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Reduction of depersonalization during social stress through cognitive therapy for social anxiety disorder: A randomized controlled trial

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ABSTRACT

Symptoms of depersonalization during feared social situations are commonly experienced by individuals with social anxiety disorder (SAD). Despite its clinical relevance, it is not addressed in standard treatment manuals and it remains unclear if depersonalization is reduced by well-established treatments. This study investigated whether cognitive therapy (CT) for SAD effectively reduces depersonalization and whether pre-treatment severity of depersonalization predicts or mediates treatment outcome.

In a randomized controlled trial, patients underwent the standardized Trier Social Stress Test before and after CT ($n = 20$) or a waitlist period ($n = 20$) and were compared to healthy controls ($n = 21$). Self-reported depersonalization was measured immediately after each stress test.

Depersonalization significantly decreased following CT, especially in treatment responders ($\eta_p^2 = 0.32$). Pre-treatment depersonalization did neither predict nor mediate post-treatment severity of social anxiety.

Further prospective studies are needed for a better scientific understanding of this effect. It should be scrutinized whether SAD-patients suffering from depersonalization would benefit from a more specific therapy.

1. Introduction

Patients with social anxiety disorder (SAD) suffer from persistent anxiety of one or more social or performance situations in which they fear negative evaluation or rejection by others. Within these social situations, patients with SAD typically experience a range of emotional symptoms such as feeling anxious and embarrassed or bodily symptoms such as sweating or blushing (American Psychiatric Association [APA], 2013). Past research provided consistent evidence that cognitive therapy (CT) is generally effective in the treatment of SAD (e.g., Clark et al., 2003, 2006; Hofmann, Asnaai, Vonk, Sawyer, & Fang, 2012; Mörtberg, Clark, & Bejerot, 2011). It also reduces specifically targeted symptoms such as fear of blushing (Härtling, Klotsche, Heinrich, & Hoyer, 2015). However, other SAD symptoms such as depersonalization and derealization (see Hoyer, Braeuer, Crawcour, Klumbies, & Kirschbaum, 2013) are not directly addressed by contemporary psychological models of SAD (e.g., Wong, Gordon, & Heimberg, 2014). Even the comprehensive model of Hofmann (2007) which focuses on the cognitive factors (e.g., overestimation of negative consequences of social situations) that maintain SAD does not emphasize or directly target depersonalization. Thus, it remains unclear to which extent these symptoms are reduced by contemporary treatments for SAD.

Depersonalization belongs to the class of dissociative symptoms and describes an experience of unreality, detachment, or being an outside observer with respect to one self's feelings, thoughts and sensations (APA, 2013). Derealization characterizes the subjective sensation of being disconnected to the outside world (APA, 2013). Due to their overlap with regards to content and to their intertwined occurrence (Michal, Sann, Grabhorn, Overbeck, & Rödler, 2005; Sierra & Berrios, 2001), depersonalization and derealization are summarized as "depersonalization" in the following. Depersonalization may occur in healthy individuals under mental distress (Michal et al., 2014) or the influence of drugs (Mathew, Wilson, Humphreys, Lowe, & Weithe, 1993). It is also associated with different mental disorders such as panic disorder (Mendoza et al., 2011; Segui et al., 2000) or borderline personality disorder (Zanarini, Frankenburg, Jager-Hyman, Reich, & Fitzmaurice, 2008), and is

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the predominant syndrome in Depersonalization-Derealization-Disorder (Hunter, Philipps, Chalder, Sierra, & David, 2003).

Moreover, intense experiences of depersonalization are frequently experienced by patients with SAD, especially under acute social stress (Gül, Simsek, Inanir, & Karaaslan, 2014; Hoyer et al., 2013; Kamaradova, Prasko, Sandolva, & Latalova, 2014). In particular, patients who predominantly experience severe cardiac sensations and a high level of paresthesia in social situations suffer from depersonalization which has been linked to higher performance anxiety and a co-occurring chronic worry (Potter, Drabick, & Heimberg, 2014). Importantly, the occurrence of depersonalization under social stress seems to be associated with an elevated use of safety behaviors and more pronounced post-event processing in patients with SAD (Hoyer et al., 2013). This link between depersonalization and these dysfunctional strategies may in turn contribute to the maintenance of SAD (McManus, Sacadura, & Clark, 2008) and may even worsen response to treatment (Price & Anderson, 2011).

