Published in final edited form as:

J Trauma Dissociation. 2012 ; 13(1): 32–50. doi:10.1080/15299732.2011.608628.

Prior peritraumatic dissociative experiences affect autonomic reactivity during trauma recall

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Abstract

To better understand the psychophysiological correlates of peritraumatic dissociation (PD), the present study examined the relationship between reports of prior PD and sympathetic and parasympathetic functioning in response to a laboratory stress paradigm in 39 traumatized female undergraduates. Participants were asked to talk about their most distressing traumatizing experience while continuous measures of heart rate (HR), pre-ejection period (PEP), and respiratory sinus arrhythmia (RSA) were taken. Overall, high dissociators had significantly larger increases in HR and larger decreases in PEP and RSA during trauma recall. In opposition to our hypothesis, these results suggest that female trauma victims reporting high levels of PD during a prior trauma may be more reactive to traumatic reminders than victims who reported lower levels of PD.

Keywords

peritraumatic dissociation; psychophysiology; heart rate; pre-ejection period; respiratory sinus arrhythmia

Experiences of dissociation during and immediately following a potentially traumatizing event are one of the most consistent predictors of posttraumatic stress disorder (PTSD) across a variety of traumatized samples (Breh & Seidler, 2007; Lensvelt-Mulders et al., 2008; Ozer, Best, Lipsey, & Weiss, 2003). For example, individuals reporting peritraumatic dissociation (PD) are approximately four times more likely to develop acute PTSD one month following a motor vehicle accident (MVA) than are individuals without PD (Ursano et al., 1999). Further, individuals reporting high levels of PD are at an increased risk for PTSD independent of other variables such as general dissociative tendencies (Marmar, Weiss, Schlenger, & Fairbank, 1994), anxiety and subjective loss (Koopman, Classen, & Spiegel, 1994), trauma exposure, and social support (Shalev, Peri, Canetti, & Schreiber, 1996). Much research has been devoted to better understanding PD, its predictors, and consequences (Marmar, Weiss, & Metzler, 1997; Noyes & Kletti, 1977); however, the role that PD plays in the development of trauma-related psychopathology remains unclear.

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PD has been conceptualized as a primitive defense mechanism used to protect the individual from the horror and overwhelming emotion that characterizes a traumatic event (Bremner & Brett, 1997; Marmar, et al., 1997). In instances involving threat where fight-or-flight is not an option, an individual may use dissociation as a psychological technique to escape the traumatic event (Huopainen, 2002). Although initially used as a coping technique to deal with trauma, peritraumatic dissociation may also serve as a means through which trauma could lead to PTSD (Marmar, Weiss, & Metzler, 1998). Researchers have tried to understand the possible mechanisms through which PD may lead to PTSD by examining the psychophysiological correlates of PD. However, results have been mixed (Delahanty, Royer, Raimonde, & Spoonster, 2003; Griffin, Resick, & Mechanic, 1997; Koopman et al., 2004; Williams, Haines, & Sale, 2003). Some studies have found PD to be related to heightened autonomic nervous system (ANS) functioning (Kuhn, Blanchard, Fuse, Hickling, & Broderick, 2006; Ladwig et al., 2002; Nixon, Bryant, Moulds, Felmingham, & Mastrodomenico, 2005) while others have found PD to be related to suppressed ANS functioning (Delahanty, et al., 2003; Elssesser, Freyth, Lohrmann, & Sartory, 2008; Griffin, et al., 1997; Koopman, et al., 2004; Pole et al., 2005; Simeon, Yehuda, Knutelska, & Schmeidler, 2008). Still others have found no relationship between ANS functioning and PD (Halligan, Michael, Wilhelm, Clark, & Ehlers, 2006; Kaufman et al., 2002). These contradictory findings could be explained by research suggesting within- and between-person variability in responses to trauma and trauma-related stimuli (Lanius, Bluhm, Lanius, & Pain, 2006; Nijenhuis & den Boer, 2009). For instance, individuals with Dissociative Identity Disorder (DID) displayed either hyperarousal, hypoarousal, or no physiological change in response to a trauma-related paradigm depending on which part of their personality was dominant during the task (Putnam, Zahn, & Post, 1990; Reinders et al., 2006). Similarly, humans and nonhuman animals may experience a cascade of responses during potentially traumatizing events that begin with freezing, fight, or flight and end with tonic immobility (TI) (Marx, Forsyth, Gallup, Fusé, & Lexington, 2008). Different parts of these defense reactions have been related to different physiological responses such that freezing, fight, and flight are associated with physiological hyperarousal while TI is typically associated with physiological hypoarousal (for a review see Nijenhuis & den Boer, 2009). Therefore, individuals may exhibit a variety of responses depending on the psychophysiological states that are being elicited at the time of measurement (within-person variability). Further, research has suggested that trauma victims may display two types of responses to trauma, each associated with different physiological underpinnings (intrusions and hyperarousal vs. dissociation and hypoarousal) and each uniquely related to the development of PTSD (between-person variability, for a review see Lanius, et al., 2006).

