Non-suicidal self-injury during an exposure-based treatment in patients with posttraumatic stress disorder and borderline features

Antje Krüger a, b, *, Nikolaus Kleindienst a, Kathlen Priebe a, Anne S. Dyer c, Regina Steil d, Christian Schmahl a, Martin Bohus a

a Department of Psychosomatic Medicine and Psychotherapy, Central Institute of Mental Health, Medical Faculty Mannheim, Heidelberg University, Germany
b Department of Psychology and Sports Sciences, Institute of Psychology, University of Münster, Germany
c Department of Psychology and Psychotherapy, School of Social Science, University of Mannheim, Germany
d Department of Psychology and Sports Sciences, Institute of Psychology, Johann Wolfgang Goethe University, Frankfurt am Main, Germany

Abstract

Patients with posttraumatic stress disorder (PTSD) and features of borderline personality disorder (BPD) often show non-suicidal self-injury (NSSI). However, patients with on-going NSSI are mostly excluded from PTSD treatments and NSSI during PTSD treatment has rarely been investigated. The aim of the present study was to evaluate the course of NSSI during an exposure-based PTSD treatment.

This study focused on a subset (n = 34) of data from a randomised controlled trial that tested the efficacy of a residential PTSD programme (DBT-PTSD) in comparison to a treatment-as-usual wait-list. In this subset we compared a) NSSI during treatment between participants who had or had not engaged in NSSI pre-treatment and b) NSSI between treatment weeks that included exposure interventions vs. those that did not. We further compared the outcome between participants with vs. without NSSI at pre-treatment.

At pre-treatment, 62% participants reported on-going NSSI. During treatment, the percentage of participants carrying out NSSI decreased to 38% (p = 0.003). The rates of NSSI were similar in treatment weeks with exposure compared to weeks without. Similar results were observed for the frequency of NSSI. At the end of treatment, participants showed comparable improvement in PTSD symptoms regardless of whether or not they had exhibited NSSI beforehand.

Introduction

Non-suicidal self-injury (NSSI) is defined as the direct and deliberate destruction of one’s own body tissue in the absence of suicidal intent (Nock & Fava, 2009). Common forms of NSSI are cutting, severely scratching, or burning the skin, banging, or hitting; often, more than one means is employed (Kleindienst et al., 2008). In adult non-clinical samples, the lifetime prevalence of engaging in NSSI has been reported to be 4–6% (Klonsky, 2011; Klonsky, Oltmanns, & Turkheimer, 2003), while in clinical samples, the prevalence is 19–25% (Briere & Gil, 1998). NSSI is most frequently performed as an emotion regulation strategy with the aim of finding relief from aversive emotional arousal, numbness, or dissociative symptoms (Klonsky, 2009).

NSSI is often seen in individuals with borderline personality disorder (BPD), BPD features (Skodol et al., 2002) or posttraumatic stress disorder (PTSD) (Gratz & Tull, 2012). Patients with a history of childhood sexual abuse (CSA) have an increased risk of developing BPD, with an odds ratio of 7.6 (Cutajar et al., 2010). Patients with PTSD that is attributable to CSA have high rates of NSSI (Weierich & Nock, 2008).

For both BPD and PTSD, effective treatment programmes do exist. Dialectical behaviour therapy (DBT) has been proven to be highly effective for BPD (Stoffers et al., 2012), while trauma-focused cognitive-behavioural therapies have been shown to be highly effective for PTSD and are recommended as first-line treatments (National Collaborating Centre for Mental Health, 2005; Watts et al., 2013). Medium to large effect sizes have been reported for patients with PTSD after CSA (Taylor & Harvey, 2010). However, for individuals with both PTSD after CSA and BPD features, neither of...
these treatments show sufficient empirical evidence, as PTSD patients with co-occurring BPD symptoms such as NSSI, severe dissociation or suicidality are often excluded from treatment studies (Bradley, Greene, Russ, Dutra, & Westen, 2005). Additionally, subgroup analyses of participants with NSSI are usually not reported.