Furthermore, depersonalization may even directly impair corrective learning during treatment. For example, emotional learning processes are reduced during dissociative experiences (Ebner-Priemer et al., 2009), which may explain why the presence of dissociation is associated with poor response to psychotherapy in various mental disorders (Kleindienst et al., 2011; Michelson, June, Vives, Testa, & Marchione, 1998; contradictive results in patients with posttraumatic stress disorder: Halvorsen, Stenmark, Neuner, & Nordahl, 2014). As depersonalization is pronounced under acute social stress, it is likely to occur during social exposure tasks used in CT for SAD. Thus, the unnoticed occurrence of depersonalization may prevent relevant learning experiences and thereby impair successful treatment. However, no study so far has investigated the change of depersonalization in SAD following CT or the mediational and predictive value of pre-treatment depersonalization for treatment outcome. As patients with SAD experience depersonalization mostly in intense anxiety-provoking situations, measuring these symptoms and their change after therapy is a complex methodological challenge. Retrospective self-reports may misjudge the true severity and be biased due to retrospective recall (Leising, 2011; Sadler & Woody, 2003). To this end, all participants of the present study indicated their depersonalization symptoms immediately after completing the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), which is a standardized paradigm to provoke social stress. The present study tested the following hypotheses: 1) CT reduces depersonalization symptoms during acute social stress in patients with SAD. 2) Depersonalization a) mediates treatment outcome and b) predicts worse treatment outcome.

2. Material and methods

2.1. Participants

Inclusion criteria for patients were a principal diagnosis of SAD (assessed with Munich-Composite International Diagnostic Interview; DIA-X/M-CIDI, Wittchen & Pfister, 1997) and a total score higher than 30 on the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987; German version: Stangier & Heidenreich, 2005). Exclusion criteria for the SAD patients were co-morbid substance related disorders, personality disorders (except avoidant, dependent or obsessive-compulsive disorder), psychotic or organic mental disorders, current psychotherapy or intake of any ataractics. Inclusion criteria for healthy controls were being 18 years or older, no lifetime psychiatric disorder (assessed with the DIA-X/M-CIDI), and a LSAS total score below 30. SAD patients were recruited from the outpatient clinic of the Institute of Clinical Psychology and Psychotherapy of the Technische Universität Dresden (Germany). Healthy controls were recruited via flyers and advertisements in local newspapers. In accordance with Cohen (1988), we strived for a sample size of $n = 30$ per group to obtain a moderate effect size. Participants were included into the study from October 2009 to August 2011. During the survey and funding period the number of patients in the outpatient clinic was lower than usual and a surprisingly high number of patients declined to participate because of the unpleasant nature of the TSST. For this reason, the group sizes were lower than previously envisaged.

The study was approved by the local ethics committee (EK137062007) and participants gave written informed consent. The trial was registered on the German Clinical Trials Register (registration number: DRKS00009741).

Following inclusion into the study, SAD patients were randomly assigned to two groups of equal size: a treatment group and a waitlist control group. Randomisation using a binary allocation sequence was carried out blind by a member of staff from another department within the Technische Universität Dresden. As Fig. 1 shows, the present sample consisted of 20 patients in the waitlist control group (female $n = 8$, age: $M = 26.05$, $SD = 3.56$), 20 patients in the treatment group ($n = 8$ female, $M_{age} = 24.60$, $SD = 5.19$) as well as 21 healthy controls ($n = 9$ female, $M_{age} = 26.38$, $SD = 7.52$) who completed the pre- and post-treatment measures. The groups did not differ in age, $H(21, 20, 20) = 2.74$, $p = 0.250$, or sex distribution, $\chi^2_{Fischer}(2, 21, 20, 20) = 0.11$, $p = 0.491$. Women were tested in the luteal phase of their menstrual cycle.