Further complicating findings is that it is unclear whether observed ANS alterations reflect activation of the sympathetic nervous system (SNS), activation of the parasympathetic nervous system (PNS), or the interaction of both. For instance, HR is determined by input from both the SNS and PNS with the PNS exerting more control (Berntson, Cacioppo, & Quigley, 1991, 1993). Porges (1995) has argued that depressed parasympathetic tone and not simply sympathetic reactivity may better determine the physiological stress response. Initially it was assumed that the SNS and PNS worked in a reciprocal fashion such that an increase in one system would be associated with a decrease in the other (doctrine of autonomic reciprocity; Berntson, et al., 1991). Although depressed parasympathetic tone is typically associated with an increase in sympathetic reactivity in healthy individuals, this reciprocal relationship may not be true for individuals vulnerable to stress-related disorders (Porges, 1995).

Due to the complex relationship between SNS and PNS activity, research should ideally measure both branches of the ANS. To date, the best noninvasive measure of PNS functioning is heart rate variability (HRV) or the beat-to-beat changes in heart rate (Task

Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). HRV is defined as the increases and decreases in the time between heart beats (i.e. heart periods or R-peaks) (Berntson et al., 1997). Vagal control of the heart changes with respiration, such that inspiration causes an increase in HR and exhalation causes a decrease in HR. Thus, respiratory sinus arrhythmia (RSA) is used to measure HRV (Berntson, et al., 1997). The best noninvasive measure of SNS functioning is pre-ejection period (PEP) which is defined as the length of time between the contraction of the heart (onset of systole) and the ejection of blood out of the heart and into the aorta (onset of left ventricular ejection) (Sherwood, Allen, Obrist, & Langer, 1986). A shortening of this time interval would correspond with a faster HR. Thus, a decrease in PEP reflects an increase in SNS activation.

Individuals with PTSD have consistently demonstrated elevated basal physiological arousal and HR hyperreactivity in response to trauma and non-trauma related cues (Buckley & Kaloupek, 2001; Pole, 2007). However, it is unclear whether these responses are due to overactivation of the SNS, underactivation of the PNS (low parasympathetic tone), or the interaction of both. More recent research has suggested that individuals with PTSD have lower parasympathetic activity (e.g. lower respiratory sinus arrhythmia [RSA]) than similarly traumatized individuals without PTSD (Cohen et al., 2000; Cohen et al., 1998; Keary, Hughes, & Palmieri, 2009; Sack, Hopper, & Lamprecht, 2004). Further, increases in HRV over the course of treatment have been associated with decreases in PTSD symptoms in one pilot study assessing the effectiveness of RSA biofeedback (Zucker, Samuelson, Muench, Greenberg, & Gevirtz, 2009). These findings suggest that hyperarousal in individuals with PTSD is at least somewhat due to lower parasympathetic tone. To date, no studies have examined the relationship between PD and both SNS and PNS functioning. Such research into autonomic correlates of PD will aid in conceptualizing the relationship between PD and PTSD as well as inform mechanisms through which PD and PTSD are physiologically related.

