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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]

Although EPT is frequently cited to account for P-

[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."

TSD 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 experience. The treatment consisted of five weekly

sessions in which participants were instructed to write about their index trauma (i.e., motor vehicle accident) with as much emotion and detail as possible. The first session lasted approximately 1 hour and consisted of psychoeducation about PTSD, a treatment rationale, and written exposure. Avoidance of trauma reminders was emphasized as a PTSD maintenance factor, and the rationale for confronting trauma memories through exposure was presented. The therapist then read the session instructions to the participant, and the printed instructions were left with participants while they wrote about the trauma for 30 minutes. The therapist then checked in with participants about the writing, and encouraged participants to allow themselves to have whatever trauma-related thoughts, feelings, or images came to mind during the upcoming week. Aside from this general instruction, no assignments were given. The remaining four sessions consisted of 30 minutes of writing about the traumatic event, followed by a brief check-in with the therapist. Instructions for each writing session varied slightly (for details of the treatment protocol, see Sloan et al., 2012).

MEASURES

The Clinician-Administered PTSD Scale (CAPS; Weathers, Keane, & Davidson, 2001) was used to establish PTSD diagnosis related to the index trauma and to measure PTSD symptom severity. The CAPS consists of ratings of the frequency and intensity of the 17 PTSD symptoms defined by the *DSM-IV* (American Psychiatric Association, 1994). Individuals who met *DSM-IV* symptom criteria and had a total CAPS score ≥ 40 received a PTSD diagnosis (Weathers et al., 2001). The CAPS was administered at pretreatment and posttreatment by master's- or doctoral-level clinicians who were unaware of treatment randomization. The CAPS has been shown to have strong convergent and discriminant validity in prior research (Weathers et al., 2001), and interrater agreement in this study was excellent ($\kappa = .94$; Sloan et al., 2012).

Self-Reported Emotion

The Self-Assessment Manikin (SAM; Bradley & Lang, 1994) measured participants' subjective arousal immediately following each writing session (participants were instructed to report how they were feeling at that moment). The arousal scale is rated on a 9-point scale, with higher ratings indicating greater arousal. The SAM was selected for its theoretical correspondence to physiological arousal and its documented psychometric properties, including strong reliability and convergent validity with semantic emotion descriptors (Bradley & Lang, 1994; Lang, Bradley, & Cuthbert, 1997).

Physiological Reactivity

Cardiac activity was used to examine physiological arousal. It was selected because cardiovascular changes are common and detectable measures of changes in one's arousal that are easily measured within therapy sessions (e.g., Berntson, Quigley, & Lozano, 2007; Sloan & Kring, 2007). In addition, other investigators have used cardiac activity to index emotional engagement during fear processing (e.g., Pitman et al., 1996a, 1996b), and a comprehensive meta-analysis found that heart rate was more strongly related to PTSD than other physiological measures, such as skin conductance (Pole, 2007).

Cardiac activity was recorded continuously, for a 5-minute baseline period and during the treatment session. Participants were seated in quiet treatment rooms at a comfortable temperature while their cardiac activity was monitored by a Polar S810 HR monitor, an ambulatory system that consists of a wristwatch receiver (display hidden from participants in this study) and the T61 transmitter chest strap, applied with a water-soluble transmitting gel to facilitate conduction. The wristwatch receiver calculated the interbeat intervals (IBIs), which reflect the number of milliseconds between heart beats. IBI is inversely related to heart rate measured in beats per minute (IBI = 60000/heart rate; Berntson et al., 2007). Raw IBI data were visually inspected for outliers, which were manually corrected. IBI data were missing from 10 treatment sessions (9.1% of session data) due to equipment malfunction; all available IBI data were used for all participants.

Data obtained using the Polar monitor system is highly correlated with recordings from electrocardiogram (e.g., Goodie, Larkin, & Schauss, 2000). The Polar S810 watch has been used extensively in psychophysiological research, including PTSD studies (e.g., Hauschildt, Peters, Moritz, & Jelinek, 2011). In order to obtain relatively reliable estimates of peak IBI within each treatment session, we averaged raw IBI measurements across 40-beat segments. We chose cardiac time (a set number of beats) rather than real time (a set number of seconds) based on recommendations for unbiased estimates of mean IBI (Berntson, Cacioppo, & Quigley, 1995; Graham, 1978). The segment with the lowest IBI (corresponding to the fastest heart rate) within each session was classified as the "peak heart period."