2.2. Measures

One week before each TSST, the German LSAS (Stangier & Heidenreich, 2005) was conducted via telephone to assess the severity of social anxiety in social interactions and performance situations. Directly after each TSST, participants filled out an adapted version of the Cambridge Depersonalisation Scale (CDS; Sierra & Berrios, 2000; German version: Michal et al., 2004, adapted version: Hoyer et al., 2013) to measure the intensity of depersonalization experiences during the period of acute social stress. The 29 items of the original CDS assess the frequency and duration of depersonalization during the last six months. The questionnaire includes items concerning depersonalization and derealization as well as items that ask for micropsia, autoscopy, déjà vu, and out-of-body experiences (Michal et al., 2004; Sierra & Berrios, 2000). It should be mentioned that both the authors of the English as well as the authors of the German version do not consider depersonalization and derealization as independent phenomena and therefore do not provide separate subscales for them (Michal et al., 2004; Sierra & Berrios, 2000).

Hoyer et al. (2013) slightly modified the introduction and the items to assess depersonalization as a state variable. The 15 items of the adapted CDS were rated on a visual analog scale ranging from 0 (*none, never, not at all*) to 100 (*very strong, always*). A mean score for all items was used for analyses. Excellent reliability (internal consistency $\alpha = 0.95$ and Guttman split half reliability coefficient $r_{tt} = 0.95$) and good validity coefficients (high significant correlations with the Dissociative Experience Scale (Bernstein & Putnam, 1986; German: Freyberger, Spitzer, & Stieglitz, 1999)) have been reported for the original German trait version (Michal et al., 2004). The state version showed good internal consistency ($\alpha = 0.87$ in 23 respondents of the TSST; Hoyer et al., 2013). In the present study, the internal consistency was $\alpha = 0.93$ (both on the first measurement [before CT/waiting time] and on the second measurement [after CT/4-6 month waiting time]). The Guttman split half reliability coefficient was $r_{tt} = 0.87$.

