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Physiological Predictors of Posttraumatic Stress Disorder

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Studies have assessed relationships between posttraumatic stress disorder (PTSD) symptoms and physiological reactivity concurrently; fewer have assessed these relationships longitudinally. This study tests concurrent and prospective relationships between physiological reactivity (heart rate and skin conductance) to a monologue procedure and PTSD symptoms in female assault survivors, tested within 1 and 3 months posttrauma. After controlling for initial PTSD and peritraumatic dissociation, 3 measures of increased physiological reactivity to the trauma monologue at 1 month predicted 3-month PTSD reexperiencing severity. Additionally, increased heart rate following trauma and neutral monologues at 1 month was predictive of 3-month numbing symptoms. Implications for the prospective relationship between physiological reactivity to trauma cues and PTSD over time are discussed.

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Increased physiological reactivity to trauma reminders is a hallmark symptom of posttraumatic stress disorder (PTSD). This has been demonstrated through self-report and psychophysiological measures (Pole, 2007). The vast majority of this prior work demonstrates the concurrent association between elevated physiological arousal and PTSD symptoms. Most studies have not addressed the prospective role of marked arousal to trauma reminders in future PTSD symptoms. This is notable because higher arousal could be a valuable early marker of the disorder. More research is needed to examine the relationship between early psychophysiological arousal and PTSD symptoms over time.

Several studies investigating physiological reactivity in PTSD have relied heavily on Lang's (1979) laboratory paradigm. This paradigm is premised on the idea that vivid recollection of emotional events depends on central processing of an associated information network, and studies have shown that emotionally charged imagery elicits strong autonomic responses. For example, when

listening to audiotaped personalized scripts of traumatic experiences, veterans with PTSD had relatively faster heart rate (HR), greater skin conductance (SC), and stronger facial electromyogram responses than veterans without PTSD. However, groups did not differ on their psychophysiological reactivity when listening to personalized neutral scripts (Orr, Pitman, Lasko, & Herz, 1993). In Pole's (2007) meta-analysis, PTSD was found to be associated with aggregate indices of larger responses to idiographic trauma cues. Analyses for HR and SC responses yielded similar effect sizes.

Most studies have implemented script-driven imagery in cross-sectional samples with male combat veterans (Orr, Metzger, Miller, & Kaloupek, 2004). Cross-sectional studies provide insight into the association between physiological measures and PTSD, but cannot address questions about the temporal relationship between physiological indices and development of, or recovery from, PTSD over time. Research examining the prospective relationship between psychophysiological reactivity to trauma reminders and the development of PTSD is fairly limited.

A few studies have examined the prospective relationship between measures of psychophysiological reactivity and PTSD. Except for Halligan, Michael, Wilhem, Clark, and Ehlers (2006), most studies have demonstrated increased HR reactivity to trauma reminders is associated with higher PTSD scores over time. For example, motor vehicle accident survivors with PTSD and greater HR reactivity to audiotaped descriptions of their crash measured 1–4 months posttrauma were more likely to maintain PTSD diagnoses at 12 months than those with less reactivity (Blanchard et al., 1996). Essler, Sartory, and Tackenberg (2005) found similar results with a more proximal initial assessment. Heart rate reactivity to idiographic trauma-related pictures (assessed on average 27 days posttrauma) significantly predicted of PTSD symptoms 3 months later. In a study that compared the predictive value of HR reactivity to a trauma narrative (1-week posttrauma) versus HR measured by paramedics at the scene of the injury, only HR reactivity to a trauma narrative was significantly correlated with PTSD scores 1-year posttrauma (O'Donnell, Creamer, Elliot, & Bryant, 2007).

In the current study, we expanded upon previous research by closely examining the relationship between physiological reactivity within 1 month of an assault and level of PTSD at 3-months posttrauma. We hypothesized that HR reactivity in response to an idiographic trauma monologue within 1 month of a traumatic event would serve as a prospective predictor of PTSD symptom severity at 3-months posttrauma. A second aim was to conduct an exploratory analysis of HR recovery (i.e., recovery over the first few minutes after the cessation of the trauma-relevant stimuli) as a prospective predictor of PTSD symptoms. This analysis was conducted on a magnitude of the physiological response to trauma content in the moment and recovery afterward because maladaptive responses to trauma content may be characterized by excessive and/or prolonged reactivity. However, studies of psychophysiology in PTSD tend to focus on the former and ignore the latter. Accordingly, we examined HR recovery following trauma cue exposure as a second measure of trauma reactivity. An exploratory aim was to examine the relationship between increased HR reactivity and specific PTSD symptom clusters. We hypothesized that clusters related to reexperiencing and arousal would be associated with increased HR reactivity because these clusters are more reflective of arousal than avoidance and numbing clusters. Finally, we conducted parallel analyses using measures of SC response to examine if the prospective relationships between increased trauma reactivity and PTSD symptoms over time is specific to HR or also generalizes to SC.