Present Study

The present study examined the relationship between reports of prior PD and autonomic reactivity to a laboratory paradigm in undergraduate female victims of interpersonal trauma. Participants were asked to talk about their most distressing trauma while physiological measurements were assessed using electrocardiography (ECG) and impedance cardiography. Despite mixed findings concerning the relationship between PD and ANS activity, our methods were modeled after those of Griffin et al. (1997); therefore, similar to their findings, we hypothesized that high peritraumatic dissociators would have lower HR during trauma recall and that lower HR would be due to increased PEP and RSA.

METHOD

Participant Recruitment

Undergraduate females 18 years of age or older completed the Traumatic Stress Schedule (TSS; Norris, 1990) during mass testing procedures for course credit. Females were considered eligible to participate in the laboratory task if they indicated experiencing at least one interpersonal potentially traumatizing event on the TSS (i.e., have you ever experienced a mugging, physical attack, or sexual assault). Given that research has suggested gender differences in HR and RSA responses during stressful situations (Tardy & Allen, 1998; Weidner, Friend, Ficarrotto, & Mendell, 1989; Ziegler, Dannehl, Muhlen, Spuler, & Gries, 1992), the present study was limited to females.

Participants were contacted by telephone and screened for exclusionary criteria including a history of heart disease or hypertension and use of antihypertensive medications. Current medication use, age and body mass index (BMI) were assessed at this time; however, these variables did not serve as exclusionary criteria. Participants reporting use of alcohol/drugs during the trauma and those reporting no memory of the event ($n=13$) were excluded from further analyses since use of alcohol/drugs can mimic dissociative states (Good, 1989). The final sample consisted of 39 females who were predominantly Caucasian, young, and healthy (see Table 1).

Procedure

The Human Subjects Institutional Review Board of Kent State University approved the following procedures. Eligible participants were told that based on their answers to some of the questionnaires they completed during mass testing; they were eligible to participate in a laboratory experiment for credit. Upon arrival at the laboratory, the study was explained in detail, and written informed consent was obtained. The participant was seated in a comfortable chair while being connected to the ECG and impedance cardiograph. After the initial setup, the participant rested for 10 minutes while HR and RSA readings were taken for baseline measurements. In addition, during this rest period, participants completed several questionnaires including a demographics questionnaire, general health questionnaire, and a menstrual cycle form.

The experimental paradigm was based on that of Griffin et al. (1997). After the 10-minute rest period, the experimental task began. The task consisted of two speech phases each lasting four minutes (neutral phase and trauma recall phase) plus a resting phase. For the neutral phase, the participant was asked to talk about the shoes they were currently wearing in detail (based on Pennebaker, Kiecolt-Glaser, & Glaser, 1988). During the trauma phase, the participant was asked to describe their most potentially traumatizing experience in detail including aspects such as where she was, what the assailant said and did, and what her thoughts and reactions were. Although participants were recruited based on their experience of a potentially traumatizing interpersonal event, not all participants recalled an interpersonal trauma as their most distressing experience during the trauma recall phase (see Table 2). However, all recalled traumas met criterion A1, and all participants reported experiencing fear, helplessness, or horror during the event (criterion A2).

The order of the neutral and trauma phases was counterbalanced and participants were told the order of the phases following the baseline period. Participants rested for five minutes (rest phase) in between the neutral and trauma phases. Following the last phase, participants sat quietly for 10 minutes of recovery measurements. Participants completed a measure of subjective distress (Subjective Units of Distress Scale, SUDS: Wolpe, 1958) at the end of each phase. The experimenter was not in the room during the tasks, but prompted the participant via a microphone when she should start and stop speaking for each phase. Continuous physiological measurements were obtained throughout the entire task.

Following the task, participants completed several questionnaires including the Posttraumatic Diagnostic Scale, (PDS: Foa, Riggs, Dancu, & Rothbaum, 1993), and the Peritraumatic Dissociative Experiences Questionnaires (PDEQ: Marmar, et al., 1997). Participants were then fully debriefed and received course credit for participating.