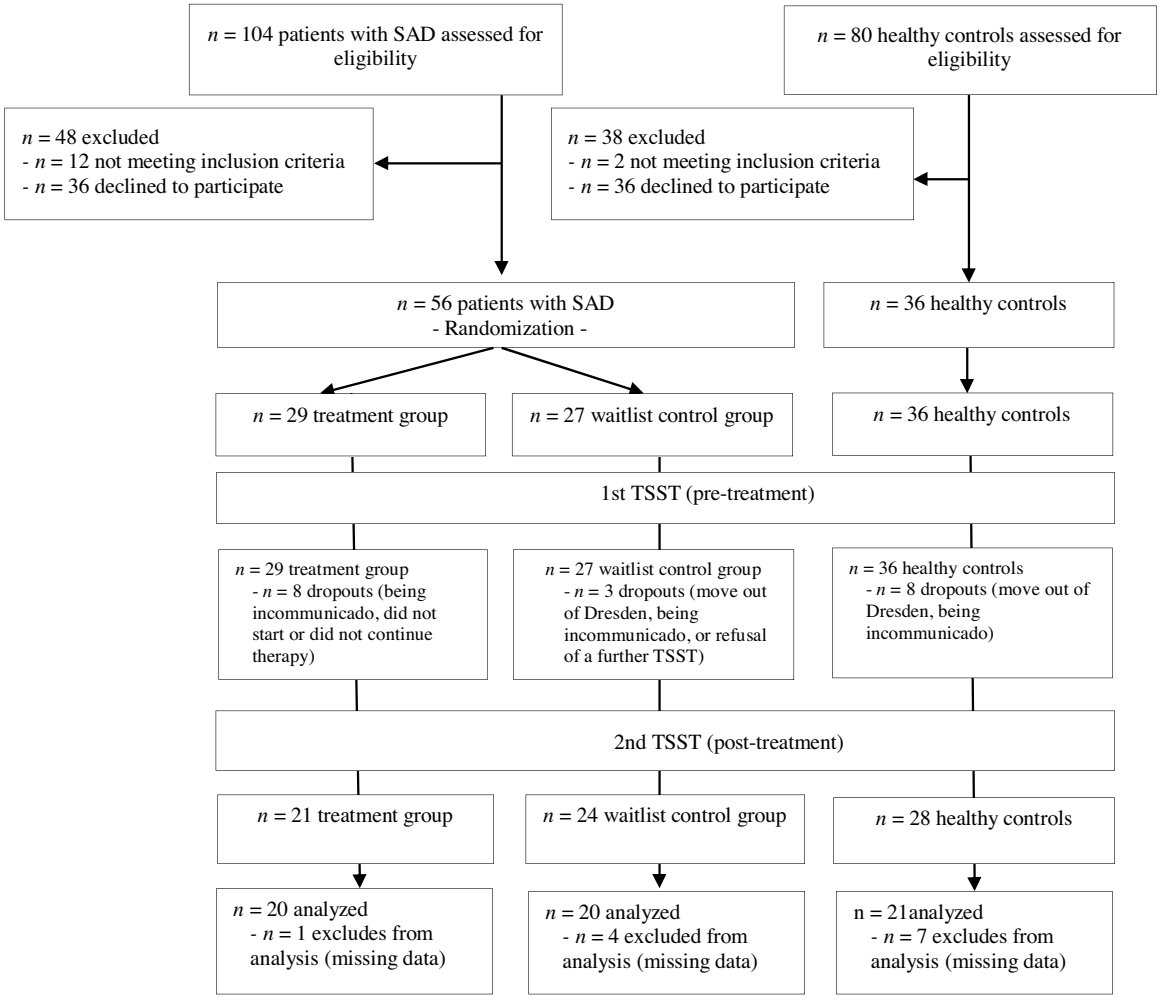

Fig. 1. Flow diagram.

2.3. Procedure and treatment

All participants repeatedly underwent the Trier Social Stress Test (TSST; Kirschbaum et al., 1993), a standardized public speaking task, before and after treatment or before and after waiting time. Similar to treatment duration, the waiting period within the waitlist group varied between four and six months. The TSST comprises a preparation time, a short speech and a mental arithmetic task in front of a two-person mixed-gendered committee. The setting of the TSST was changed from first to second assessment to minimize habituation. Severity of self-reported depersonalization during the TSST was evaluated immediately after each TSST.

The treatment comprised up to 25 sessions of individual CT according to the manual of Stangier, Clark, and Ehlers (2006) and was carried out between October 2009 to June 2012 at the Institute of Clinical Psychology and Psychotherapy of the Technische Universität Dresden (Germany). Sessions were delivered either in a 50 min or 100 min format. The manual of Stangier et al. (2006) is based on the cognitive model of SAD (Clark & Wells, 1995) and includes components such as an idiosyncratic model of SAD, discussion of self-focused attention and safety behavior as well as role play-based behavioral experiments. Importantly, depersonalization is not mentioned in this manual. Treatment was performed by psychotherapists in training who received a one-day manual-training and were supervised every fourth session. The present data are part of a larger clinical trial investigating the stress response of patients with SAD before and after CT (see Hoyer et al., 2013; Klumbies, Braeuer, Hoyer, & Kirschbaum, 2014). In addition to further subjective measures (see Hoyer et al., 2013), heart rate was continuously recorded and saliva as well as blood samples were taken eight times before and after each TSST to assess cortisol levels. As this article specifically focuses on change of depersonalization through CT in SAD, results of physiological and additional subjective measures have already been (see Hoyer et al., 2013; Klumbies et al., 2014) or will be reported elsewhere.