METHOD

Participants

Participants were part of a larger study designed to investigate the natural recovery of victims of a sexual or physical assault and were included in the current study if they had completed both the psychophysiological task and the Clinician Administered PTSD Scale (CAPS; Blake et al., 1990) at Time 1 ($N = 107$). At the initial assessment, participants were primarily single (57%), African American (72%) women with a mean age of 30.2 years ($SD = 8.7$) and a mean education of 12.6 years ($SD = 2.3$). At Time 1, participants reported experiencing an average of 2.4 ($SD = 1.7$) different types of potentially traumatic events in their lifetime other than the index assault and 74% met criteria for PTSD with the CAPS ($M = 67$, $SD = 24$), anchored to the index assault. Sixty-one women (57%) participated in a 3-month follow-up (Time 2), 49% of whom met criteria for PTSD at that time point ($M = 47$, $SD = 28$). Between the two assessments, 80% of participants received fewer than six therapy sessions and 50% received no therapy or counseling.

Measures

The Clinician Administered PTSD Scale (Blake et al., 1990) is a structured diagnostic interview with clinician-rated frequency and intensity of each DSM-IV symptom according to the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition Text Revision* (*DSM-IV-TR*; American Psychiatric Association, 2000). Although *DSM-IV-TR* delineates three symptom clusters, factor analyses have shown a 4-cluster model best fits current PTSD symptoms (King, Leskin, King, & Weathers, 1998). Severity scores were computed for total CAPS (range = 0–136) and four symptom clusters: reexperiencing, range = 0–40; avoidance, range = 0–16; numbing, range = 0–40; hyperarousal, range = 0–40). The CAPS has been shown to have excellent psychometric properties (Weathers, Keane, & Davidson, 2001). For this study, reliability coefficients for the CAPS total and four clusters at both time points as measured by Cronbach's alpha, ranged from .89 to .98. Interviewers were trained masters-level clinicians.

The Peritraumatic Dissociative Experiences Questionnaire (PDEQ; Marmar et al., 1994) is an eight-item

interviewer-administered measure of dissociative experiences during a traumatic event. Two items from the original measure were removed as they were less appropriate to rape victims (Griffin, Resick, & Mechanic, 1997) and internal consistency of the revised scale was .68 (Mechanic, Resick, & Griffin, 1998). The removed items were "Did you feel confused or disoriented?" and "Did you feel numb?"

To clarify the physiological responses used in the analyses, we have defined the various measures used in this study. Average heart rate level (HRL) in beats/minute and frequency of skin conductance responses (SCR) were computed for each phase. From these data, two HR variables and two SC variables were computed for use as independent variables in subsequent regression equations. Heart rate reactivity was computed by subtracting average heart rate level (HRL) during the first baseline phase (Phase 1) from average heart rate level (HRL) during the trauma phase (Phase 4). This index measures HR reactivity in response to discussing the trauma relative to baseline HR. Heart rate recovery was computed by subtracting baseline HRL (Phase 1) from HRL during the final recovery (Phase 5). This index measures the degree to which HR returns to baseline following both monologue tasks. The SC variables (i.e., SC reactivity and SC recovery) were computed to provide parallel SC measures to those computed for HR.

Procedure

Women who reported a completed sexual or physical assault were recruited for this study (see Gutner, Rizvi, Monson, & Resick, 2006; Griffin et al., 1997). Potential participants were screened and scheduled for the first assessment within 1 month of the assault. Individuals were ineligible for participation if they were unable to attend an assessment within this period, if they were illiterate, demonstrated psychosis, or were intoxicated at the time of assessment.

Participants were assessed at two time points: Time 1 (less than 1 month postassault) and at Time 2 (3 months postassault); mean number of days posttrauma until Time 1 was 13.1 ($SD = 5.0$; range = 3–26) and mean days posttrauma until Time 2 was 104.6 ($SD = 18.3$; range = 79–198). The procedures were identical for both assessment periods. After obtaining informed consent (session 1), participants completed self-report scales (about 1.5 hours to complete) and were given a 15-minute break before entering the laboratory. There was a 10–15-minute period when participants were seated, informed of procedures, and recording devices were attached. Thus, there was approximately 30 minutes between when the self-report questionnaires and the psychophysiological measurements were obtained. Following the psychophysiological tasks, a diagnostic interview, including the CAPS and modified PDEQ, was conducted.