Measures

Demographic information and details about the trauma—During the baseline rest phase, participants answered several questions concerning basic demographic information, general health, and current stage of the menstrual cycle. Following the laboratory task,

participants provided descriptive information regarding their traumatizing experience. This questionnaire assessed to what extent participants feared for their own life during the trauma, could have avoided the trauma, and had control over things that happened during the event. These three items were rated on a scale from 1 (not at all) to 7 (a lot). Several additional items assessed the length of time since the trauma occurred, age at time of trauma, if they had consumed alcohol/drugs during the trauma, presence of lapses in memory during the trauma, and if they sought professional help for mental health issues.

Trauma history—Trauma history was assessed with a modified version of the Traumatic Stress Schedule (TSS; Norris, 1990). Participants were asked to report the occurrence of 10 different potentially traumatizing events in their lifetime (yes/no; e.g., assault, robbery, natural disasters, accidents). Items were summed to provide a total trauma history score assessing the number of different traumas endorsed.

Current PTSD diagnosis—Current PTSD diagnostic levels were measured as a possible covariate. The Posttraumatic Diagnostic Scale-Self Report (PDS; Foa, et al., 1993) is a self-report questionnaire that assesses symptoms of re-experiencing, avoidance, and hyperarousal experienced in the past month. In order to receive a likely diagnosis of PTSD, participants had to report at least one re-experiencing, three avoidance, and two hyperarousal symptoms (American Psychiatric Association, 2000). Cronbach's alpha for the present sample was .91.

Peritraumatic Dissociation—The Peritraumatic Dissociative Experiences Questionnaire-Self-Report Version (PDEQ; Marmar, et al., 1997) is a 10-item self-report questionnaire that assesses retrospective reports of depersonalization, derealization, amnesia, out of body experiences, and altered time perception. The PDEQ is scored on a 5-point Likert-scale from 1 (not at all) to 5 (extremely true). Items were summed to yield a total PD score. Cronbach's alpha was .85 for the present sample.

Subjective Distress—The Subjective Units of Distress Scale (SUDS; Wolpe, 1958) served as a manipulation check to ensure that the trauma recall phase was perceived as more stressful than the neutral and rest phases. Participants were asked to rate how they generally feel and then how they felt after the neutral, trauma, and resting phases on a scale from 0 (Calm/Relaxed) to 100 (Nervous/Tense) with a midpoint of 50 representing Neutral.

Physiological Measurements—Four different physiological measurements were obtained using a combination of electrocardiography and impedance cardiography. Respiration rate (RR) was examined as a covariate since changes in RSA could be due to an increase in respiration (Porges & Byrne, 1992). HR and RSA were measured via electrocardiography while RR and PEP were measured via impedance cardiography. Electrocardiography detects changes in the electrical activity of the heart while impedance cardiography detects electrical resistance due to changes in blood flow in the torso. Electrocardiographic measures were taken via three disposable, self-adhesive Ag-AgCl electrodes attached to the torso in a three-lead configuration. Impedance measurements were taken via four tetrapolar aluminum/mylar tape bands placed around the neck and torso. The electrocardiographic and impedance measures were sampled continuously at 500 Hz using the HIC-2000 Impedance Cardiograph (Instrumentation For Medicine, Inc.) and commercial software (Litvack, Lozano, Ernst, Berntson, & Cacioppo, 1999). Data were digitized and imported into ANS Suite in 60-second epochs. R-peaks were marked using a hybrid method with detection based on the ANS Suite software program and visual inspection of the graph to correct for errors. After R-peak detection, the distance between the R-peaks was calculated and a heart period time series was created using a "weighted" beat algorithm. Any

sharp transitions in the heart period time series were smoothed using the Hanning method (Litvack, et al., 1999).