DATA ANALYTIC PLAN

We used hierarchical linear modeling (HLM), which is ideal for nested data structures; HLM 7.01 software was used (Raudenbush & Bryk, 2002). Because SAM ratings were obtained once per session, we could analyze BSC but not IFA or WSC in self-reported arousal. Therefore, we planned tests of BSC in self-reported arousal, and tests of all three indices using physiological response. To test BSC of self-reported arousal, we created a two-level HLM model of observations nested within participants. We included session number (coded as 0 to 4) as a level 1 predictor and CAPS change (posttreatment CAPS – pretreatment CAPS) as a level 2 (individual-level) predictor. The coefficient of interest corresponded to CAPS change as a predictor of changes in self-reported arousal across sessions, which reflects an association between BSC and treatment response (see Equation S1 in Supplemental Online Material [SOM]).

Our outcome variable for all physiological analyses was IBI. To test IFA, we examined data from the first exposure session only, consistent with prior approaches (Bluett, et al., 2014; Jaycox et al., 1998; van Minnen & Hagenaars, 2002). Time was coded as time from the beginning of baseline (time = 0) to the time of the peak IBI (time = X, with X corresponding to the number of IBIs from baseline to peak IBI) during the first session. We entered time to peak IBI as a level 1 predictor and CAPS change as a level 2 (individual-level) predictor. The coefficient of interest corresponded to CAPS change as a predictor of changes in IBI (see Equation S2 in SOM). Different IBI data (IBIs from the peak IBI to the end of each session) were used for our tests of WSC and BSC. To test WSC and BSC, we included all available session data (sessions 1–5) and created a three-level model examining predictors of IBI, with time (level 1) nested within sessions (level 2) nested within individuals (level 3). Time within session was coded as the time from peak IBI (time = 0) to the end of the session (time = Y, with Y corresponding to the number of IBIs from peak IBI to the end of that session). For WSC, we entered time within session as a level 1 predictor and CAPS change as a level 3 (individual-level) predictor. The coefficient of interest corresponded to CAPS change as a predictor of changes in IBI within a session (see Equation S3 in SOM). For BSC, we entered the same level 1 and 3 predictors, and added session number (coded as 0 to 4) as a level 2 (session-level) predictor. The coefficient of interest corresponded to CAPS change as a predictor of change in peak IBI across sessions (see Equation S4 in SOM). All effects were calculated with robust standard errors, all intercepts and slopes were modeled as random coefficients, and partial correlation coefficients (*pr*) were calculated to provide effect sizes.[2]

Results

The results of the randomized controlled trial are provided elsewhere and will not be discussed in detail here (Sloan et al., 2012). Briefly, participants randomly assigned to WET showed significantly larger treatment gains relative to participants assigned to the waitlist. At posttreatment, 5% of WET participants met diagnostic criteria for PTSD according to the CAPS, compared with 88% of the waitlist participants. Between-condition effect sizes were calculated with Hedges' unbiased *g*, and the effect size was $g = 3.49$ posttreatment. Among WET participants, CAPS score changed from a pretreatment mean of 63 to a posttreatment mean of 19, or a mean change of 42 points ($SD = 16.8$), with a standardized mean gain (an effect size measure) of 3.18.

BSC IN SELF-REPORTED AROUSAL

We first ran the HLM model including only session number as a level 1 predictor, to examine whether self-reported arousal changed significantly across treatment sessions. This analysis indicated that participants in general showed a significant decrease in self-reported arousal across sessions, $B = -0.6$, $t(20) = 5.0$, $p < .001$, $pr = .75$ (see Table 1). This finding indicates that participants on average showed BSC in self-reported arousal. We then added CAPS change as a level 2 (individual-level) predictor to examine the association between treatment response and BSC. There was a significant effect, such that participants with greater CAPS change also showed greater decreases in self-reported arousal, $B = -0.01$, $t(19) = 3.0$, $p = .007$, $pr = .57$ (see Figure 1).