DBT focuses on dysfunctional behaviour patterns including NSSI and suicidality (Linehan, 1993). Reducing posttraumatic stress is supposed to be a treatment target only once previous targets are under control, and thus should take place in the second phase of therapy. This implies that patients must usually undergo many months of standard DBT before PTSD treatment can start. These rules are based on clinical experience and on the assumption that dysfunctional behaviour such as self-harm might increase under the emotional distress triggered by exposure to traumatic memories. Therefore, it seems reasonable that many clinical trials on PTSD treatments have excluded patients with on-going suicidal ideation and/or NSSI due to safety reasons (Bradley et al., 2005). This practice is in line with a substantial number of therapists who question the safety of exposure elements for PTSD patients and report a reluctance to use these interventions (Becker, Zayfert, & Anderson, 2004; Cahill, Foa, Hembree, Marshall, & Nacash, 2006).

However, this leads to the problem that a large proportion of PTSD patients with on-going NSSI are not receiving appropriate PTSD treatment. Such patients usually find themselves in a vicious circle of intrusive traumatic memories, highly aversive emotions, a lack of emotion regulation strategies, and dysfunctional behavioural strategies which are highly effective in reducing stress in the short run (Reitz et al., 2012). Standard DBT is able to teach such patients stress-tolerance skills and emotion regulation strategies, which leads to a reduction of NSSI and suicidality but not to a sufficient reduction in PTSD symptoms (Harned et al., 2008; Linehan, Comtois, Murray, et al., 2006). Early promising data suggest a combination of DBT and trauma-focused interventions as an effective treatment approach for patients suffering from co-morbid PTSD and BPD features (Bohus et al., 2013; Harned, Korslund, Foa, & Linehan, 2012; Harned, Korslund, & Linehan, 2014).

Despite the high prevalence of NSSI in PTSD, the occurrence and course of dysfunctional behaviours such as NSSI during PTSD treatment has rarely been investigated. To the authors’ knowledge, only two studies, both by Harned and colleagues, have assessed the efficacy and safety of exposure-based PTSD treatment in patients with PTSD and BPD and recent suicidal or serious NSSI behaviour (Harned et al., 2012, 2014).

In the first study (Harned et al., 2012), all participants (N = 13) received one year of standard DBT. The prolonged exposure protocol was implemented during this year only if all the following criteria were met: no current risk of suicide, no suicide attempts or NSSI in the past two months, the ability to control life-threatening behaviours, no serious therapy-interfering behaviour, the patient’s first priority target had to be PTSD, and the patient had to be able and willing to experience intense emotions without escaping. Ten participants started the exposure phase on average at Week 18.5. During this phase, two (20%) participants engaged in NSSI, and one of these individuals also made a suicide attempt. PTSD symptom reduction was significant in the post- and follow-up assessments, and revealed large effect sizes.

In the second study (Harned et al., 2014) participants (N = 26) were randomly assigned to DBT either with or without a prolonged exposure (PE) component. The criteria for starting PE were the same as those used in the earlier trial. Only six participants (35%) in the DBT + PE condition completed the treatment. Of these six participants, two (33.3%) had a relapse in intentional self-injury (one suicide attempt, one NSSI event). PTSD symptoms decreased in both conditions, with large effect sizes. Significantly higher improvement was observed in the DBT + PE condition than in the DBT-alone condition.

To examine the efficacy of a DBT treatment designed specifically for patients with CSA-related PTSD and co-morbid BPD features, we conducted a RCT that tested the efficacy of a 12-week modularised DBT programme for PTSD patients who also met at least 4 of the 9 DSM-IV BPD criteria or had certain other diagnoses. The results of this trial are reported elsewhere (Bohus et al., 2013). This programme, titled DBT-PTSD, is a multi-component trauma-focused treatment that combines PTSD-specific interventions with DBT strategies (Steil, Dyer, Priebe, Kleindienst, & Bohus, 2011) with the reduction of PTSD symptoms as one of its main treatment goals. It was conducted as a 12 weeks residential treatment with 2 weekly individual sessions and different group interventions, e.g. DBT skills-training. The DBT-PTSD programme is based on a dynamic treatment hierarchy and contains three treatment phases. In the first phase (three weeks), participants learn to identify their individual avoidance strategies (e.g., NSSI or other non-life-threatening dysfunctional behaviours, emotions, or cognitions) and to use specific DBT skills to control these behaviours. The second phase contains trauma-focused interventions, and aims at reducing PTSD symptoms. Trauma focused interventions include cognitive and exposure therapy. The third phase of treatment combines DBT and trauma-focused interventions as a micro-behavioural analysis is conducted with the goal of finding better strategies to control the dysfunctional behaviour. The third phase focuses on interventions to radically accept the traumatic events.