RSA was defined as the heart rate variability (HRV) in the frequency band between .12 and .40 Hz which represents parasympathetic activity that responds to respiration and can influence HR (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). The power spectrum of the heart period time series was calculated using a Fast Fourier Transform (FFT) and scaled to msec²/Hz. RSA was calculated as the natural log of the area under the heart period power spectrum within the corner frequencies of the band pass filter. The mean HR was calculated as 60,000 divided by the mean of the unfiltered heart period time series. PEP (in milliseconds) was defined as the duration of time between the onset of left ventricular depolarization (initial upswing of the QRS complex) and the ejection of blood into the aorta (dZ/dt B-point) (Sherwood et al., 1990). The respiratory power spectrum was calculated using the FFT and scaled to units²/Hz. This power spectrum was then used to calculate the mean RR. RR did not differ between groups over time, $F(8, 140)=1.76$, $p=.09$, and was not significantly correlated with RSA during any phase of the laboratory protocol (all p values $>.05$), thus it was not included as a covariate in subsequent RSA analyses.

RESULTS

Data Reduction and Statistical Analyses

Analyses of variance (ANOVAs: continuous dependent variables), Pearson product correlations (two continuous variables), and chi-square analyses (two categorical variables) were calculated to test for possible control variables.

Baseline levels of physiological variables were computed by averaging the last five minutes of data collected. Reactivity scores were computed by subtracting the baseline from the average levels of physiological data collected during the 4-minute trauma phase. PD scores on the PDEQ ranged from 10 – 46 with an average score of 26.59 ($SD = 8.60$). Adapted from Griffin et al. (1997), participants were categorized into three groups based on their PD scores with high dissociators scoring at least 1 SD above the mean (scores from 32–40), low dissociators scoring at least 0.50 SD below the mean (scores from 10 to 21) and moderate dissociators representing intermediate scores (between 22 and 31).

Separate repeated measures analyses of covariance (RM ANCOVAs) were used to examine the relationship between dissociation group and HR, PEP and RSA across laboratory phases (baseline, trauma, rest, neutral, recovery). Further, three separate ANCOVAs were conducted to examine the relationship between dissociation group and mean reactivity during the trauma phase. In addition, Pearson product moment correlations were conducted to assess the linear relationship between total dissociation scores and HR, PEP, and RSA reactivity during the trauma phase.

Control Variables

Table 1 displays the descriptive and inferential statistics for sociodemographic, PTSD diagnosis, and trauma related characteristics based on dissociation group status. ANOVAs revealed no significant differences between dissociation groups on age, general health, or any trauma-related characteristics (all p values $>.05$). However, BMI differed between dissociation groups and was used as a covariate in subsequent analyses, $F(2, 36)=5.54$, $p=.008$. A Tukey's Honestly Significant Difference post hoc test revealed that moderate dissociators ($M=24.36$, $SD=3.6$) had significantly higher BMI compared to low dissociators ($M=20.73$, $SD=2.2$). High dissociators ($M=21.8$, $SD=2.3$) did not differ from either group. Chi-square analyses revealed that dissociation group was not significantly related to race/

ethnicity, medication use, smoking status, caffeine use, menstrual phase, seeking psychological help following the trauma, suffering physical injuries during the trauma, or type of trauma recalled (all p values $>.05$). Results did not change when analyses were run with and without participants on psychiatric medications ($n=2$).

Despite an average of almost 4 years since trauma experience, the current sample reported high rates of PTSD with 51% ($n=20$) of the sample likely meeting current diagnostic criteria for PTSD. A chi-square analysis revealed that moderate and high dissociators were more likely to meet current likely PTSD diagnostic criteria compared to low dissociators, $\chi^2(1, n=39)=6.73, p=.035$. In addition, continuous PDEQ scores were significantly related to the intrusion ($r=.344, p=.032$), avoidance ($r=.422, p=.007$) and hyperarousal subscales ($r=.452, p=.004$) of the PDS.