INITIAL ACTIVATION AND CHANGE OF PHYSIOLOGICAL RESPONSE

IFA

We first ran an HLM model with time entered as the sole predictor, to examine whether IBI changed significantly within the first exposure session (from baseline to peak IBI). The analysis indicated that there was, on average, a significant decrease in IBI (reflecting an increase in heart rate), $B = -6.80$, $t(21) = -2.20$, $p = .04$, $pr = .19$. This finding indicates that participants generally experienced initial activation of a physiological fear response during the first exposure session. We then added CAPS change as an individual-level predictor to test the association between treatment response and IFA. CAPS change

[2] Partial correlation coefficients (*pr*) are interpreted the same way as correlation coefficients. Conventionally, an *r* of .10 would be considered small, .30 medium, and .50 large (Cohen, 1988).

Table 1
Mean Changes in Self-Reported Arousal and Peak Heart Rate Across Exposure Sessions

	Session				
	1	2	3	4	5
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Self-reported Arousal	5.95 (1.70)	4.73 (1.67)	4.19 (1.60)	3.95 (1.64)	3.20 (1.80)
Peak Heart Rate (bpm)	90.51 (23.17)	94.45 (17.97)	89.81 (15.76)	94.36 (29.67)	85.06 (10.48)

Note. SD = Standard deviation, bpm = beats per minute. Self-reported arousal was measured by the Self-Assessment Manikin, with possible scores ranging from 1 = very calm to 9 = very aroused. Interbeat interval (IBI), measured in milliseconds, was used for all physiological analyses. For ease of interpretability, IBI was converted to heart rate, measured in beats per minute, for this table (heart rate = 60000/IBI). Average resting heart rate for American adults is approximately 60–80 beats per minute. Heart rate data reflect means for all participants with data available for that session.

was a significant predictor of IFA, B = -0.31, $t(20)$ = -2.58, p = .02, pr = .17, such that individuals who showed greater increases in physiological arousal in the first exposure session also showed the largest treatment response.[3]

WSC

For our models of WSC and BSC, time reflects time from peak IBI to the end of each exposure session. We first ran an HLM model with time entered as the sole predictor, to examine whether IBI changed significantly from peak IBI to the end of the session. This analysis indicated that there was, on average, a significant increase in IBI (reflecting a decrease in heart rate), B = 2.10, $t(21)$ = 5.87, $p < .001$, pr = .62. This finding indicates that participants generally experienced WSC in physiological response. We then added CAPS change as an individual level predictor to test the association between treatment response and WSC. CAPS change did not significantly predict WSC, B = 0.01, $t(20)$ = 0.49, p = .63 pr = .01.

BSC

We examined whether peak IBI changed significantly across the five treatment sessions by including time within session as a level 1 predictor and session number as a level 2 predictor. Peak IBI did not change significantly across sessions among participants on average, B = -2.08, $t(21)$ = -0.20, p = 0.85, $pr < .01$. This finding indicates that participants on average did not experience BSC in physiological response. We then entered CAPS change as an individual-level predictor to test the association between treatment response and BSC. CAPS change was not a significant predictor of change in peak IBI across sessions, B = -0.07, $t(20)$ = -0.22, p = .83, $pr < .01$.[4]

[3] To examine whether the IFA effect is specific to the first exposure session, we ran four additional HLM models examining fear activation (change in IBI from the beginning of the baseline to the time of the peak IBI) in Sessions 2–5. We included CAPS change as a level 2 predictor of change in IBI from baseline to peak IBI within each session. Fear activation in Sessions 2–5 was not associated with treatment outcome, $ts < 1.5$, *ns*.

Discussion

We tested four indices of EPT and their relation to PTSD treatment response. Two indices, BSC in self-reported arousal and initial activation of physiological arousal, were positively associated with treatment gains. Neither WSC nor BSC in physiological arousal was related to treatment response.

Our self-report findings are consistent with prior research demonstrating that BSC in self-reported arousal predicts treatment gains among individuals who receive PE. To our knowledge, our results are the first to provide support for an association between BSC in self-reported arousal and changes in PTSD severity in a treatment package other than PE. Unlike PE, which includes several treatment components, the central feature of the treatment in this study is a written form of trauma memory exposure with minimal therapist instruction. Our findings therefore increase confidence that BSC of self-reported arousal can be attributed to trauma memory exposure specifically. These findings also indicate that self-reports of arousal offer potential clinical utility as a means of tracking treatment response.