Laboratory assessments were conducted in an 8 × 10 ft. room that was sound-insulated and temperature and humidity-controlled. Heart rate and skin conductance were collected throughout the entire laboratory assessment at a rate of five samples per second. Physiological measures of heart rate were generated by means of a Coulbourn Instruments (Allentown, PA) modular system. Heart rate measurements were obtained with an optical blood flow transducer attached to the nondominant hand on the distal phalanx of the second finger. Analogue outputs were converted to digital signals by an analog-digital converter (Coulbourn model S25-12). Digital outputs were interfaced with an IBM-compatible computer with the use of a Coulbourn LabLinc Interface, which allowed real-time waveform display of the data. Skin conductance data were obtained with silver/silver chloride 9-mm electrodes filled with isotonic paste and attached to the nondominant hand on the first and third fingers at the distal phalanx. Electrodes were attached to a skin conductance module (Coulbourn model S71-22), and a constant voltage (0.5 V) was applied and was used in the AC coupled (quick change) mode. Skin conductance waveforms were evaluated using a computer scoring algorithm. A response in excess of 0.10 ms was considered a valid SC response and frequency per phase was calculated.

There were five phases of assessment: (a) initial baseline, (b) neutral monologue, (c) recovery, (d) traumatic monologue, and (e) final recovery. During phase 1, baseline physiology was measured while the participant was alone for 5 minutes. Phase 2 commenced when the interviewer returned and prompted the participant to discuss a neutral topic for 5 minutes. Prompt sheets listing possible topics to discuss during the neutral phase were provided. These topics required participants to recall and describe some past neutrally valenced event (e.g., "a movie you saw"). Participants were instructed that the interviewer would be present but not speak during the phase. Next, participants were left alone for a 5-minute resting phase (Phase 3). In Phase 4, the interviewer returned and prompted participants to talk about the assault for 5 minutes. Participants were asked to describe the assault in detail and were given a prompt sheet that included aspects such as location and the assailant's words and actions. Participants were reminded that the experimenter would not speak during this phase. Phase 5 consisted of a third 5-minute resting (recovery) period. During the narrative phases, if participants fell silent, the experimenter prompted them to continue speaking.

Data Analysis

A series of hierarchical regression equations was conducted using CAPS severity and CAPS cluster scores at both time points as dependent variables and the various physiological variables as independent variables. Analyses included (a) physiological reactivity at Time 1 as a predictor of concurrent PTSD symptom severity (i.e., cross-sectional analyses Time 1), (b) physiological reactivity at Time 1 as a prospective predictor of PTSD symptom severity at Time 2 (i.e., prospective analyses), and (c) physiological reactivity at Time 2 as a predictor of concurrent PTSD symptom severity (i.e., cross-sectional analyses Time 2). To account for research

showing decreased physiological reactivity among individuals with high peritraumatic dissociation (Griffin et al., 1997) with this sample, the PDEQ was controlled for in the first step of all the analyses. In the prospective analyses, Time 1 CAPS was also controlled for in the first step of the analyses.

RESULTS

Study completers and noncompleters did not significantly differ on demographics, psychophysiological response measures, or Time 1 CAPS total and cluster scores. Additionally, participants who experienced a sexual assault did not significantly differ from those who experienced a physical assault on these measures, with the exception of income, χ^2 (5, $N = 105$) = 12.0, $p < .05$, where physical assault victims had higher incomes. Because half of the women received at least one counseling session between the two time points, women who did not receive any crisis counseling were compared to women who partook in at least one counseling session on Time 2 (CAPS severity scores at Time 2). The groups did not differ on the CAPS, $t(59) = 1.29$, *ns*, suggesting that counseling posttrauma did not account for Time 2 PTSD.

Analyses were done to examine the relationship between HR reactivity and PTSD symptoms. After controlling for PDEQ, the regression equation examining the associations between Time 1 HR reactivity (IV) and Time 1 PTSD total symptom severity (dependent variable) was not significant, $R^2 = .07$, $F(2, 57) = 2.23$, *ns*. Similar regression equations controlling for PDEQ and including Time 1 HR reactivity as an independent variable were conducted for each of the four PTSD symptom clusters as the dependent variables. Three of the four regression equations were significant: for reexperiencing, $R^2 = .07$, $F(2, 112) = 4.86$, $p < .05$; for avoidance, $R^2 = .07$, $F(2, 113) = 4.46$, $p < .05$; for numbing, $R^2 = .10$, $F(2, 113) = 6.18$, $p < .01$. However, HR reactivity was not a significant predictor of the PTSD cluster severity scores: for reexperiencing, $\beta = -.08$, *ns*; for avoidance, $\beta = .17$, *ns*; for numbing, $\beta = .05$, *ns*.

Table 1 summarizes the results from the five regression equations examining the associations between Time 1 HR reactivity (independent variable) and Time 2 PTSD symptoms (dependent variables), after controlling for Time 1 PDEQ and CAPS. Of note, Time 1 HR reactivity significantly predicted CAPS reexperiencing symptoms at Time 2 above and beyond initial PTSD and dissociation symptoms.