A RM ANOVA revealed a significant difference in SUDS ratings between phases, $F(1,35)=36.43, p=.001$. A Tukey's Honestly Significant Difference post hoc test revealed that participants rated the trauma recall phase ($M=70.13, SD=14.4$) as being significantly more distressing than the neutral recall phase ($M=47.10, SD=20.82$). Dissociation groups did not differ on their reported SUDS ratings during the trauma recall phase, $F(2, 35)=1.30, p=.286$.

Phase Analyses

Huynh-Feldt statistics are reported for RM ANCOVA analyses due to violations of sphericity ($p<.05$). A series of 3 (low, moderate, high dissociator) X 5 (baseline, trauma recall, rest, neutral recall, recovery) RM ANCOVAs controlling for BMI revealed significant group by phase interactions for HR, $F(4, 68)=4.4, p=.003$, PEP, $F(7, 114)=2.2, p=.047$, and HRV, $F(5, 92)=2.28, p=.05$: see Figure 1). Decomposition of the interaction terms revealed that high dissociators had significantly higher HR, $F(2, 36)=3.75, p=.033$, and lower HRV, $F(2,36)=3.88, p=.030$, compared to moderate dissociators and shorter PEP compared to low dissociators, $F(2,36)=3.6, p=.038$ during the trauma recall phase. There were no group differences on psychophysiological measures during baseline, neutral recall, rest, or recovery (all p values $>.05$).

Reactivity Analyses

A series of one-way ANCOVAs revealed significant group differences on HR, PEP, and HRV reactivity scores during the trauma recall phase, all F values (2,35) >4.0 , all p values $>.05$. Tukey Honestly Significant Difference post hoc analyses revealed that higher dissociators had significantly higher HR reactivity compared to the low and moderate dissociators, greater decreases in PEP compared to the low dissociators, and greater decreases in HRV compared to the moderate dissociators (see Figure 2). Similarly, bivariate correlations revealed that higher levels of dissociation were significantly linearly related to higher HR reactivity ($r=.341, p=.034$) and decreased PEP reactivity ($r=-.422, p=.008$). Dissociation was not significantly linearly related to HRV reactivity ($r=-.195, p=.234$).

Discussion

The present study was designed to examine the relationship between retrospective reports of PD and autonomic reactivity during trauma recall in females who had experienced an interpersonal trauma. Based on prior research, we hypothesized that higher levels of dissociation would be related to lower HR and higher RSA during trauma recall. However, results revealed the opposite pattern. Overall, high dissociators had significantly larger increases in HR and larger decreases in PEP and RSA during trauma recall. Further, HR and

PEP were linearly associated with total PD scores suggesting that the relationship between PD and SNS functioning may lie on a continuum.

To date, this is the first study to examine differences in SNS and PNS reactivity between prior dissociators and nondissociators. Contrary to our predictions, dissociators appeared hyperreactive during trauma recall due to heightened SNS activity (i.e. shortened PEP) coupled with suppressed PNS functioning (i.e. decreased RSA). These findings support research suggesting that individuals may dissociate in response to hyperarousal (Kuhn, et al., 2006; Ladwig, et al., 2002; Nixon, et al., 2005; Sterlini & Bryant, 2002). Further, these results are consistent with physiological studies of PTSD that have shown higher HR reactivity and lower parasympathetic reactivity in response to trauma and non-trauma related stimuli in participants with PTSD (Buckley, Holohan, Greif, Bedard, & Suvak, 2004; Pitman, Orr, & Shalev, 1993; Sack, et al., 2004).

There are several possible explanations for why our results differ from findings suggesting suppressed autonomic functioning among high dissociators including differences in measurement of PD, heterogeneity of the trauma sample, and the length of time between the trauma and laboratory assessment. Measurement and definitions of dissociators have varied across studies. For example, other studies have used different dissociation measures and scoring methods such as median splits (Nixon, et al., 2005), continuous measures (Delahanty, et al., 2003; Halligan, et al., 2006; Koopman, et al., 2004), and interview based measures with a validated cutoff score (Ladwig, et al., 2002; Pole, et al., 2005). The present study used both a continuous and categorical scoring method to determine PD. Similar to Griffin et al. (1997) we used an arbitrary cutoff score of 1SD above the mean to clearly define the high PD group and .5SD below the mean to define a low PD group. In addition, we analyzed the linear relationship between autonomic reactivity and PD using PDEQ total scores. Both scoring methods provided similar results suggesting that increases in dissociation were related to increases in SNS and decreases in PNS functioning.