We also found that initial activation of the physiological fear response occurred during the first exposure session and was associated with treatment gains. This finding is consistent with prior research indicating that IFA is associated with positive response to PTSD treatment (Foa et al., 1995; Pitman et al., 1996b; van Minnen & Hagenaars, 2002). This study extends prior work by demonstrating that physiological fear activation can be achieved in the

[4] We ran additional WSC and BSC analyses including baseline IBI as a covariate; these analyses yielded the same results. Because our BSC model simultaneously models WSC and BSC, we also ran a simplified BSC model that isolates BSC. The simplified two-level BSC model had peak IBI as the outcome variable, session number as a level 1 predictor, and CAPS change as a level 2 predictor; this model yielded the same results as the full BSC model. All IFA, WSC, and BSC results were also the same when controlling for factors which might affect cardiac reactivity (smoker status, caffeine use, and medication use).

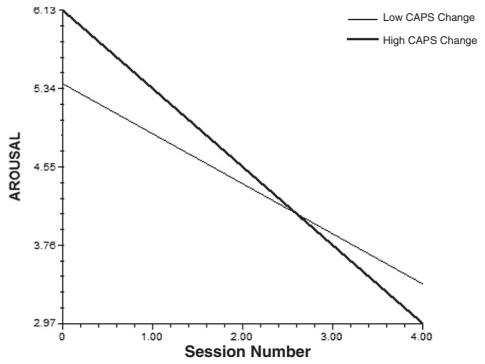

FIGURE 1 Association between Treatment Response and Between-Session Change in Self-Reported Arousal. Note. CAPS = Clinician-Administered PTSD Scale. CAPS change was analyzed as a continuous variable, but is split into low and high groups here for ease of graphical presentation. Low and high CAPS change scores were defined as one standard deviation below and above the mean, respectively. Sessions were coded as 0 to 4 for the purposes of the HLM analyses; session 0 was the first treatment session.

written exposure treatment protocol, even though this protocol includes a relatively brief (30 minutes) and largely self-guided written exposure to trauma memories. Moreover, this finding suggests that initial activation of the physiological fear response could be an important predictor of treatment outcome in exposure-based treatments. The inclusion of heart rate recording could provide clients and therapists with additional information to facilitate successful treatment outcome. For example, if a client shows little change in heart rate in the first exposure session, this could be an "early warning sign" for the therapist that the exposure is not being implemented as effectively as possible. The therapist could then provide feedback to the client that he or she is not demonstrating a physiological response, and provide suggestions to elicit a stronger response in the next exposure session (e.g., provide more sensory detail). The ambulatory heart rate monitor used in the current study can be easily implemented in a treatment session as it is simple and affordable.

Importantly, we found no evidence that WSC in physiological arousal was associated with treatment outcome. On average, participants showed significant decreases in physiological arousal within sessions, indicating that they did experience WSC. However, the amount of WSC was not related to treatment gains, a finding consistent with prior research (e.g., Jaycox et al., 1998; Pitman et al., 1996a, 1996b; van Minnen & Hagenaars, 2002). Indeed, in an update of EPT, Foa and colleagues (2006) acknowledged that WSC has been inconsistently related to symptom change, and suggested that WSC may not be necessary for effective treatment outcome. These findings are noteworthy given the importance assigned to WSC in the implementation of many exposure-based treatments for PTSD. Guidelines regarding the duration of exposure sessions (e.g., to remain in an *in vivo* exposure until subjective distress has dropped in half) are based on the original EPT, which assigned a primary role to WSC. There is a need for future research determining minimally sufficient doses of different types of exposure necessary for treatment gains (see Minnen & Foa, 2006, and Nacasch et al., 2015, for examples of such studies).