For safety reasons, as well as due to the lack of empirical data at the time the study was planned, the programme was conducted under residential conditions. As reported in the main publication on this study (Bohus et al., 2013), we found a significant symptom reduction in the treatment group in comparison to the treatment-as-usual wait list (TAU-WL) group with large between-group effect sizes.

The present article presents an evaluation of a subset of the data from this RCT that looks specifically at the relationship between NSSI and exposure interventions. The following research questions were investigated: 1) would the actual occurrence of NSSI events and the urge to commit NSSI increase during exposure-based treatment; and if yes, would these increases differ between participants who had engaged in NSSI at pre-treatment and those who had not? 2) Would participants who had engaged in NSSI pre-treatment and those who had not differ in PTSD symptomatology at post-treatment?

Methods

Participants

Participants in the RCT were females who ranged in age from 17 to 65 years. Inclusion criteria were a DSM-IV diagnosis of CSA-related PTSD and at least one of the following additional diagnoses: eating disorder, current major depression, current substance abuse, or meeting >4 DSM-IV criteria for BPD. The latter inclusion criterion was defined to increase variance in order to study the impact of the number of BPD criteria on treatment outcome in the initial study (Bohus et al., 2013). We followed the recommendation of Skodol et al. (2002) who call a cluster of 4 BPD criteria “borderline features”.

Exclusion criteria were a lifetime diagnosis of schizophrenia, current substance dependence, body mass index <16.5, intellectual disability, medical conditions contradicting the exposure protocol (e.g., severe cardiovascular disorder), and a life-threatening suicide attempt within the last 4 months.
Assessments

At pre-treatment, the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, Williams, & Benjamin, 1997) and the International Personality Disorder Examination (IPDE; Loranger et al., 1994) were used to diagnose Axis I and II disorders. The Severe Behaviour Dyscontrol Interview (SBD-I; Bohus & Borgmann, 2012) was used to collect participants’ history of self-harm behaviours and suicide attempts during the 12 weeks prior to study entry. The SBD-I is a structured interview based on the Suicide Attempt Self-Injury Interview (SASII; Linehan, Comtois, Brown, Heard, & Wagner, 2006). Lifetime history of NSSI was not assessed systematically.

During the treatment period, participants were asked to keep a daily record of the occurrence of NSSI events, the urge for NSSI, and suicidal ideation. NSSI was reported as the number of events, while the other two measures were rated on a 6-point Likert scale from 0 (no urge for NSSI/no suicidal ideation) to 5 (I cannot control my urge/my suicidal ideation and need immediate help).

The PTSD outcome measures were scores on the Clinician-Administered PTSD Scale (CAPS; Blake et al., 2000) and the self-rating Posttraumatic Diagnostic Scale (PDS; Griesel, Wessa, & Flor, 2006).

The severity of depressive symptoms and global psychopathology was measured by Beck’s Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996) and the Symptom Checklist-90-Revised, respectively (SCL-90-R; Derogatis, 1992). The short version of the Borderline Symptom List (BSL-23; Bohus et al., 2009) was used to assess borderline-typical symptoms and NSSI events at post-treatment and at 6- and 12-weeks follow up. The short version of the Dissociative Experiences Scale (DES; Spitzer, Mestel, Klingelhoefer, Gaensicke, & Freyberger, 2004) was used as well.

Procedures

The RCT was conducted at the Central Institute of Mental Health, Mannheim, Germany. Eligible participants were randomised in a 1:1 ratio to either the treatment group (DBT-PTSD) or a treatment-as-usual wait-list group (TAU-WL). Raters remained blinded to the study condition.