Research has also differed concerning the type of traumatized sample assessed. Prior studies examining the psychophysiology of dissociation have focused on a specific target trauma such as assault victims (Griffin, et al., 1997; Halligan, et al., 2006), MVA victims (Delahanty, et al., 2003), and survivors of cardiac events (Ladwig, et al., 2002). In the present study we had a heterogeneous trauma sample of participants who were asked to recall their most distressing trauma. It is possible that this heterogeneity contributed to our differing findings.

Another difference between our study and prior research involves the length of time elapsed between the trauma and assessments. For example, Delahanty et al. (2003) obtained physiological samples and reports of dissociation immediately following the trauma, Griffin et al. (1997) interviewed rape victims within two weeks following the trauma and Nixon et al. (2005) interviewed victims within 4 weeks following the trauma. In contrast, the present sample recalled events that occurred from two months to 16 years prior. The longer period between the trauma and laboratory assessment may account for the present findings. By recalling events several years prior, the trauma recall may have elicited different psychological and physiological reactions than the participants would have displayed at the time of the potentially traumatizing event (within-person variability). Similar to Reinders and colleagues (2006), future research examining the physiological correlates of PD should assess experiences of dissociative phenomena and physiological functioning as they co-occur during the laboratory assessment. Alternatively, the hyperarousal elicited in the present study may have been due to the development of chronic PTSD. It is possible that PTSD symptoms may alter the relationship between PD and physiological reactivity. For example, Ladwig et al. (2002) found that dissociators with PTSD demonstrated exaggerated

startle responses (EMG) compared to dissociators without PTSD suggesting that the combination of dissociation and PTSD may result in hyperarousal. However, PTSD diagnosis was not related to physiological variables in the current sample.

Limitations and Future Directions

The present study suffers from several limitations. First, our final sample size was small, and the generalizability of our findings may be limited given that the present sample was predominantly young, Caucasian, college educated females. However, there was still a considerable range of psychopathology underscoring the feasibility of assessing mental health in “convenience” samples. Due to the small sample size, there were a small number of participants that had reported high levels of prior PD. However, the average HR reactivity during trauma recall for these high dissociators was double that of the low and moderate dissociators, suggesting a rather robust relationship. Further, analyses assessing the linear relationship between autonomic functioning and total PD scores supported subgroup analyses.

Another limitation is that reports of PD may have been inaccurate since participants were asked to remember their peritraumatic experiences that occurred years earlier. However, a recent meta-analysis suggested that retrospective reports of PD appear to be reliable over time (Lensvelt-Mulders, et al., 2008). In addition, although not measured, it is possible that differences in speech rate between dissociators and nondissociators may have affected the psychophysiology results; however, there were no differences in respiration rate across dissociation group. Future research should consider script-driven imagery or tasks that do not rely on participants’ speech in order to examine the impact of trauma recall on physiological reactivity. Finally, participants’ physiological reactivity during the actual trauma is unknown. Laboratory paradigms assessing psychophysiological reactivity are not intended to reflect actual responses to trauma. Similarly, the goal of the present study was to determine whether dissociators and nondissociators reacted differently upon recall of a traumatizing event. Differences in reactivity to acute lab paradigms may not generalize to more intense stressful or traumatizing situations.