We also found that BSC in physiological arousal was not associated with treatment outcome. On average, participants showed no BSC in physiological arousal, and the amount of BSC was not significantly associated with PTSD symptom change. These findings are inconsistent with prior research examining BSC with self-report measures in prolonged exposure therapy (Bluett et al., 2014; Jaycox et al., 1998; Rauch et al., 2004). It is possible that the lack of BSC in physiological arousal observed in this study is due to the modality of trauma memory exposure that we used. In prolonged exposure, trauma memory exposure (or imaginal exposure) consists of instructions to close one's eyes and engage in vivid mental imagery of the trauma memory while describing one's memory out loud. In WET, participants are instructed to write a narrative of their trauma memory. Although WET instructs participants to include sensory details in their narratives, the act of writing may elicit more verbal-linguistic processing and less imagery relative to imaginal exposure. Greater reliance on verbal-linguistic processing may interfere with fear extinction processes (e.g., Borkovec, Alcaine, & Behar, 2004; Borkovec & Hu, 1990). It seems unlikely that the act of writing interfered with fear extinction in this study, however, because WET patients showed large declines in PTSD symptoms, with only 5% still meeting criteria for PTSD at posttreatment (Sloan et al., 2012). The finding is consistent with prior research on exposure for phobias, indicating that there can be large reductions in symptoms in the absence of BSC in heart rate (Lang & Craske, 2000; Rowe & Craske, 1998; Tsao & Craske, 2000). Additionally, we are aware of no evidence that BSC in physiological arousal occurs during prolonged exposure, as all published work on mechanisms of prolonged exposure has relied upon self-report measures of distress (e.g., Bluett et al., 2014; Jaycox et al., 1998; Rauch et al., 2004). There is a need for future research examining physiological arousal during prolonged exposure to examine whether the BSC effect extends to physiological arousal or is limited to subjective arousal.

In fact, our study provides evidence for desynchrony between physiological and self-report measures of BSC in arousal. In the same sample, we

found that BSC occurs and is related to treatment outcome when assessed by self-report, but not when assessed by physiological response. This discrepancy between BSC findings in self-reported and physiological arousal may be due to differences in how BSC was measured in this study. Physiological arousal was measured continuously throughout each treatment session and peak arousal within each session was identified. Self-reported arousal was a general index assessed at the end of each treatment session. Additionally, prior research has indicated that SAM arousal ratings are more strongly associated with skin conductance than with HR, which may also explain the discrepancy in our study (e.g., Greenwald, Cook, & Lang, 1989). Therefore, it is not clear whether the desynchrony between BSC in self-reported and physiological arousal is due to measurement or to a more substantive discrepancy between participants' subjective appraisal of their arousal and their physiological state. Participants' subjective appraisal of their arousal may be influenced not only by their physiological arousal, but also by their perceptions that such sensations are nonthreatening and tolerable (e.g., Hodgson & Rachman, 1974). The experience of physiological arousal in the absence of any negative outcome during exposure sessions (i.e., expectancy violation) may lead to changes in subjective appraisal even in the absence of changes in physiological response. Future research including more comparable measures of BSC in subjective and physiological arousal is indicated to examine this possibility.

It is important to acknowledge this study's limitations. Our sample was relatively small. Small sample sizes are associated with unreliable estimates of population effects and limit statistical power. However, the effect sizes obtained for null findings were very small (*pr*s < .01), suggesting the null findings are unlikely the result of Type II error. Importantly, the small effect sizes also suggest that these effects are unlikely to be clinically meaningful even if statistical significance could be obtained with a larger sample. Another possible explanation for the null physiological results is the use of an ambulatory physiological acquisition system, which could be less sensitive than a stationary system. However, the specific Polar watch system that was used has been well-validated against stationary physiological acquisition systems (Goodie et al., 2000), and some significant effects on physiological arousal were obtained, indicating that the Polar device was sensitive to changes in heart rate. We elected to use this relatively inexpensive and portable system as it could be easily translated into clinical practice. Our sample was also limited to individuals with PTSD related to a motor vehicle accident; these results should be replicated in samples with PTSD related to other trauma types. Another limitation is that we could not examine initial activation or WSC in self-reported arousal because self-reported arousal was only assessed once per session. Although prior findings assessing IFA and WSC with self-report measures are consistent with our physiological results, it would have been useful to compare self-report and physiological assessment of these constructs in the same study. Additionally, we relied upon heart rate as our sole measure of physiological arousal. Heart rate is influenced by both the sympathetic and parasympathetic branches of the autonomic nervous system. Other measures, such as skin conductance, offer purer measures of sympathetic arousal (Boucsein, 1992). Measurement across multiple channels would have provided stronger evidence for the reported patterns of reduction (or lack thereof) in physiological arousal. Strengths of this study include the assessment of physiological arousal, the use of a PTSD treatment, written exposure treatment, that more specifically isolates exposure to trauma memories, and the use of HLM, which allowed us to analyze all session data.