2.4. Statistical analysis

Main treatment outcome as defined by reduction in LSAS scores was analyzed with repeated-measures ANOVAs with the LSAS sum score as dependent variable and Group as between-subject factor and Time as within-factor. Response to treatment, as defined by Leichsenring et al. (2013), was determined as a reduction of LSAS scores of 31% or higher of the pre-to the post-treatment value. CDS pre- and post-treatment scores were logarithmized to compensate for strong deviations from the normal distribution (CDS_{log}). To quantify treatment changes in the severity of self-reported depersonalization across the factor Time (pre- vs. post-treatment), repeated measures ANOVAs were calculated for 1) treatment group compared to waitlist control group, 2) treatment responders compared to waitlist control, and 3) treatment responders compared to non-responders as the between-participants factor. ANOVAs were carried out separately due to different theoretical implications of their results. In addition, comparisons with healthy controls were

Table 1
Means and standard deviations of the social anxiety and the depersonalization self-reports in treatment and comparison groups (pre- and post-treatment/wait period).

	TG		WCG		HC	
	pre	post	pre	post	pre	post
LSAS	71.70 (18.63)	42.80 (15.20)	59.90 (18.65)	63.35 (20.69)	9.43 (6.28)	7.38 (6.90)
CDS_{log}	4.33 (0.71)	3.33 (1.13)	4.32 (0.67)	4.01 (0.80)	3.16 (1.15)	2.66 (1.07)

Notes: TG = treatment group, WKG = waitlist control group, HC = healthy controls, LSAS = Liebowitz Social Anxiety Scale, CDS_{log} = logarithmized value of the adapted Cambridge Depersonalisation Scale, pre = pre-treatment/therapy wait period, post = post-treatment/therapy wait period.

conducted to investigate a potential normalization of depersonalization. In case of violation against sphericity, Greenhouse-Geisser corrections were applied.

Spearmen's correlations were calculated to test the relationship between the severity of depersonalization (CDS_{log}) and the severity of social anxiety (LSAS) in the treatment group. To examine whether the pre- to post-treatment change of depersonalization and social anxiety were associated, correlations between difference-scores (pre-treatment minus post-treatment) of the LSAS score and CDS_{log} were calculated.

To determine whether depersonalization (CDS_{log} pre-treatment, measured after the TSST) mediates treatment outcome, three separate linear regression models were planned to be conducted within the treatment group: First, a regression analysis whether the LSAS pre-treatment score (measured before the TSST) predicts the LSAS post-treatment score; second, whether the LSAS pre-treatment score predicts the CDS_{log} pre-treatment score (measured after the TSST) and third, whether CDS_{log} pre-treatment score predicts the LSAS post-treatment score. To analyze whether the statistical power was sufficient to detect the supposed association between pre and post values, we a) calculated the Pearson correlation with its 95% confidence interval which indicates the range of values that are in line with the data and b) calculated how large the sample correlation had to be at least to be found statistically significant.

To test the hypothesis that depersonalization is a predictor of a worse treatment outcome, we performed a linear regression with the CDS_{log} pre-treatment score as a predictor of therapy outcome (LSAS post-treatment). LSAS pre-treatment score was also entered as a predictor to control for pre-treatment social anxiety symptom severity.

3. Results

3.1. Descriptive statistics

Table 1 shows the descriptive results of social anxiety and depersonalization severity before and after treatment for the different groups.

3.2. Main therapy outcome

The repeated measures ANOVA with Group (treatment group, waitlist control group, healthy controls) and Time (pre vs. post) revealed a main effect of Time, $F(2, 58) = 20.62$, $p < 0.001$, and Group, $F(2, 58) = 103.18$, $p < 0.001$, as well as a significant Group x Time interaction effect, $F(2, 58) = 24.23$, $p < 0.001$, $\eta_p^2 = 0.46$. Before treatment, LSAS scores were significantly higher in patients with SAD compared to healthy controls, $t(52.14) = -13.00$, $p < 0.001$, $d = 2.65$, whereas treatment group and waitlist control group did not differ, $t(38) = -2.00$, $p = 0.052$, $d = 0.63$. After treatment, 60% ($n = 12$) participants of the treatment group (and $n = 2$ [10%] in waitlist control group) fulfilled the criterion for therapy-response (as defined in Leichsenring et al., 2013). The LSAS post-treatment scores of treatment responders (CDS_{log} score pre-treatment: $M = 4.45$, $SD = 0.58$; post-treatment: $M = 3.05$, $SD = 1.12$) significantly changed pre- to post-treatment, $U(12, 12) = -3.06$, $p = 0.002$, $d = 2.85$, and were significantly lower compared to the waitlist control group, $U(12, 20) = -3.51$, $p < 0.001$, $d = -0.18$, however, treatment non-responders (pre-treatment CDS_{log} score: $M = 4.15$, $SD = 0.88$; post-treatment CDS_{log} score: $M = 3.74$, $SD = 1.08$) and waitlist control group did not differ significantly, $U(8, 20) = 1.45$, $p = 0.15$, $d = 1.34$.