In the DBT-PTSD group, data were collected at admission (t1), at discharge (t2), at 6-week follow-up (t3), and at 12-week follow-up (t4). During treatment, the frequency of NSSI, the urge for NSSI, and the intensity of suicidal ideation was assessed daily.

Since the goal of the current analysis was to assess the course of NSSI during DBT-PTSD treatment, only the participants in the DBT-PTSD arm are discussed here.

Statistical methods

Non-parametric tests were used as to analyse within-group and between-group changes for count data. A multivariate analysis of variance with NSSI status at pre-treatment as a fixed factor and PDS and CAPS scores as outcome variables was conducted at four assessment points (pre-treatment, post-treatment, 6-week follow-up, and 12-week follow-up) to evaluate the impact of NSSI at pretreatment on treatment outcome. Analyses were carried out using SAS (v.9.3) and SPSS (v.22).

Results

Study population

A total of 82 female participants were randomly allocated to either the DBT-PTSD or the TAU-WL group, 74 participants started the treatment (for details see Fig. 1). Of these 74 participants, 36 were in the DBT-PTSD arm and 38 were in the TAU-WL arm. Two participants in the DBT-PTSD arm dropped out for personal reasons before the exposure intervention started. Since the present research questions were concerned specifically with individuals who received the full treatment, only the 34 treatment completers are included in the current analyses.

NSSI events and suicidality prior to treatment

No participants had attempted suicide during the 12 weeks prior to randomisation period. With respect to NSSI, participants...
were categorized into two groups based on whether or not they had engaged in NSSI: the “NSSI pre-treatment group” (n = 21; 61.7%) and the “no-NSSI pre-treatment group” (n = 13; 38.2%). Of the participants in the NSSI pre-treatment group, five had been engaging in NSSI more than three times per week. The median rate of NSSI events per week in the NSSI pre-treatment group was .44.

Compared to participants in the no-NSSI pre-treatment group, those who had engaged in NSSI during the 12 weeks prior to study start had experienced their first sexual abuse at a significantly younger age (M = 5.68 years [SD = 2.83] vs. M = 10.42 years [SD = .58]). Participants in the no-NSSI pre-treatment group had a higher PTSD symptom severity which approached significance (two-sided t-test: t, df 32 = 1.9, p = 0.07). No other significant differences between the two groups were found. 90.5% of the participants (n = 19) in the NSSI pre-treatment group fulfilled a BPD diagnosis, in the no-NSSI pre-treatment 15.4% (n = 2) of the participants fulfilled the criteria for a BPD. See Table 1.

**NSSI events during treatment**

In the study sample (n = 34) the rate of participants reporting on-going NSSI decreased from 62% pre-treatment to 38% during treatment (p = .003, McNemar-test). In the NSSI pre-treatment group, n = 9 (42.9%) participants had no NSSI events during the 12 weeks of treatment while n = 12 (57.1%) did, with a mean of M = 1.81 (SD = 3.25) events over the entire treatment period. In the no-NSSI pre-treatment group, just one participant (7.7%) had one NSSI event during treatment (no medical intervention was needed), for a group mean of 0.08 (SD = 0.29) events over the entire treatment period. This participant had a lifetime history of NSSI without NSSI during the 12 weeks prior to randomisation. The number of events during treatment was significantly higher in the NSSI pre-treatment group than in the no-NSSI pre-treatment group (Mann–Whitney U-test, p = 0.013). In the NSSI pre-treatment group, the total number of NSSI events decreased significantly from M = 13.97 (SD = 19.99) pre-treatment to M = 1.81 (SD = 3.25) during treatment (Wilcoxon test, p < 0.001; Cohen’s d = 0.85). According to the BSI, NSSI events also revealed a significant and large reduction from pre-treatment to follow-up (12-weeks follow-up; Wilcoxon test, p < 0.001; Cohen’s d = 1.04). See Table 2.

Analysis on an individual level revealed that only two out of 34 participants (5.9%) had more NSSI events during treatment than during pre-treatment: the above-mentioned patient who had no events pre-treatment but one during treatment, and another who had one event pre-treatment and three during treatment.

There were no suicide attempts during the treatment period in either group.