Our findings point to several important areas for future research in the study of exposure therapy for PTSD. A key question is whether our findings will generalize to other exposure-based treatments for PTSD, most notably prolonged exposure. Because all published research on prolonged exposure has relied upon self-report measures of distress, it is unclear whether the observed patterns of IFA, WSC, and BSC in physiological arousal are unique to WET or will generalize to prolonged exposure. Future clinical trials of prolonged exposure could include measures of heart rate and skin conductance within each session to examine whether similar patterns emerge. Additionally, it would be informative to conduct experimental research comparing written and imaginal forms of exposure directly across multiple channels of physiological response. We found that written exposure was successful in eliciting fear in this study, but it would be helpful to know whether the magnitude of fear activation is similar across different exposure modalities. Given prior research on verbal-linguistic versus imagery-based thought (Vrana, Cuthbert, & Lang, 1986), we might expect imaginal exposure to elicit even larger fear activation than written exposure, with potentially important implications for treatment outcome. In such research, it would be important to measure the extent of verbal-linguistic versus imagery-based processing in written versus imaginal exposure, to examine the assumption that writing encourages more verbal-linguistic thought. Finally, future research could examine whether physiological indicators might be developed

into a tool for treatment matching. Currently, prolonged exposure and cognitive processing therapy are both recognized as empirically supported treatments for PTSD (APA Presidential Task Force on Evidence-Based Practice, 2006), yet little is known about which treatment works best for which patient. Perhaps patients who show large physiological fear responses to trauma memories in a pretreatment laboratory session would fare better in prolonged exposure, which explicitly targets fear extinction.

In conclusion, these findings add to a growing literature calling for refinements in our understanding of PTSD treatment mechanisms (Bluett et al., 2014; Craske et al., 2008). First, our finding that IFA of physiological arousal predicts treatment response suggests that cardiac monitoring could be a useful tool for clinicians as an objective measurement of engagement with exposure. Second, our finding that WSC of physiological arousal was not related to treatment outcome adds to the evidence that WSC does not predict PTSD treatment gains (Foa et al., 2006; Jaycox et al., 1998; Pitman et al., 1996a, 1996b; van Minnen & Hagenaars, 2002). Third, the finding that BSC in physiological arousal does not predict PTSD treatment outcome contradicts the original EPT, which explicitly included both self-reported and physiological arousal (Foa & Kozak, 1986). These findings also support interest in developing new theories of exposure, which emphasize mechanisms other than fear reduction (e.g., Bouton, 2004; Craske et al., 2008; Foa et al., 2006). Such models argue that the key mechanism of exposure treatment is inhibitory learning, or the learning of new associations between trauma cues and lack of threat. Because WSC and BSC are poor markers of new learning, they are unlikely to be strongly associated with treatment outcome (Craske et al., 2008). Therefore, there is a need to develop measures of inhibitory learning, which may serve as more important predictors of treatment outcome than fear responding. A better understanding of the mechanisms through which exposure therapy reduces PTSD will help us continue to refine exposure treatment to achieve even larger and more durable effects.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.beth.2015.09.005.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

APA Presidential Task Force on Evidence-Based Practice. (2006). Evidence-based practice in psychology. *American Psychologist*, *61*, 271–285. http://dx.doi.org/10.1037/0003-066X.61.4.271

Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1995). The metrics of cardiac chronotropism: Biometric perspectives. *Psychophysiology*, *32*, 162–171.

Berntson, G. G., Quigley, K. S., & Lozano, D. (2007). Cardiovascular psychophysiology. In J. T. Cacioppo, C. G. Tassinary, G.G. Berntson (Eds.), *Handbook of Psychophysiology, 3rd ed.* (pp. 182–210). New York, NY: Cambridge University Press.