3.3. Changes in depersonalization

Fig. 2 indicates that every group experienced a decrease in the severity of depersonalization pre- to post-treatment. However, the change in treatment responders is clearly the largest.

3.3.1. Treatment group vs. waitlist control group

Examining the pre-treatment CDS_{log} score, patients with SAD reported a significantly higher score compared to healthy controls, $U(40, 21) = -4.14$, $p < 0.001$, $d = 1.23$. No differences were found between treatment group and waitlist control group before treatment, $t(37.87) = 0.05$, $p = 0.964$. Table 1 indicates that every group experienced a decrease in the severity of depersonalization from pre- to post-treatment. The repeated-measures ANOVA revealed a significant effect of Time, $F(1, 38) = 23.35$, $p < 0.001$, no significant effect of Group, $F(1, 38) = 2.14$, $p = 0.148$, and a significant interaction effect of Time x Group, $F(1, 38) = 6.59$, $p = 0.014$, $\eta_p^2 = 0.15$. Treatment group and waitlist control did not differ at the pre-treatment measuring time, $t(38) = 0.05$, $p = 0.964$, $d = 0.01$, but the treatment group reported significantly lower depersonalization scores after treatment compared to the waitlist controls, $t(38) = 2.22$, $p = 0.033$, $d = 0.43$.

3.3.2. Treatment responders vs. waitlist control group

For the sake of completeness and to show that the effect in the group of treatment responders is caused by a therapy effect rather than by habituation, we calculated repeated-measures ANOVA with treatment responders and waitlist controls as the between-participants factor. There was a significant effect of Time, $F(1, 30) = 34.64$, $p \leq 0.001$, no significant effect of Group, $F(1, 30) = 2.72$, $p = 0.110$, and a significant interaction effect of Time x Group, $F(1, 30) = 14.23$, $p = 0.001$, $\eta_p^2 = 0.32$. While these groups did not differ at pre-treatment, $t(30) = 0.56$, $p = 0.579$, $d = 0.18$, the treatment responders had a significantly lower score at post-treatment, $t(20) = -2.84$, $p = 0.008$, $d = 1.11$.

3.3.3. Treatment responders vs. treatment non-responders

In order to examine whether treatment responders had a larger reduction in depersonalization compared to treatment non-responders, a further repeated-measures ANOVA with treatment responders and treatment non-responders as the between-participants factor was calculated. This analysis revealed a significant effect of Time, $F(1, 18) = 16.94$, $p = 0.001$, no significant effect of Group, $F(1, 18) = 0.30$, $p = 0.590$, and a significant interaction effect of Time x Group, $F(1, 18) = 4.89$, $p = 0.040$, $\eta_p^2 = 0.47$. Treatment responders and treatment non-responders did not differ at pre-treatment, $U(8, 12) = 0.77$, $p = 0.44$, $d = 0.42$, nor at post-treatment, $U(8, 12) = -1.39$, $p = 0.181$, $d = 0.63$, $CI = -0.30$ to 1.54.