**The relationship between exposure intervention, NSSI events, urge for NSSI, and suicidality**

No exposure interventions were provided to any participants during the first three weeks. The timing of starting the exposure was individualised, but on average took place in Week 4 and continued, with short interruptions if necessary, until discharge. On average, participants received a total of 4.06 (SD = 2.19) weeks of exposure.

In the NSSI pre-treatment group, the number of participants who engaged in NSSI during treatment dropped from ten (47.6%) during the non-exposure phase to six (21.6%) during the exposure phase. The mean number of NSSI events per patient and week was on average M = 0.33 (SD = 0.42) during the non-exposure phase and M = 0.30 (SD = 0.17) during the exposure phase. No increase in the number of participants with NSSI or in the number of NSSI events per participant was seen during the exposure phase.

In the no-NSSI pre-treatment group, no participants had NSSI events during the exposure phase. The single NSSI event reported in this group occurred prior to the first exposure intervention.

In regards to urge for NSSI, participants in the NSSI pre-treatment group reported a significantly stronger urge than did those in the no-NSSI pre-treatment group during both the non-exposure and the exposure phase (Mann–Whitney U-Test, p = 0.03 for both comparisons). However, within each group, the urge did not differ significantly between the non-exposure and exposure phases. For the NSSI pre-treatment group, the urge for NSSI had a mean rating of 1.83 (SD = 1.31) during the non-exposure phase compared to 2.01 (SD = 1.61) during the exposure phase, while for the no-NSSI pre-treatment group these values were 0.98 (SD = 1.39) and 0.92 (SD = 1.01), respectively. The same pattern was seen for suicidal ideation: in the NSSI pre-treatment group, the mean rating was 0.92 (SD = 1.01) during the non-exposure phase (Mann–Whitney U-Test, p = 0.013; Cohen’s d = 0.85). Of the participants who had engaged in NSSI during the 12 weeks prior to study start had experienced their first sexual abuse at a significantly younger age (M = 5.68 years [SD = 2.83] vs. M = 10.42 years [SD = .58]). Participants in the no-NSSI pre-treatment group had a higher PTSD symptom severity which approached significance (two-sided t-test: t, df 32 = 1.9, p = 0.07). No other significant differences between the two groups were found. 90.5% of the participants (n = 19) in the NSSI pre-treatment group fulfilled a BPD diagnosis, in the no-NSSI pre-treatment 15.4% (n = 2) of the participants fulfilled the criteria for a BPD. See Table 1.

**Table 1**

<table>
<thead>
<tr>
<th>Baseline characteristics</th>
<th>Patients with NSSI pre-treatment (n = 21)</th>
<th>Patients without NSSI pre-treatment (n = 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>34.62 (10.14)</td>
<td>37.62 (11.24)</td>
</tr>
<tr>
<td>p value</td>
<td>0.38</td>
<td></td>
</tr>
<tr>
<td>Number of axis I disorders – current</td>
<td>2.91 (0.94)</td>
<td>3.15 (1.14)</td>
</tr>
<tr>
<td>p value</td>
<td>0.74</td>
<td></td>
</tr>
<tr>
<td>Age at first sexual abuse, years</td>
<td>5.68 (2.83)</td>
<td>10.42 (4.58)</td>
</tr>
<tr>
<td>p value</td>
<td>0.004†</td>
<td></td>
</tr>
<tr>
<td>Number of BPD criteria</td>
<td>4.86 (1.39)</td>
<td>8.8 (1.50)</td>
</tr>
<tr>
<td>p value</td>
<td>0.004†</td>
<td></td>
</tr>
<tr>
<td>Duration of childhood sexual abuse</td>
<td></td>
<td></td>
</tr>
<tr>
<td>p value</td>
<td>0.34</td>
<td></td>
</tr>
<tr>
<td>Relative as abuser, %</td>
<td>52.4%</td>
<td>53.8%</td>
</tr>
<tr>
<td>p value</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Sexual stimulation with penetration, %</td>
<td>71.4%</td>
<td>83.3%</td>
</tr>
<tr>
<td>p value</td>
<td>0.68†</td>
<td></td>
</tr>
</tbody>
</table>

Data are expressed as mean (SD).