After controlling for PDEQ, the regression equation examining the association between Time 2 HR reactivity (independent variable) and Time 2 PTSD total symptom severity (dependent variable) was significant, $R^2 = .12$, $F(2, 56) = 3.81$, $p < .05$. Similar regression equations controlling for the PDEQ and including Time 2 HR reactivity as the independent variable were conducted for each of the four PTSD symptom clusters (dependent variables). Three of the four were significant: for reexperiencing, $R^2 = .10$, $F(2, 60) = 3.35$, $p < .05$; for avoidance, $R^2 = .12$, $F(2, 62) = 4.05$, $p < .05$; for hyperarousal, $R^2 = .15$, $F(2, 60) = 5.27$, $p < .01$. However, HR reactivity was not a significant predictor of the PTSD total or cluster severity scores: for $CAPS_{total}$, $\beta = -.13$, *ns*; for reexperiencing, $\beta = .00$, *ns*; for avoidance, $\beta = -.12$, *ns*; for hyperarousal, $\beta = -.08$, *ns*.

Analyses were done to examine the relationship between SC reactivity and PTSD symptoms. After controlling for PDEQ, the regression equation examining the association between Time 1 SC reactivity (independent variable) and Time 1 PTSD total symptom severity (dependent variable) was not significant, $R^2 = .07$, $F(2, 58) = 2.32$, *ns*. Similar regression equations controlling for the PDEQ and including Time 1 SC reactivity (independent variable) were conducted for each of the four PTSD symptom clusters (dependent variables) and two were significant: for reexperiencing, $R^2 = .07$, $F(2, 113) = 4.15$, $p < .05$; for numbing, $R^2 = .10$, $F(2, 114) = 6.42$, $p < .01$. However, SC reactivity was not a significant predictor of the PTSD cluster severity scores: for reexperiencing, $\beta = .03$, *ns*; for numbing, $\beta = -.04$, *ns*.

Table 2 summarizes the results from the five regression equations examining the associations between Time 1 SC reactivity (independent variable) and Time 2 PTSD symptoms (dependent variables), after controlling for Time 1 PDEQ and CAPS. Skin conductance reactivity was not a significant predictor of the PTSD total or cluster severity scores.

After controlling for PDEQ, the regression equation examining the association between Time 2 SC reactivity (independent variable) and Time 2 PTSD total symptom severity (dependent variable) was significant: $R_2 = .11$, $F(2, 56) = 3.48$, $p < .05$. Similar regression equations controlling for the PDEQ and including Time 2 SC reactivity as the independent variable were conducted for each of the four PTSD symptom clusters (dependent variables). Three of the four were significant: for reexperiencing, $R^2 = .10$, $F(2, 60) = 3.40$, $p < .05$; for avoidance, $R^2 = .12$, $F(2, 62) = 4.13$, $p < .05$; for hyperarousal, $R^2 = .15$, $F(2, 60) = 5.20$, $p < .01$. However, SC reactivity was not a significant predictor of the PTSD total or cluster severity scores: for $CAPS_{total}$, $\beta = -.08$, *ns*; for reexperiencing, $\beta = -.04$, *ns*; for avoidance, $\beta = -.12$, $p = .32$; for hyperarousal, $\beta = -.07$, *ns*.

Analyses were done to examine the relationship between HR recovery and PTSD symptoms. After controlling for PDEQ, the regression equation examining the association between Time 1 HR recovery (IV) and Time 1 PTSD total symptom severity was not significant, $R^2 = .07$, $F(2, 57) = 2.25$, *ns*. Similar regression equations controlling for PDEQ and including Time 1 HR recovery as the independent variable were conducted for each of the four symptom clusters (dependent variables) and two equations were significant: for reexperiencing, $R^2 = .07$, $F(2, 112) = 4.45$, $p < .05$; for numbing, $R^2 = .10$, $F(2, 113) = 6.01$, $p < .01$. However, HR recovery was not a significant predictor of the PTSD cluster severity scores: for reexperiencing, $\beta = -.07$, *ns*; for numbing: $\beta = .03$, *ns*.

Table 1. Regression Analyses of CAPS Scores at Time 2 on HR Trauma Reactivity Scores at Time 1 Controlling for Time 1 CAPS and PDEQ