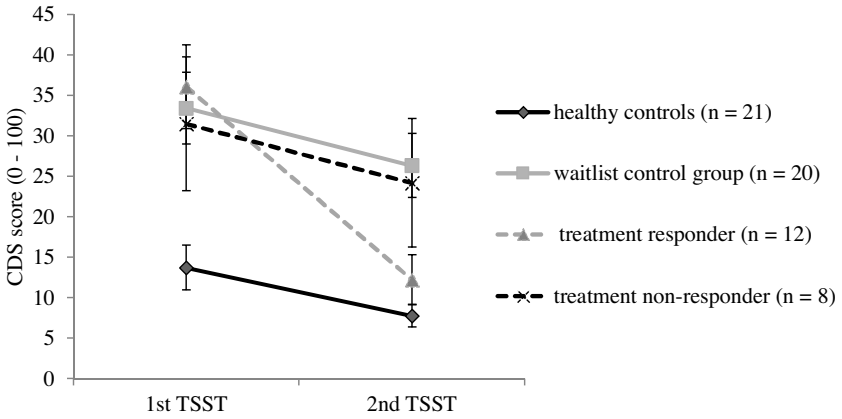

Fig. 2. Means (± standard error) in the Cambridge Depersonalisation Scale, in the first and second Trier Social Stress Test (Kirschbaum et al., 1993) in therapy-non-responders, waitlist control group and healthy controls. For treatment responders and non-responders, TSSTs were completed before (1st TSST) and after treatment (2nd TSST). For waitlist control and healthy control individuals, TSSTs were completed before (1st TSST) and after a 4–6 month waiting period without treatment (2nd TSST).

However, the CDS_{log} score of the treatment responder changed significantly pre- to post-treatment, $U(12, 12) = -3.06$, $p = 0.002$, $d = 1.57$, while the CDS_{log} score of the treatment non-responders did not, $U(8, 8) = -1.40$, $p = 0.161$, $d = 0.42$.

An additional analysis showed that treatment non-responders had a significantly higher CDS_{log} score compared to healthy controls at the post-treatment, $U(8, 21) = -2.00$, $p = 0.047$, $d = 0.91$, but treatment responders and healthy controls did not differ at post-treatment $U(12, 21) = -1.09$, $p = 0.291$, $d = 1.31$.

3.4. Relationship between severity of depersonalization and social anxiety

In the treatment group, significant correlations were found between CDS_{log} pre- and CDS_{log} post-treatment scores, $r = 0.49$, $p = 0.025$, as well as between CDS_{log} post-treatment and LSAS post-treatment, $r = 0.55$, $p = 0.012$. The correlation between CDS_{log} pre-treatment and LSAS pre-treatment was not significant, $r = 0.41$, $p = 0.070$. We found no significant correlation between the LSAS pre- and LSAS post-treatment score, $r = 0.11$, $p = 0.641$. No significant correlation was found between the difference scores of LSAS (LSAS pre-treatment – LSAS post-treatment) and CDS_{log} (CDS_{log} pre-treatment – CDS_{log} post-treatment), $r = 0.27$, $p = 0.261$, in the treatment group.

In the treatment group the LSAS pre-treatment score could not be identified as a significant predictor of the LSAS post-treatment score within the treatment group, $F(1,20) = 2.34$, ß = 0.34, $p = 0.143$, rendering all further mediation analyses obsolete (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). The result that we cannot find evidence for our hypothesis that pretreatment depersonalization, measures with the CDS_{log} score, mediates the treatment outcome is, however, largely due to an insufficient sample size: For the given sample size and a two-sided $\alpha = 0.05$, the true correlation has to be at least $r = 0.59$ to be found with a power of $1 - ß = 0.80$. Increasing the power to $1 - ß = 0.90$ would even require a correlation of at least $r = 0.66$ (calculated with the command POWER in Stata 14.1).

In a further linear regression within treatment group, the CDS_{log} pre-treatment score, controlled for the LSAS pre-treatment score, did not significantly predict a higher LSAS post-treatment score, $F(2,20) = 1.755$, ß = 0.25, $p = 0.203$, $R^2_{adjusted} = 0.074$, $\Delta R^2 = 0.056$, although it accounted for more than 5% of the LSAS post-treatment variance.