Bluett, E. J., Zoellner, L. A., & Feeny, N. C. (2014). Does change in distress matter? Mechanisms of change in prolonged exposure for PTSD. *Journal of Behavior Therapy and Experimental Psychiatry*, *45*, 97–104. http://dx.doi.org/10.1016/j.jbtep.2013.09.003

Borkovec, T. D., & Hu, S. (1990). The effect of worry on cardiovascular response to phobic imagery. *Behaviour Research and Therapy*, *28*, 69–73. http://dx.doi.org/10.1016/0005-7967(90)90056-O

Borkovec, T. D., Alcaine, O., & Behar, E. (2004). Avoidance theory of worry and generalized anxiety disorder. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice*. New York, NY: Guilford Press.

Boucsein, W. (1992). *Electrodermal activity*. Berlin: Springer.

Bouton, M. E. (2004). Context and behavioral processes in extinction. *Learning & Memory*, *11*, 485e494. http://dx.doi.org/10.1101/lm.78804.

Bradley, M. M., & Lang, P. J. (1994). Measuring emotion: The Self-Assessment Manikin and the semantic differential. *Journal of Behavior Therapy and Experimental Psychiatry*, *25*(1), 49–59. http://dx.doi.org/10.1016/0005-7916(94)90063-9

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy*, *46*(1), 5–27. http://dx.doi.org/10.1016/j.brat.2007.10.003

Foa, E. B., Huppert, J. D., & Cahill, S. P. (2006). Emotional processing theory: An update. In B. O. Rothbaum (Ed.), *Pathological anxiety: Emotional processing in etiology and treatment*. (pp. 3–24). New York, NY: Guilford Press.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, *99*, 20–35. http://dx.doi.org/10.1037/0033-2909.99.1.20

Foa, E. B., Riggs, D. S., Massie, E. D., & Yarczower, M. (1995). The impact of fear activation and anger on the efficacy of exposure treatment for posttraumatic stress disorder. *Behavior Therapy*, *26*, 487–499. http://dx.doi.org/10.1016/S0005-7894(05)80096-6

Goodie, J. L., Larkin, K. T., & Schauss, S. (2000). Validation of the polar heart rate monitor for assessing heart rate during

physical and mental stress. *Journal of Psychophysiology*, *14*, 159–164. http://dx.doi.org/10.1027//0269-8803.14.3.159

Graham, F. K. (1978). Constraints on measuring heart rate and period sequentially through real and cardiac time. *Psychophysiology*, *15*, 492–495.

Greenwald, M. K., Cook, E. W., & Lang, P. J. (1989). Affective judgment and psychophysiological response: Dimensional covariation in the evaluation of pictorial stimuli. *Journal of Psychophysiology*, *3*, 51–64.

Hauschildt, M., Peters, M. J., Moritz, S., & Jelinek, L. (2011). Heart rate variability in response to affective scenes in posttraumatic stress disorder. *Biological Psychology*, *88*(2), 215–222.

Hodgson, R., & Rachman, S. (1974). II. Desynchrony in measures of fear. *Behaviour Research and Therapy*, *12*(4), 319–326.

Institute of Medicine. (2008). *Treatment of posttraumatic stress disorder: An assessment of the evidence.* Washington, DC: National Academies Press.

Jaycox, L. H., Foa, E. B., & Morral, A. R. (1998). Influence of emotional engagement and habituation on exposure therapy for PTSD. *Journal of Consulting and Clinical Psychology*, *66*(1), 185–192. http://dx.doi.org/10.1037/0022-006X.66.1.185

Lang, A. J., & Craske, M. G. (2000). Manipulations of exposure-based therapy to reduce return of fear: A replication. *Behaviour Research & Therapy*, *38*, 1–12. http://dx.doi.org/10.1016/S0005-7967(99)00031-5

Lang, P. J. (1979). A bio-informational theory of emotional imagery. *Psychophysiology*, *16*, 495–512.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). International affective picture system (IAPS): Technical manual and affective ratings. *NIMH Center for the Study of Emotion and Attention*, 39–58.

Marx, B. P., Monson, C. M., Bovin, M. J., Fredman, S. J., Sloan, D. M., Humphreys, K., Suvak, M., Kaloupek, D. G., & Keane, T. M. (2012). Concordance between physiological arousal and self-reported distress among Veterans with PTSD. *Journal of Traumatic Stress*, *25*, 416–425. http://dx.doi.org/10.1002/jts.21729

Minnen, A. V., & Foa, E. B. (2006). The effect of imaginal exposure length on outcome of treatment for PTSD. *Journal of Traumatic Stress*, *19*(4), 427–438.