Outcome predictors	*B*	*SE B*	β	$R^2/\Delta R^2$
CAPS total severity at Time 2				
1. PDEQ total score at Time 1	0.46	0.41	.12	.45***
CAPS total severity at Time 1	0.71	0.12	.63***	
2. PDEQ total score at Time 1	0.59	0.41	.15	.03***
CAPS total severity at Time 1	0.70	0.11	.62***	
HR reactivity	0.59	0.36	.16	
CAPS total reexperiencing cluster at Time 2				
1. PDEQ total score at Time 1	0.16	0.14	.13	.29***
CAPS reexperiencing cluster at Time 1	0.51	0.12	.48***	
2. PDEQ total score at Time 1	0.23	0.14	.18	.07***
CAPS reexperiencing cluster at Time 1	0.49	0.12	.47 ***	
HR reactivity	0.31	0.12	.27*	
CAPS total avoidance cluster at Time 2				
1. PDEQ total score at Time 1	0.12	0.07	.18	.30***
CAPS avoidance cluster at Time 1	0.56	0.13	.47	
2. PDEQ total score at Time 1	0.12	0.07	.19	.00***
CAPS avoidance cluster at Time 1	0.55	0.14	.46***	
HR reactivity	0.02	0.06	.04	
CAPS total numbing cluster at Time 2				
1. PDEQ total score at Time 1	−0.04	0.15	−.03	.29***
CAPS numbing cluster at Time 1	0.54	0.11	.54***	
2. PDEQ total score at Time 1	0.01	0.15	.01	.04***
CAPS numbing cluster at Time 1	0.54	0.11	.54***	
HR reactivity	0.25	0.14	.20	
CAPS total hyperarousal cluster at Time 2				
1. PDEQ total score at Time 1	0.32	0.15	.22*	.44***
CAPS hyperarousal cluster at Time 1	0.65	0.11	.57***	
2. PDEQ total score at Time 1	0.33	0.15	.23*	.00***
CAPS hyperarousal cluster at Time 1	0.65	0.11	.57***	
HR reactivity	0.07	0.13	.05	

Note. CAPS = Clinician Administered PTSD Scale; HR = heart rate; PDEQ = Peritraumatic Dissociative Experiences Questionnaire.
* $p \leq .05$. *** $p < .001$.

Table 3 summarizes the results from the five regression equations examining the associations between HR recovery (independent variable) and Time 2 PTSD symptoms (dependent variables), after controlling for Time 1 PDEQ and CAPS. Of note, Time 1 HR recovery significantly predicted reexperiencing and numbing symptoms at Time 2. Thus, the degree to which HR does not recover following trauma and neutral monologues at Time 1 is predictive of reexperiencing and numbing symptoms at 3-months follow-up, beyond initial PTSD and dissociative symptoms.

After controlling for PDEQ, the regression equation examining the association between Time 2 HR recovery (independent variable) and Time 2 PTSD total symptoms severity (dependent variable) was significant, $R^2 = .12$, $F(2, 55) = 3.73$, $p < .05$. Similar regression equations controlling for PDEQ and including Time 2 HR recovery as the independent variables were conducted for each of the four PTSD symptom clusters (dependent variables). Three of the four were significant: for reexperiencing, $R^2 = .10$, $F(2, 59) = 3.36$, $p < .05$; for avoidance: $R^2 = .11$, $F(2, 61) = 3.82$, $p < .05$; for hyperarousal: $R^2 = .16$, $F(2, 59) = 5.77$, $p < .01$. However, HR recovery was not a significant predictor of the PTSD total or cluster severity scores: for $CAPS_{total}$: $\beta = -.13$, *ns*; for reexperiencing: $\beta = -.05$, *ns*; for avoidance: $\beta = -.10$, *ns*; for hyperarousal: $\beta = -.15$, *ns*).

Analyses were done to examine the relationship between SC recovery and PTSD symptoms. After controlling for PDEQ, the regression equation examining the association between Time 1 SC

Table 2. Regression Analyses of CAPS Scores at Time 2 on HR Recovery Scores at Time 1 Controlling for Time 1 CAPS and PDEQ

Outcome predictors	*B*	*SE B*	β	$R^2/\Delta R^2$
CAPS total severity at Time 2				
1. PDEQ total score at Time 1	0.45	0.40	.11	.45***
CAPS total severity at Time 1	0.71	0.11	.63***	
2. PDEQ total score at Time 1	0.68	0.44	.17	.02***
CAPS total severity at Time 1	0.71	0.11	.63***	
SC trauma reactivity	0.21	0.16	.14	
CAPS total reexperiencing Cluster at Time 2				
1. PDEQ total score at Time 1	0.17	0.14	.14	.29***
CAPS reexperiencing cluster at Time 1	0.50	0.12	.48***	
2. PDEQ total score at Time 1	0.25	0.15	.20	.02***
CAPS reexperiencing cluster at Time 1	0.49	0.12	.46***	
SC trauma reactivity	0.07	0.06	.16	
CAPS total avoidance cluster at Time 2				
1. PDEQ total score at Time 1	0.11	0.07	.17	.30***
CAPS avoidance cluster at Time 1	0.57	0.13	.47***	
2. PDEQ total score at Time 1	0.15	0.07	.24*	.02***
CAPS avoidance cluster at Time 1	0.57	0.13	.47***	
SC trauma reactivity	0.04	0.03	.15	
CAPS total numbing cluster at Time 2				
1. PDEQ total score at Time 1	−0.04	0.15	−.03	.30***
CAPS numbing cluster at Time 1	0.54	0.11	.55***	
2. PDEQ total score at Time 1	0.05	0.16	.04	.02***
CAPS numbing cluster at Time 1	0 .56	0.11	.57***	
SC trauma reactivity	0.09	0.06	.17	
CAPS total hyperarousal cluster at Time 2				
1. PDEQ total score at Time 1	0.31	0.15	.21*	.43***
CAPS hyperarousal cluster at Time 1	0.64	0.11	.57***	
2. PDEQ total score at Time 1	0.33	0.16	.23*	.00***
CAPS hyperarousal cluster at Time 1	0.64	0.11	.57***	
SC trauma reactivity	0.02	0.06	.04	

