-harming patients, including patients with varying forms of TAU.

Furthermore, although findings that two baseline clinical characteristics (i.e. emotion dysregulation and BPD symptom severity) predicted greater improvements in emotion dysregulation during treatment and follow-up (respectively) differ from the findings of Gratz et al. (2014a), who did not find any significant predictors of response on the measure of emotion dysregulation, they are consistent with the findings of both the current and Gratz et al. (2014a) studies that several indicators of greater clinical severity predicted better responses on some of the primary measures of interest. Specifically, in both this study and the Gratz et al. (2014a) study, greater baseline DSH was significantly associated with greater improvements in DSH during follow-up. Notably, although Gratz et al. also found that other indices of baseline clinical severity (i.e. greater BPD symptom severity and a past-year suicide attempt) predicted better treatment response on measures of BPD symptoms and experiential avoidance (outcomes not examined in this study), they were not associated with greater improvements in DSH as they were here. Findings of the differential relevance of clinical severity indices to improvements in DSH in particular in the current study, relative to the Gratz et al. (2014a) study, may be driven by the different settings and levels of therapist training in the two studies.
Specifically, given that the current study was conducted in routine clinical care and the groups were provided by therapists with only 3 days of training, there may have been more variability in outcomes and the potential for more factors to influence responses to the treatment. Thus, more predictors of improvements in DSH may have emerged as significant.

Regardless, however, it is important to note that the aforementioned findings of greater clinical severity predicting better responses on some key measures are consistent with past findings that greater clinical severity generally does not negatively influence treatment outcomes in treatments for BPD (although most studies have focused on improvements in BPD symptoms versus DSH or emotion dysregulation; Barnicot et al., 2012). For example, Black et al. (2009) studied a broad range of predictors influencing change in BPD symptoms, social functioning, and depression following a 20-week group treatment programme (systems training for emotional predictability and problem solving; STEPPS; Blum et al., 2008) and found that greater clinical severity at baseline predicted better response on all outcomes except for depression (Black et al., 2009). Likewise, in a study seeking to identify indicators of clinical severity (e.g. symptom distress, co-occurring disorders, BPD severity) that could be used to identify patients most likely to benefit from a specialized BPD treatment (i.e. MBT), Bateman and Fonagy (2013) found that none of the indicators of clinical severity predicted treatment response (Bateman & Fonagy, 2013). Finally, although some studies have found that greater baseline DSH has a negative impact on improvements in BPD symptoms (Ryle & Golynkina, 2000) and DSH (Harned, Rizvi, & Linehan, 2010) over treatment, others have found positive effects (Black et al., 2009) or no effects (Arntz et al., 2015; Davidson, Tyrer, Norrie, Palmer, & Tyrer, 2010) of baseline DSH frequency on treatment response. Although findings that greater baseline clinical severity predicts better treatment response may reflect simply the greater magnitude of change possible when scores are elevated at baseline, these findings clearly suggest that patients with high levels of both DSH and BPD can benefit from treatments for BPD and related pathology.

Also consistent with the results of Gratz et al. (2014a) are findings that the presence of co-occurring psychiatric disorders generally did not influence treatment response on measures of DSH or emotion dysregulation during treatment (with the one exception that co-occurring MDD was associated with greater improvements in DSH during treatment in the current study). Together, these findings suggest the utility of this treatment for DSH and emotion dysregulation among a wide range of BPD patients with and without co-occurring psychopathology.

Notably, despite finding comparable results in several domains to the original predictors of response to the ERGT study (Gratz et al., 2014a), other findings of the current study differed from those obtained in the original. In particular, although Gratz et al. (2014a) identified very few significant predictors of treatment response on the measure of DSH either during treatment (where past year inpatient hospitalization predicted fewer improvements in DSH) or during the follow-up period (where baseline and lifetime DSH predicted greater improvements in DSH, and co-occurring GAD predicted an absence of continued improvements in DSH), the current study found multiple significant predictors of treatment response on the measure of DSH (both better and poorer) during treatment and follow-up. Specifically, and in contrast to the
findings of Gratz et al. (2014a), younger age, lower education, greater baseline BPD severity, past 6-month DSH frequency, and past 3-month suicidal behaviour were associated with greater improvements in DSH during treatment; having a CBT therapist, being on psychotropic medication, and co-occurring GAD and SUD were associated with greater improvements in DSH during follow-up; and greater baseline emotion dysregulation and co-occurring MDD, SAD, and PTSD were associated with increases in DSH during follow-up.