Nacasch, N., Huppert, J. D., Su, Y. J., Kivity, Y., Dinshtein, Y., Yeh, R., & Foa, E. B. (2015). Are 60-Minute Prolonged Exposure sessions with 20-Minute imaginal exposure to traumatic memories sufficient to successfully treat PTSD? A randomized noninferiority clinical trial. *Behavior Therapy*, *46*, 328–341. http://dx.doi.org/10.1016/j.beth.2014.12.002

Pitman, R. K., Orr, S. P., Altman, B., & Longpre, R. E. (1996a). Emotional processing and outcome of imaginal flooding therapy in Vietnam Veterans with chronic post-traumatic stress disorder. *Comprehensive Psychiatry*, *37*(6), 409–418. http://dx.doi.org/10.1016/S0010-440X(96)90024-3

Pitman, R. K., Orr, S. P., Altman, B., & Longpre, R. E. (1996b). Emotional processing during eye movement desensitization and reprocessing therapy of Vietnam Veterans with chronic post-traumatic stress disorder. *Comprehensive Psychiatry*, *37*(6), 419–429. http://dx.doi.org/10.1016/S0010-440X(96)90025-5

Pole, N. (2007). The psychophysiology of posttraumatic stress disorder: A meta-analysis. *Psychological Bulletin*, *133*(5), 725. http://dx.doi.org/10.1037/0033-2909.133.5.725

Rauch, S., Foa, E., Furr, J., & Filip, J. (2004). Imagery vividness and perceived anxious arousal in prolonged exposure treatment for PTSD. *Journal of Traumatic Stress*, *17*(6), 461–465. http://dx.doi.org/10.1007/s10960-004-5794-8

Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical Linear Models: Applications and data analysis methods* (2nd ed.). Thousand Oaks, CA: Sage.

Rowe, M. K., & Craske, M. G. (1998). Effects of varied-stimulus exposure training on fear reduction and return of fear. *Behaviour Research and Therapy*, *36*, 719–734. http://dx.doi.org/10.1016/S0005-7967(97)10017-1

Sloan, D. M., & Kring, A. M. (2007). Measuring changes in emotion during psychotherapy: Conceptual and methodological issues. *Clinical Psychology: Science and Practice*, *14*, 307–322. http://dx.doi.org/10.1111/j.1468-2850.2007.00092.x

Sloan, D. M., Marx, B. P., Bovin, M. J., Feinstein, B. A., & Gallagher, M. W. (2012). Written exposure as an intervention for PTSD: A randomized clinical trial with motor vehicle accident survivors. *Behaviour Research and Therapy*, *50*(10), 627–635. http://dx.doi.org/10.1016/j.brat.2012.07.001

Sripada, R. K., & Rauch, S. A. (2015). Between-session and within-session habituation in prolonged exposure therapy for posttraumatic stress disorder: A hierarchical linear modeling approach. *Journal of Anxiety Disorders*, *30*, 81–87. http://dx.doi.org/10.1016/j.janxdis.2015.01.002

Tsao, J. C. I., & Craske, M. G. (2000). Timing of treatment and return of fear: Effects of massed, uniform-, and expanding-spaced exposure schedules. *Behavior Therapy*, *31*, 479–497. http://dx.doi.org/10.1016/S0005-7894(00)80026-X

van Minnen, A., & Hagenaars, M. (2002). Fear activation and habituation patterns as early process predictors of response to Prolonged Exposure treatment in PTSD. *Journal of Traumatic Stress*, *15*(5), 359. http://dx.doi.org/10.1023/A:1020177023209

Vrana, S. R., Cuthbert, B. N., & Lang, P. J. (1986). Fear imagery and text processing. *Psychophysiology*, *23*(3), 247–253. http://dx.doi.org/10.1111/j.1469-8986.1986.tb00626.x

Weathers, F., Keane, T. M., & Davidson, J. (2001). Clinician-Administered PTSD Scale: A review of the first ten years of research. *Depression and Anxiety*, *13*, 132–156. http://dx.doi.org/10.1002/da.1029

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