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Mechanisms of Change in Written Exposure Treatment of Posttraumatic Stress Disorder

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Although the effectiveness of exposure therapy for PTSD is recognized, treatment mechanisms are not well understood. Emotional processing theory (EPT) posits that fear reduction within and between sessions creates new learning, but evidence is limited by self-report assessments and inclusion of treatment components other than exposure. We examined trajectories of physiological arousal and their relation to PTSD treatment outcome in a randomized controlled trial of written exposure treatment, a protocol focused on exposure to trauma memories. Hierarchical linear modeling was used to model reduction in Clinician Administered PTSD Scale score as a predictor of initial activation and within- and between-session change in physiological arousal. Treatment gains were significantly associated with initial physiological activation, but not with within- or between-session changes in physiological arousal. Treatment gains were associated with larger between-session reductions in self-reported arousal. These findings highlight the importance of multimethod arousal assessment and add to a growing literature suggesting refinements of EPT.

Keywords: exposure; emotional processing theory; PTSD; psychophysiology; written exposure treatment

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There is clear evidence that exposure treatment for posttraumatic stress disorder (PTSD) is effective (Institute of Medicine, 2008). What is not well understood is the mechanism of change in exposure treatment for PTSD. The most commonly cited theory for why exposure works is emotional processing theory (EPT; Foa, Huppert, & Cahill, 2006; Foa & Kozak, 1986), which combines learning and cognitive theories. In EPT, Foa and Kozak (1986) state that cognitive changes mediate fear reductions observed during exposure. This theory draws from the bioinformational theory of emotion (Lang, 1979), in which pathological fear is construed as a cognitive structure that includes erroneous information about stimuli, responses, and their meanings. Foa and Kozak (1986) proposed that exposure techniques work by activating the fear structure through exposure to feared stimuli and providing corrective information about the stimuli, responses, and their meanings. Thus, emotional processing has occurred when the fear structure has been activated (high initial arousal) and there is a decrease of arousal both within the exposure session (within-session change [WSC]) and between exposure sessions (between-session change [BSC]).[1]

[1] It should be noted that the terms within- and between-session habituation are frequently used. Because this process is more accurately described as extinction of fear responding through learning, rather than habituation, we use the term "change" rather than "habituation."

Although EPT is frequently cited to account for PTSD treatment response, inconsistent findings have been reported (for a review, see Craske et al., 2008). In the literature examining PTSD treatment, initial fear activation (IFA) has been associated with successful PTSD treatment outcome in some studies (e.g., Foa, Riggs, Massie, & Yarczower, 1995; Pitman, Orr, Altman, & Longpre, 1996a; van Minnen & Hagenaars, 2002). BSC has also been positively related to PTSD treatment outcome in a number of studies (e.g., Bluett, Zoellner, & Feeny, 2014; Jaycox, Foa, & Morral, 1998; Rauch, Foa, Furr, & Filip, 2004; Sripada & Rauch, 2015), but not in other studies (e.g., Pitman et al., 1996a, 1996b). Notably, most studies have not found WSC to be positively related to PTSD treatment outcome (e.g., Foa et al., 2006; Jaycox et al., 1998; Pitman et al., 1996a, 1996b; Sripada & Rauch, 2015; van Minnen & Hagenaars, 2002).

The PTSD treatment mechanisms literature, however, is limited by methodological aspects of the studies conducted to date. First, although EPT explicitly predicts change in self-reported and physiological arousal (Foa & Kozak, 1986; Foa et al., 2006), most PTSD studies have relied solely on self-report (e.g., Bluett et al., 2014; Jaycox et al., 1998; Rauch et al., 2004; van Minnen & Hagenaars, 2002). Emotion theorists generally view subjective experience and physiological reactions as two separate, but related, components of an emotion (e.g., Lang, 1979). Self-reported distress and physiological arousal often correspond (e.g., Marx et al., 2012), but they do not always co-occur (fear discordance), nor do they necessarily change together (fear desynchrony; Hodgson & Rachman, 1974). Consequently, physiological assessment offers an objective measure of physiological arousal distinct from subjective, self-reported emotional experience. Of note, only two studies with small samples have incorporated physiological measures to investigate PTSD treatment (Pitman et al., 1996a, 1996b). These studies found limited evidence that treatment outcome was associated with IFA, and no evidence that it was associated with WSC or BSC.

Within the PTSD treatment literature, another important consideration is that all but two studies have examined EPT in prolonged exposure (PE) treatment (Bluett et al., 2014; Jaycox et al., 1998; Rauch et al., 2004; van Minnen & Hagenaars, 2002; but see also Craske et al., 2008). The two studies that used other therapies (imaginal flooding and eye movement desensitization and reprocessing) did not find the BSC effect (Pitman et al., 1996a, 1996b), raising the possibility that BSC only predicts treatment outcome in the context of PE. As PE includes multiple components (i.e., psychoeducation, imaginal exposure to trauma memories, *in vivo* exposure, and relaxation), and between-session assignments, it is unclear whether the reported BSC is the result of exposure to trauma memories or some other treatment component.

The goal of the present study was to investigate IFA, WSC, and BSC of physiological arousal in exposure treatment for PTSD. This study draws from a randomized controlled trial reported elsewhere (Sloan, Marx, Bovin, Feinstein, & Gallagher, 2012). The current study has unique aspects that lend well to the investigation of EPT accounting for PTSD treatment outcome. First, the treatment consisted of a written form of trauma memory exposure that took place over five sessions with no between-session assignments. Therefore, we can more confidently attribute fear reduction patterns to trauma memory exposure rather than other intervention components. Second, physiological reactivity was measured. Third, this study used hierarchical linear modeling rather than the more traditional difference score approach (Bluett et al., 2014; Pitman et al., 1996a, 1996b, Rauch et al., 2004; but see also Sripada & Rauch, 2015), allowing for a more sensitive test of changes in arousal. Based on EPT, we predicted that PTSD treatment outcome would be positively associated with IFA and BSC. Given prior findings demonstrating no effect of WSC, we predicted that PTSD treatment outcome would not be associated with WSC.

Method

PARTICIPANTS

Inclusion criteria were age of 18 or older and a primary diagnosis of PTSD related to a motor vehicle accident. Exclusion criteria were current psychotic diagnosis, organic mental disorder, current substance dependence, unstable bipolar disorder, English illiteracy, and high risk for suicidal behavior. Forty-six individuals satisfied inclusion/exclusion criteria and were randomized to either a brief, exposure-based treatment condition (n = 22) or a waitlist condition (n = 24; for details on participant recruitment and screening and CONSORT flowchart, see Sloan et al., 2012). Given the goal of this study, only the 22 participants assigned to treatment are presented.

Participants randomized to the treatment condition had an average age of 39.45 (SD = 14.84), 16 (73%) were women, and racial background was diverse (40.9% White, 27.4% African-American, 13.5% Hispanic, 18.2% "other"). Participants reported exposure to multiple traumas (median = 11.09). Two individuals (9.1%) dropped out of treatment. All available data were used for all participants, including the two who dropped out.

TREATMENT

Treatment was provided by three master's- or doctoral-level clinicians with prior PTSD treatment

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