As noted previously, findings of a greater number of significant predictors of response on the measure of DSH during treatment in this study versus the Gratz et al. (2014a) study could reflect the differences in treatment setting and therapist training in the two studies, with the current trial producing more variability in (and, thus, significant predictors of) DSH outcomes. Likewise, the fact that the group leaders in the present study received less training in ERGT and arguably did not have the same level of expertise in delivering this treatment as the therapists in the Gratz et al. (2014a) trial could have influenced the extent to which treatment gains were maintained during the follow-up period, particularly for patients with more severe or complex clinical presentations. This, in turn, could have led to the emergence of more significant predictors of both better and poorer response on the measure of DSH during follow-up. For example, the ongoing support provided by an individual clinician of a similar theoretical perspective as ERGT (i.e. a CBT therapist) could facilitate continued improvements in DSH during the follow-up period (relative to patients who were not receiving continued CBT). Moreover, psychotropic medication use could increase stability for patients during the follow-up, also facilitating the maintenance of treatment gains. Finally, it may be that ERGT delivered in routine clinical care by therapists with minimal training, although useful in the treatment of DSH and emotion dysregulation among women with BPD (Sahlin et al., 2017), may be associated with less-stable decreases in DSH among participants with more complex clinical presentations, particularly co-occurring emotional disorders.

Findings of a greater number of significant predictors of treatment response on the measure of DSH in the current study could also reflect differences in sample sizes across the two studies (with the present study having a larger sample and thus more statistical power), sample differences (including greater access to publicly funded psychiatric care and higher rates of CBT TAU in the current sample), or differences in the diagnostic interviews used to assess co-occurring psychiatric disorders (i.e. the MINI in this study vs. the SCID-I/P in the Gratz et al., 2014a study). Indeed, the lack of consistent assessments of co-occurring disorders across the two studies, combined with the larger sample size in the current study, could (at least in part) account for the different pattern of findings regarding the impact of co-occurring disorders on treatment response on the measure of DSH. Regardless, further research examining the relative utility of ERGT for patients with different co-occurring disorders in a range of clinical settings is needed. Moreover, given that greater baseline emotion dysregulation and co-occurring MDD, PTSD, and SAD were associated with increases in DSH during the follow-up period within the current sample, future research should examine whether the incorporation of booster sessions post-treatment, ongoing contact with a CBT therapist, or the inclusion of specific skills targeting symptoms of PTSD, MDD, or SAD would facilitate greater maintenance of treatment gains during the follow-up period.
There are some limitations worth noting when considering the results of this study. First, although the goal of this study was to replicate the findings of Gratz et al. (2014a), the naturalistic setting and related time constraints of the study therapists limited the number of assessments that could be administered. Thus, it was not possible to fully explore the similarities and differences between our findings and those of Gratz et al. (2014a). Nonetheless, it is important to note that we were able to examine two of the three primary outcomes of interest (i.e. DSH and emotion dysregulation), albeit not BPD symptoms. Further understanding of the predictors of response to ERGT or other specialized BPD treatments will require the systematic assessment of standard predictors and outcomes across studies. Second, due to the moderate sample size (albeit larger than the sample in the Gratz et al., 2014a study), the power to detect differences was limited for categorical variables with small cell sizes. Moreover, we did not control for multiple tests, which might have increased the risk of Type II error or chance findings. Although the fact that this study focused on confirming previously identified predictors of treatment response to ERGT, rather than conducting exploratory analyses to identify new predictors, limited our number of analyses relative to some previous studies of predictors of response to BPD treatments, future research is needed to replicate these findings to ensure that the results are not spurious. Finally, as is common in most studies on BPD, our study included only women, thus limiting the generalizability of the results to men or adolescents.

Nonetheless, this study adds to the growing literature on predictors of outcome in treatments for BPD, with an emphasis on two highly relevant targets for this population: DSH and emotion dysregulation. Our results replicated many of the findings of the one previous study examining predictors of response to ERGT, particularly with regard to emotion dysregulation. In particular, and consistent with the premise that research on predictors of treatment response may guide clinical decision making, our results suggest that ERGT is a useful treatment even for individuals with more frequent DSH and more severe BPD pathology, although booster sessions may be needed to maintain treatment gains for patients with co-occurring emotional disorders.

Notes

1. When the DERS was included as a predictor in the multiple regression, it did not remain significant, but the model did not converge. Thus, we removed the DERS from the final multiple regression model.

2. When GAD was included as a predictor in the multiple regression, it did not remain significant, but the model did not converge. Thus, we removed GAD from the final multiple regression model.

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