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Harm expectancy violation during exposure therapy for posttraumatic stress disorder

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ABSTRACT

Exposure therapy has proven efficacy in the treatment of posttraumatic stress disorder (PTSD). Emotional processing theory proposes that fear habituation is a central mechanism in symptom reduction, but the empirical evidence supporting this is mixed. Recently it has been proposed that violation of harm expectancies is a crucial mechanism of action in exposure therapy. But to date, changes in harm expectancies have not been examined during exposure therapy in PTSD. The goal of the current study was to examine harm expectancy violation as mechanism of change in exposure therapy for posttraumatic stress disorder (PTSD). Patients (N = 50, 44 female) with a primary diagnosis of chronic PTSD received intensive exposure therapy. Harm expectancies, harm experiences and subjective units of distress (SUDs) were assessed at each imaginal exposure session, and PTSD symptoms were assessed pre- and posttreatment with the Clinician Administered PTSD Scale (CAPS). Results showed that harm expectancies were violated within and strongly declined in-between exposure therapy sessions. However, expectancy violation was not related to PTSD symptom change. Fear habituation measures were moderately related to PTSD symptom reductions. In line with theory, exposure therapy promotes expectancy violation in PTSD patients, but this is not related to exposure therapy outcome. More work is warranted to investigate mechanisms of change during exposure therapy in PTSD.

1. Introduction

Exposure therapy has proven efficacy for the treatment of posttraumatic stress disorder (PTSD). In emotional processing theory (Foa & Kozak, 1986), it has been argued that fear habituation[1] both within and between sessions, denotes exposure therapy success. However, studies investigating the predictive value of fear habituation (as indexed by a decrease in subjective units of distress (SUD) ratings) for treatment outcome have yielded mixed findings (Bluett, Zoellner, & Feeny, 2014; Rauch, Foa, Furr, & Filip, 2004; Sripada & Rauch, 2015). Extinction learning is thought to be one of the mechanisms of action in exposure therapy, and refers to the process in which a conditioned stimulus (CS; i.e., a trauma reminder) is repeatedly presented in the absence of the unconditioned stimulus (US; i.e., the traumatic experience) thereby leading to reduction of the conditioned response (CR; i.e., fear). It is now believed that extinction learning is not so much the deletion of the original CS-US association, but rather a new learning of a CS – No US relationship (Bouton, 1993), referred to as inhibitory learning (Craske et al., 2008). According to the theory of inhibitory learning, extinction occurs after a mismatch between the expectancy of an aversive event and the absence of its occurrence (Rescorla & Wagner, 1972), i.e. violation of the harm expectancy. Translated to exposure therapy for PTSD, this means that a PTSD patient learns that confrontation with traumatic stimuli (CS) will not lead to the expected hazardous outcome (No US). Hypothetically, as an alternative to the fear habituation model, violation of the idea that exposure to trauma-related stimuli would be harmful could lead to successful extinction learning and favourable treatment outcome in the end. Although this expectancy violation hypothesis has been affirmed by both theory and experimental work (Craske et al., 2008; Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014), there are no studies that have tested harm expectancy violation as mechanism of

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[1] Note that the process referred to is not actual fear habituation, but rather extinction learning. However, to align with the terminology of EPT we will refer to this process as fear habituation throughout this report.

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change in exposure therapy for PTSD. In experimental fear conditioning paradigms changes in harm (US) expectancies are often used as an indication of successful extinction learning (Boddez et al., 2013), and there is evidence to suggest that PTSD patients are characterized by elevated harm expectancies during experimental extinction learning (Blechert, Michael, Vriends, Margraf, & Wilhelm, 2007). Moreover, this expectancy bias was found to predict the onset (Lommen, Engelhard, Sijbrandij, van den Hout, & Hermans, 2013) and maintenance (Engelhard, de Jong, van den Hout, & van Overveld, 2009) of PTSD symptoms. However, to the best of our knowledge, there is no study investigating expectancy violations during exposure therapy for PTSD. As such, it is still unclear *which* harm expectancies should be targeted and violated during exposure therapy for PTSD in order to optimize learning. There is ample evidence that cognitive changes occur during exposure therapy, even without explicitly addressing dysfunctional cognitions (Foa et al., 2005; Hagenaars, van Minnen, & de Rooij, 2010; McLean, Yeh, Rosenfield, & Foa, 2015; Zalta et al., 2014), and that these cognitive changes precede PTSD symptom decline (McLean et al., 2015; Zalta et al., 2014). However, in these studies general dysfunctional cognitions were studied and not so much expectancy violations. Investigating changes in the CS-US relationship could provide us with a better understanding of the mechanisms of change during exposure therapy.

The aims of this study are: 1) to gain more insight into harm expectancies during exposure therapy for PTSD; 2) to examine whether harm expectancies are violated during exposure therapy; 3) to examine whether harm expectancy violation is related to exposure therapy outcome; and 4) to explore the relationship between expectancy violation and fear habituation during exposure therapy.