Note. CAPS = Clinician Administered PTSD Scale; SC = skin conductance; PDEQ = Peritraumatic Dissociative Experiences Questionnaire.
$^{*}p \leq .05$. $^{***}p < .001$.

recovery (independent variables) and Time 1 PTSD total symptom severity (dependent variables) was not significant, $R^2 = .09$, $F(2, 58) = 2.99$, *ns*. Similar regression equations controlling for PDEQ and including Time 1 SC recovery as the independent variable were conducted for each of the four PTSD symptom clusters (dependent variables). Three of the four regressions were significant: for reexperiencing, $R^2 = .10$, $F(2, 113) = 6.22$, $p < .01$; for avoidance, $R^2 = .06$, $F(2, 114) = 3.61$, $p < .05$; for numbing, $R^2 = .10$, $F(2, 114) = 6.49$, $p < .01$. Furthermore, for reexperiencing, SC recovery emerged as a significant predictor ($\beta = .18$, $p < .05$). Thus, the degree to which SC does not recover following trauma and neutral monologues at Time 1 is predictive of concurrent reexperiencing symptoms, after controlling for initial dissociative symptoms. However, for all other variables, SC recovery was not a significant predictor: for avoidance, $\beta = .13$, *ns*; for numbing, $\beta = -.05$, *ns*.

Results from the five regression equations examining the associations between Time 1 SC recovery (independent variable) and Time 2 PTSD symptoms severity (dependent variable) after controlling for Time 1 PDEQ and CAPS are included in Table 4. Of note, SC recovery at Time 1 was significantly related to reexperiencing symptoms at Time 2 ($p < .05$). Thus, the degree to which SC does not recover following trauma and neutral monologues at Time 1 is predictive of reexperiencing symptoms at 3-months follow-up, after controlling for initial reexperiencing and dissociative symptoms.

Table 3. Regression Analyses of CAPS Scores at Time 2 on HR Recovery Scores at Time 1 Controlling for Time 1 CAPS and PDEQ

Outcome predictors	*B*	*SE B*	β	$R^2/\Delta R^2$
CAPS total severity at Time 2				
1. PDEQ total score at Time 1	0.46	0.41	.12	.45***
CAPS total severity at Time 1	0.71	0.12	.63***	
2. PDEQ total score at Time 1	0.57	0.41	.14	.02***
CAPS total severity at Time 1	0.70	0.11	.62***	
HR recovery	0.64	0.42	.15	
CAPS total reexperiencing cluster at Time 2				
1. PDEQ total score at Time 1	0.16	0.14	.13	.29***
CAPS reexperiencing cluster at Time 1	0.51	0.12	.48***	
2. PDEQ total score at Time 1	0.22	0.14	.17	.07***
CAPS reexperiencing cluster at Time 1	0.49	0.12	.47 ***	
HR recovery	0.35	0.14	.26*	
CAPS total avoidance cluster at Time 2				
1. PDEQ total score at Time 1	0.12	0.07	.18	.30***
CAPS avoidance cluster at Time 1	0.56	0.13	.47***	
2. PDEQ total score at Time 1	0.12	0.07	.19	.00***
CAPS avoidance cluster at Time 1	0.56	0.13	.46***	
HR recovery	0.02	0.07	.03	
CAPS total numbing cluster at Time 2				
1. PDEQ total score at Time 1	−0.04	0.15	−.03	.29***
CAPS numbing cluster at Time 1	0.54	0.11	.54***	
2. PDEQ total score at Time 1	0.01	0.15	.01	.05***
CAPS numbing cluster at Time 1	0.54	0.11	.54***	
HR recovery	0.34	0.16	.23*	
CAPS total hyperarousal cluster at Time 2				
1. PDEQ total score at Time 1	0.32	0.15	.22*	.44***
CAPS hyperarousal cluster at Time 1	0.65	0.11	.57***	
2. PDEQ total score at Time 1	0.31	0.15	.21*	.00***
CAPS hyperarousal cluster at Time 1	0.65	0.11	.58***	
HR recovery	−0.05	0.16	−.03	

Note. CAPS = Clinician Administered PTSD Scale; HR = heart rate; PDEQ = Peritraumatic Dissociative Experiences Questionnaire.
$^{*}p \leq .05$. $^{***}p < .001$.