4. Discussion

To our knowledge, the present study is the first to show that CT reduces the severity of depersonalization during acute social stress in patients with SAD. Before CT, depersonalization was higher in patients with SAD compared to healthy controls. After treatment, severity of depersonalization normalized for treatment responders. The present findings are noteworthy as neither the psychoeducation about depersonalization symptoms nor their direct treatment are an explicit treatment component; neither in the Clark and Wells' model used in the treatment manual of the present study (Stangier et al., 2006) nor in the more recent model of Hofmann (2007).

There are several explanations why manualized CT results in a decrease in depersonalization: Firstly, the manual includes psychoeducation which aimed at counteracting the catastrophic misinterpretation of bodily and mental symptoms. Patients may be able to transfer what they have learned about other symptoms to their experiences of depersonalization, thereby reducing catastrophic interpretations about depersonalization. Due to the fact that catastrophic misinterpretation of depersonalization is known as a prominent mechanism of development and maintenance of depersonalization (Hunter et al., 2003; Hunter, Salkovskis, & David, 2014), reducing this over-interpretation may contribute to a reduction of these symptoms. However, some patients might not have been able to perform this transfer themselves. Thus, future research is necessary to investigate whether such patients may benefit from a more targeted therapy that comprises, e.g., psychoeducation about depersonalization.

Another possible explanation for the reduction of depersonalization may be that depersonalization symptoms are merely an expression of severe social anxiety and thus show a concurrent decrease. However, depersonalization symptoms were not significantly correlated with the severity of social anxiety before treatment. More importantly, decreases in depersonalization and in social anxiety due to CT were also not correlated. These findings may suggest that depersonalization is not just an indicator of anxiety severity but rather a distinct aspect of social anxiety that needs to be better integrated into models of social anxiety disorder.

Depersonalization might be a hidden factor that explains the relative high number of treatment non-responders found in our own and several other studies of social phobia (Davidson et al., 2004; Stangier, Heidenreich, Peitz, Lauterbach, & Clark, 2003). The fact that the severity of depersonalization did neither mediate nor predict the therapy outcome is in line with the findings of Halvorsen et al. (2014), but contradicts previous evidence that depersonaliza-

tion is a predictor of a worse therapy outcome (Kleindienst et al., 2011; Michelson et al., 1998). It needs, however, to be emphasized that our results should be interpreted with caution as the statistical power was shown to be insufficient to detect small and medium effects. A higher severity of depersonalization before treatment was significantly associated with a higher degree of post-treatment social anxiety (on a bivariate level). In addition, although not statistically significant, depersonalization before treatment accounted for more than an additional 5% of the variance in social anxiety after treatment when pre-treatment social anxiety was controlled. It is likely that our study was underpowered to detect potential effects of depersonalization on the therapeutic efficacy of the CT manual. Given these results, our findings need to be replicated within a larger sample to address the question of whether depersonalization should be specifically targeted in treatment non-responders.

As shown in the study by Potter et al. (2014), depersonalization occurs predominantly in clusters of patients with SAD that fall in the category of performance anxiety. Further research is needed to scrutinize the occurrence, characteristics and consequences of depersonalization in the new DSM-5 subtype of "performance only" social anxiety disorder (APA, 2013). Moreover, studies comprising follow-up data are required to investigate whether depersonalization influences the relapse-rate after psychotherapy for SAD (see Michelson et al., 1998).

One shortcoming of our study is that the measure used to determine the severity of depersonalization (adapted version of the CDS; Hoyer et al., 2013) does not differentiate between depersonalization and derealization symptoms. Although several studies report a high overlap between these symptoms (Michal et al., 2005; Sierra & Berrios, 2001), we could not analyze this overlap in the current sample or perform separate analyses for subscales, but only report data that merged these symptoms. Future research may scrutinize whether there are differences in the response to CT between depersonalization and derealization.

In summary, little is known to date about depersonalization in SAD even though these symptoms are frequently reported by patients with SAD. A successful CT can significantly reduce the severity of self-reported depersonalization in SAD. Our results suggest that these symptoms are more than a mere indicator of overall symptom severity. No proof was discovered that the severity of pre-treatment depersonalization predicts therapy outcome if pre-treatment symptom severity was controlled for.

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Conflict of interest

None.

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