- Mann-Whitney U Test.
- Kolmogorov-Smirnoff-Test.
- Fisher’s exact test.

**Table 2**

<table>
<thead>
<tr>
<th>NSSI events pre- and during treatment.</th>
<th>NSSI pre-treatment (N = 21)</th>
<th>No NSSI pre-treatment (N = 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total; M (SD); median</td>
<td>13.97 (19.99); 5.3</td>
<td></td>
</tr>
<tr>
<td>Per week; M (SD); median</td>
<td>1.16 (1.67); 0.4</td>
<td></td>
</tr>
<tr>
<td>NSSI events during treatment period</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total; M (SD); median</td>
<td>1.81 (3.25); 1.0</td>
<td>0.08 (0.28)</td>
</tr>
<tr>
<td>Per week; M (SD); median</td>
<td>0.14 (0.25); 0.1</td>
<td>0.01 (0.02)</td>
</tr>
<tr>
<td>NSSI events based on BSI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-treatment</td>
<td>1.30 (1.34)</td>
<td>0.31 (0.75)</td>
</tr>
<tr>
<td>Post-treatment</td>
<td>0.43 (0.93)</td>
<td>0.23 (0.60)</td>
</tr>
<tr>
<td>6 weeks follow up</td>
<td>0.52 (1.03)</td>
<td>0.31 (0.86)</td>
</tr>
<tr>
<td>12 weeks follow up</td>
<td>0.24 (0.54)</td>
<td>0.38 (1.12)</td>
</tr>
<tr>
<td>Cohen’s d (pre-12 weeks FU)</td>
<td>1.04</td>
<td>0.07</td>
</tr>
</tbody>
</table>
outcome (to assess treatment outcome. Using Pillai’s trace, there was no variance was conducted. Scores of the CAPS and the PDS were used effect of treatment on PTSD symptoms, a multivariate analysis of

NSSI as predictor for PTSD treatment outcome

To examine if the presence of NSSI at pre-treatment predicts the effect of treatment on PTSD symptoms, a multivariate analysis of variance was conducted. Scores of the CAPS and the PDS were used to assess treatment outcome. Using Pillai’s trace, there was no significant effect of NSSI status at pre-treatment on treatment outcome ($A_{\text{Pillai}} = 0.205, F(8,25) = 0.807, p = 0.603$). See Table 3.

Discussion

This study investigated the course of NSSI on a 12-week exposure-based treatment (DBT-PTSD) in patients with PTSD and borderline features. NSSI decreased significantly during treatment and rates of NSSI were similar between treatment weeks with vs. without exposure. Treatment outcome on PTSD symptoms was comparable regardless of whether or not participants had engaged in NSSI beforehand. This finding and the high completion rate of 94% indicate a high tolerability of the treatment in participants with NSSI. The high completion rate is in line with previous studies indicating no difference in dropout rates between exposure therapy and other treatments (Hembree et al., 2003).

In the subgroup of patients who had engaged in NSSI prior randomisation, both the number of participants with NSSI and the number of NSSI events decreased significantly during treatment in comparison to pre-treatment. Importantly, the reduction in NSSI events remained stable at follow-up. During treatment, there was no significant difference between weeks with vs. without exposure interventions in terms of the number of patients who engaged in NSSI, the number of NSSI events, the urge for NSSI, and suicidal ideation. With regard to PTSD treatment outcome, patients who engaged in NSSI pre-treatment showed a comparable response to those who did not.

In the studies by Harned et al. (2012; 2014) 25–27% of the participants re-engaged in NSSI during the exposure treatment, whereas in the present study one patient (7%) without NSSI pre-treatment re-engaged in NSSI. In terms of urge for self-harm and suicidal ideation, we did not observe any increase in relationship with exposure, which is in line with previous findings (Harned et al., 2012, 2014).

Our data indicate that DBT-PTSD can be carried out safely under residential conditions, even before full behavioural control over NSSI has been reached. It is important to note, that these results achieved under residential conditions cannot be generalized to an outpatient setting without further studies.