After controlling for PDEQ, the regression equation examining the association between Time 2 SC recovery (independent variable) and Time 2 PTSD total symptom severity (dependent variable) was significant, $R^2 = .11$, $F(2, 56) = 3.52$, $p < .05$. Similar regression equations controlling for PDEQ and including Time 2 SC recovery as the independent variable were conducted for each of the four PTSD symptom clusters. Three of the four regressions were significant: for reexperiencing, $R^2 = .10$, $F(2, 60) = 3.38$, $p < .05$; for avoidance, $R^2 = .10$, $F(2, 62) = 3.72$, $p < .05$; for hyperarousal, $R^2 = .15$ $F(2, 60) = 5.50$, $p < .01$. However, SC recovery was not a significant predictor of the PTSD total or cluster severity scores: for $CAPS_{total}$, $\beta = -.09$, *ns*; for reexperiencing, $\beta = -.03$, *ns*; for avoidance, $\beta = -.60$, *ns*; for hyperarousal, $\beta = -.11$, *ns*.

DISCUSSION

Previous research has demonstrated a prospective relationship between HR reactivity to trauma reminders and increased/maintained PTSD symptoms over time. The current study adds to this literature by addressing two primary questions: Is this relationship specific to increased HR during exposure to the traumatic memory (i.e., through the monologue procedure) or does it generalize to a prolonged increase in HR after the trauma

Table 4. Regression Analyses of CAPS Scores at Time 2 on SC Recovery Scores at Time 1 Controlling for Time 1 CAPS and PDEQ

Outcome predictors	*B*	*SE B*	β	$R^2/\Delta R^2$
CAPS total severity at Time 2				
1. PDEQ total score at Time 1	0.45	0.40	.11	.45***
CAPS total severity at Time 1	0.71	0.11	.63***	
2. PDEQ total score at Time 1	0.49	0.40	.12	.03***
CAPS total severity at Time 1	0.68	0.11	.60***	
SC trauma recovery	0.36	0.21	.16	
CAPS total reexperiencing cluster at Time 2				
1. PDEQ total score at Time 1	0.17	0.14	.14	.29***
CAPS reexperiencing cluster at Time 1	0.50	0.12	.48***	
2. PDEQ total score at Time 1	0.19	0.14	.15	.04***
CAPS reexperiencing cluster at Time 1	0.45	0.12	.43 ***	
SC trauma recovery	0.15	0.07	.21*	
CAPS total avoidance cluster at Time 2				
1. PDEQ total score at Time 1	0.11	0.07	.17	.30***
CAPS avoidance cluster at Time 1	0.57	0.13	.47***	
2. PDEQ total score at Time 1	0.11	0.07	.18	.01***
CAPS avoidance cluster at Time 1	0.55	0.13	.46***	
SC trauma recovery	0.04	0.04	.11	
CAPS total numbing cluster at Time 2				
1. PDEQ total score at Time 1	−0.04	0.15	−.03	.30***
CAPS numbing cluster at Time 1	0.54	0.11	.55***	
2. PDEQ total score at Time 1	−0.04	0.15	−.03	.03***
CAPS numbing cluster at Time 1	0.54	0.11	.55***	
SC trauma recovery	0.13	0.08	.17	
CAPS total hyperarousal cluster at Time 2				
1. PDEQ total score at Time 1	0.31	0.15	.21*	.43***
CAPS hyperarousal cluster at Time 1	0.64	0.11	.57***	
2. PDEQ total score at Time 1	0.33	0.14	.22*	.02***
CAPS hyperarousal cluster at Time 1	0.62	0.11	.56***	
SC trauma recovery	0.11	0.08	.14	

Note. CAPS = Clinician Administered PTSD Scale; SC = skin conductance; PDEQ = Peritraumatic Dissociative Experiences Questionnaire.
* $p \leq .05$. *** $p < .001$.

monologue is finished, or even to other measures of physiology such as skin conductance? What are the specific relationships between increased physiological reactivity and the different PTSD symptom clusters?

In contrast to the results found by a previous study (e.g., Blanchard et al., 1996), HR reactivity during a trauma monologue within 1 month of the traumatic event was not a significant predictor of greater overall PTSD symptom severity at 3 months. This may be explained by the current study's more stringent analyses. We assessed the relationship between HR reactivity and PTSD symptoms at Time 2 after controlling for initial PTSD and dissociative symptoms, which was not done previously. Essler and colleagues (2005) conducted secondary analyses in which initial reexperiencing symptoms and their HR reactivity measure were entered together in the model predicting PTSD symptoms and found that HR reactivity no longer emerged as an independent predictor, which parallels the current study.