2. Materials and methods

2.1. Participants

Participants were 50 treatment-seeking patients (44 women, mean age = 37 year) with a primary diagnosis of DSM-IV defined chronic PTSD (as established by the MINI; (Sheehan et al., 1998) following interpersonal victimization (see Table 1). All were enrolled in an open exposure therapy trial. Those with acute suicidal risk and inadequate proficiency of the Dutch language were excluded from participation. Written informed consent was obtained from all participants. Prior to enrolment, PTSD diagnoses were verified by means of the Clinician Administered PTSD Scale (Blake et al., 1990), and mean scores indicated severe PTSD symptoms (M = 83.68, SD = 13.83).

2.2. Measures

2.2.1. Clinician administered PTSD scale (Blake et al., 1990)

Outcome was assessed with the Dutch translation of the Clinician-Administered PTSD Scale (CAPS-1; Blake et al., 1995; Hovens et al., 2005 Hovens, Luinge, & van Minnen, 2005), a clinician-rated structured interview developed to test for the presence of the 17 DSM-IV-TR criteria for PTSD.

Table 1
Baseline characteristics of study participants.

	n (%)
Demographics	
Female	44 (88)
Post high school education	37 (74)
Married/Co-habitating	22 (44)
Trauma history	
Childhood (≤16 year)	
Sexual abuse	43 (86)
Physical abuse	33 (66)
Adult	
Sexual assault	25 (50)
Domestic violence/physical assault	27 (54)

2.2.2. Subjective units of distress (SUDs)

During each exposure session subjective units of distress ratings (SUDs; Wolpe, 1958) were obtained. Participants rated their levels of distress on a 0–10 point scale (no anxiety – maximum anxiety). Following previous studies (e.g. Rauch et al., 2004; Rothbaum et al., 2014; van Minnen & Hagenaars, 2002), within session habituation was calculated by subtracting the end of exposure SUD score from the peak SUD score, and between session habituation was calculated as the difference between SUD peak scores from successive sessions. The mean of these differences were used as indices of average within- and between session habituation over treatment.

2.2.3. Harm expectancy and harm experience ratings

To gain more insight into harm expectancies in PTSD patients, at the start of treatment, participants formulated their harm expectancy regarding imaginal exposure by completing an open-ended sentence: “Doing imaginal exposure, I fear….”. In addition, to assess changes in harm expectancy ratings over treatment participants rated their belief in three commonly expressed harm expectancies regarding (imaginal) exposure (Craske et al., 2014; Foa & Kozak, 1986; Foa & Rothbaum, 1998) prior to each exposure session on a 0 (totally disagree) to 10-point (completely agree) scale. These expectancies were: During imaginal exposure I will get so anxious, that I will: 1) go crazy; 2) lose control; 3) panic. Immediately after each session, participants rated their harm experience. That is, they rated (on a scale from 0 (not at all) to 10 (completely)) whether they actually experienced their feared outcome, i.e. had the feeling they went crazy, lost control, or panicked. Internal consistency of both the harm expectancy and experience questionnaire was deemed satisfactory ($\alpha = 0.94$ and $\alpha = 0.90$, respectively), hence we used mean scores of both measures in all analyses. In analogue to the fear habituation measures, we calculated within session expectancy violation by subtracting harm experience from harm expectancy ratings and between session expectancy change by subtracting harm expectancy scores from successive sessions. The mean of these differences were used as indices of average expectancy violation and change over treatment.

2.3. Procedure

Prior to treatment all participants completed a baseline assessment, comprising clinician administered and self-report instruments. Treatment consisted of brief intensive exposure therapy (Hendriks, de Kleine, van Rees, Bult, & van Minnen, 2010), which is largely based on the Prolonged Exposure (PE) protocol (Foa, Hembree, & Rothbaum, 2007). The intensive phase comprised 12 exposure-based sessions provided on four consecutive treatment days. Each day’s first session consisted of manualized 60 min imaginal exposure following the PE protocol. Patients were instructed to close their eyes and recount the traumatic memories aloud. Following these imaginal exposure sessions, patients engaged each day in two more exposure sessions, that included imaginal exposure but also additional exposure-based treatment components (such as in-vivo exposure). To align with previous studies in this field (Bluett et al., 2014; Rauch et al., 2004; van Minnen & Foa, 2006 ; van Minnen & Hagenaars, 2002) and limit variance due to different treatment procedures, we only assessed expectancy violation and fear habituation during the imaginal exposure sessions that followed the PE protocol. The intensive phase was followed by a maintenance phase, wherein participants received up to four weekly exposure-based booster sessions. One week after completion of the total treatment program (six weeks), participants (N = 48, 2 missing) completed the post-treatment assessment.