Additionally, the quick reduction in NSSI could have been influenced by the residential setting in terms of e.g. the impact of stimulus control; therapeutic support; or anticipated aversive consequences in the form of treatment rules which e.g. require written behavioural analyses following NSSI (for details see Bohus et al., 2000). As discussed by Harned et al. (2012), a key mechanism might also be the strong motivation of the participants to receive PTSD treatment; therefore behavioural control was a strict condition for receiving the exposure protocol in this study. That same strategy was used in the DBT-PTSD protocol, with the start of the exposure phase varying between Weeks 4 and 6 for different participants. Other underlying mechanisms, such as improved emotion regulation strategies, are possible mediating factors as well.

While our study does not indicate an exacerbation of NSSI during treatment, it has to be kept in mind that this finding is based on a fairly small sample size and requires replication. The fairly small sample further resulted in limited statistical power. Accordingly, the absence of evidence with respect to exacerbation of NSSI in our study is not sufficient to conclude that such a risk is low or even nonexistent. The same limitation must be kept in mind when interpreting our finding that participants who had vs. had not engaged in NSSI pre-treatment did not differ in treatment outcome. Also, as two patients in the NSSI pre-treatment subgroup do not fulfil a BPD diagnosis, our results need to be replicated in a sample of individuals with comorbid PTSD and full BPD.

Despite these limitations, the present study is one of the first to investigate NSSI in an exposure-based PTSD treatment. In further studies the examination of triggers for NSSI during treatment phases with and without exposure-based interventions would increase the understanding of NSSI in patients with CSA-related PTSD and co-morbid BPD symptoms.

Table 3

MANOVA for NSSI pre-treatment on therapy outcome.

<table>
<thead>
<tr>
<th></th>
<th>Patients with NSSI pre-treatment ($n = 21$)</th>
<th>Patients without NSSI pre-treatment ($n = 13$)</th>
<th>$F$ (df); $p$</th>
<th>Overall analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-treatment</td>
<td>Pre-treatment</td>
<td>$3.66 (1,32); p = 0.065$</td>
<td>$A_{\text{Pillai}} = 0.205$</td>
</tr>
<tr>
<td></td>
<td>Post-treatment</td>
<td>Post-treatment</td>
<td>$0.33 (1,32); p = 0.569$</td>
<td>$F(8,25) = 0.807$</td>
</tr>
<tr>
<td></td>
<td>6 weeks follow-up</td>
<td>6 weeks follow-up</td>
<td>$0.95 (1,32); p = 0.337$</td>
<td>$p = 0.603$</td>
</tr>
<tr>
<td></td>
<td>12 weeks follow-up</td>
<td>12 weeks follow-up</td>
<td>$1.04 (1,32); p = 0.316$</td>
<td>$e$</td>
</tr>
<tr>
<td><strong>CAPS</strong></td>
<td>Pre-treatment</td>
<td>Pre-treatment</td>
<td>$3.66 (1,32); p = 0.065$</td>
<td>$A_{\text{Pillai}} = 0.205$</td>
</tr>
<tr>
<td></td>
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<td>$e$</td>
</tr>
<tr>
<td><strong>PDS</strong></td>
<td>Pre-treatment</td>
<td>Pre-treatment</td>
<td>$1.11 (1,32); p = 0.300$</td>
<td>$A_{\text{Pillai}} = 0.205$</td>
</tr>
<tr>
<td></td>
<td>Post-treatment</td>
<td>Post-treatment</td>
<td>$1.39 (1,32); p = 0.247$</td>
<td>$F(8,25) = 0.807$</td>
</tr>
<tr>
<td></td>
<td>6 weeks follow-up</td>
<td>6 weeks follow-up</td>
<td>$2.53 (1,32); p = 0.122$</td>
<td>$p = 0.603$</td>
</tr>
<tr>
<td></td>
<td>12 weeks follow-up</td>
<td>12 weeks follow-up</td>
<td>$2.43 (1,32); p = 0.129$</td>
<td>$e$</td>
</tr>
</tbody>
</table>

$^a$ Data are expressed as mean (SD).
A better knowledge of mediating and moderating factors could enable us to individualise and improve our treatments. In order to expand our findings beyond those that apply to the residential setting, we are currently conducting a large RCT in which DBT-PTSD is being delivered under outpatient conditions.

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References