However, when PTSD symptom severity was deconstructed into the four symptom clusters, three of the four measures of physiological reactivity in response to the trauma monologue predicted reexperiencing symptoms at 3 months after controlling for initial PDEQ and reexperiencing. Reexperiencing symptoms are hallmark symptoms of PTSD and are theorized as unique symptoms differentiating PTSD from other anxiety disorders (Simms

et al., 2002). The current results therefore suggest that physiological reactivity to trauma monologues prospectively predicts the hallmark reexperiencing symptoms of PTSD. This finding is compelling given that HR reactivity, HR recovery, and SC recovery emerged as significant predictors after controlling for initial dissociation and reexperiencing symptoms. This pattern of results highlights the contribution of the sympathetic nervous system (SNS) to the physiological reactivity in response to the trauma monologue procedure (e.g., Bernston, Cacioppo, & Quigley, 1993; Pole, 2007). The SNS works to initiate the body's stress response and the parasympathetic nervous system (PNS) is involved in homeostasis. Skin conductance is primarily reflective of SNS responses, whereas both the SNS and the PNS contribute to HR.

After controlling for initial numbing and dissociative symptoms, HR recovery at Time 1 emerged as a significant predictor of increased numbing symptoms such that sustained increase in HR after completing the monologues predicted more numbing symptoms. Although the other three physiological measures did not reach significance, the effects were in the predicted direction. This pattern of results provides support for physiological reactivity to the traumatic memory predicting increased numbing over time. This is particularly notable because the relationship between sustained HR elevations after the end of the trauma narrative and numbing symptoms cannot be explained by initial dissociation.

Given the findings that increased physiological reactivity to the trauma narratives were predictive of reexperiencing symptoms and perhaps numbing (significant for HR recovery only), it is interesting to consider these findings in relation to Horowitz's (1986) model. Horowitz describes trauma as evoking the internal processes of intrusion and denial, where trauma survivors shift between these two opposing responses until the trauma resolves. In this model, reexperiencing symptoms of PTSD are included in intrusions and emotional numbing is a component of denial. Thus, based on this model, it is not surprising that individuals with greater reactivity to the trauma monologue may have both increased reexperiencing symptoms over time coupled with increased numbing symptoms.

Although numbing and avoidance symptoms share a cluster within the *DSM-IV-TR* framework of PTSD, factor analyses have supported these as distinct clusters (e.g., King et al., 1998). The current study provides indirect support for this distinction because the regression equations involving avoidance did not parallel those found for numbing. This may be because numbing is an emotional process and avoidance symptoms focus on overt behaviors. Perhaps increased physiological reactivity may influence counter-emotional processes more so than overt behaviors.

Although the primary focus of the current study was the prospective relationship between heightened psychophysiological reactivity to a trauma monologue procedure and PTSD severity, it is important to note that the concurrent associations between these variables were mostly nonsignificant. The one exception was the concurrent relationship between SC recovery and reexperiencing symptoms at Time 1. Although Pole's (2007) meta-analysis supports concurrent associations between similar psychophysiological variables and PTSD symptoms, not all published studies have found significant relationships. One important difference that distinguishes the current study from most studies on this topic is that the assessments in this investigation all occur within 3 months of the traumatic event. Due to high rates of PTSD at these time points with victims of interpersonal trauma, there may be a truncated range of PTSD symptoms, and this might contribute to the lack of significant findings.

The primary strengths of the current study are the use of a prospective design and the timing of the first assessment within 1-month posttrauma. However, several limitations to the study are important to note. First, although there were several significant prospective associations between three measures of physiological reactivity and the reexperiencing cluster, as well as a significant prospective relationship between HR recovery and numbing, the added variance of these physiological measures (change in R^2) was small. In addition, the intensity of idiosyncratic trauma monologues may vary non-systematically among participants. We believe this limitation to be mitigated by the evidence that individuals' reactivity has been demonstrated to be more robust in response to personalized trauma-related stimuli than standardized scripts (e.g., Pole, 2007). An additional limitation is the significant drop out rate (43%), which is likely at least in part due to the stress in the participants' lives following being raped or physically assaulted. However, because of the lack of significant differences between the dropouts and completers on demographic measures, psychophysiological response measures, and CAPS total and cluster scores at Time 1, we believe this limitation to be relatively minor.

In sum, the current study suggests that greater physiological reactivity to self-generated trauma monologues within 1 month of trauma predicts PTSD reexperiencing symptom severity at 3 months and there is limited evidence for the prediction of numbing symptom severity as well. Thus, it appears that the previously demonstrated prospective relationship between HR reactivity to trauma cues and PTSD symptoms is most robust for reexperiencing symptoms, after controlling for initial PTSD and dissociative symptoms. In addition, this relationship generalizes to a measure of prolonged increase in HR after the trauma cues are no longer present and to a measure of SC as well. These findings highlight the predictive value of increased reactivity to trauma cues in the development and maintenance of PTSD symptoms. Individuals who are most reactive to trauma reminders shortly after the trauma might be at increased risk for chronic PTSD; therefore, it might be most fruitful to target early intervention efforts toward these individuals.

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