Finally, a limitation of the present study and of the literature in general, is the lack of a clear definition of PD (Van der Hart, Nijenhuis, Steele, & Brown, 2004; Van der Hart, van Ochten, Van Son, Steele, & Lensvelt-Mulders, 2008). Although the present study used the PDEQ, the most commonly used measure of PD (Lensvelt-Mulders, et al., 2008), it remains unclear whether the items are assessing dissociative phenomena (e.g. dissociative amnesia), altered states of consciousness (e.g. altered time perception), or a combination of both (Van der Hart, et al., 2004). Researchers have suggested including measures of somatoform dissociation in future studies to help clarify the conceptualization of trauma-related dissociation (Lensvelt-Mulders, et al., 2008; Van der Hart, et al., 2008). Further, future research should also include measures of TI due to recent findings suggesting a strong relationship between PD and TI constructs (Abrams, Carleton, Taylor, & Asmundson, 2009).

Despite these limitations, the results of the present study question the idea that peritraumatic dissociators represent a physiologically non-reactive subset of individuals who develop PTSD (Bremner & Brett, 1997; Griffin, et al., 1997; Lanius, et al., 2006), but rather support research suggesting that individuals highly reactive to stress may be more likely to experience PD (Bryant, Harvey, Guthrie, & Moulds, 2000; Nixon, et al., 2005). The present study highlights the need to more closely examine autonomic correlates of PD and their role in the development of PTSD.

Acknowledgments

Preparation of this manuscript was supported by the National Institute of Mental Health (R34MH073014).

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Fig. 1.
Pattern of physiological responding of high, moderate, and low peritraumatic dissociation across the laboratory protocol

Fig. 2.
Pattern of physiological reactivity of high, moderate, and low peritraumatic dissociation during trauma recall

TABLE 1

Sociodemographic, Diagnostic, and Trauma Characteristics of the Sample (n=39)

Variable	Low PD n=11	Moderate PD n=20	High PD n=8	Group Differences		
				χ^2 , F	df	p
Age	18.9 (1.7)	18.75 (.79)	18.5 (.53)	.33	2,36	.723
Race/Ethnicity						
Caucasian	10 (91%)	20 (100%)	7 (88%)	6.54	4	.162
African American	1 (9%)	0	0			
Other	0	0	1 (12%)			
Medication Use (% Yes)						
Psychiatric Meds	0	2 (100%)	0	2.00	2	.367
Birth Control	6 (55%)	4 (20%)	3 (38%)	3.89	2	.143
General Health (n=30)	3.7 (.80)	3.6 (.51)	3.8 (.46)	.20	2,22	.818
Body Mass Index	20.7 (2.2)	24.4 (3.6)	21.8(2.3)	5.5	3, 36	.008*
Smoking status (% Yes)	4 (50%)	4 (50%)	0	3.8	2	.152
Caffeine drinker (% Yes)	4 (21%)	12 (63%)	3 (15%)	2.1	2	.351
Menstrual Phase				4.5	2	.106
Luteal	8 (80%)	11 (55%)	2 (29%)			
Follicular	2 (20%)	9 (45%)	5 (71%)			
Total number of traumas	2.6 (1.5)	2.0 (1.1)	2.6 (1.8)	1.1	3,36	.359
PTSD diagnosis (% Yes)	2 (18%)	13 (65%)	5 (62.5%)	6.7	2	.035*
Recalled Trauma Characteristics						
Feared for life	2.3 (1.5)	3.9 (2.3)	3.5 (3.0)	1.8	3,36	.185
Avoided trauma	2.8 (1.9)	2.9 (1.9)	2.6(1.7)	.04	3,36	.960
Control during trauma	2.5 (1.6)	2.0 (1.4)	1.3 (.70)	1.8	3,36	.180
Age at time of trauma	15.2 (2.5)	14.8 (4.3)	14.3 (6.0)	.10	3,36	.909
Time since trauma (years%)	4.1 (2.5)	4.3 (4.7)	4.6 (6.4)	.03	3,36	.974
mental health therapy (% yes)	1 (9%)	2 (10%)	0 (0%)	4.2	2	.126
SUDDS rating during recall	64.7 (16.33)	73.4 (14.9)	69.4 (6.9)	1.3	2,35	.286

Note: PTSD = posttraumatic stress disorder; frequency (%) is reported for categorical variables and mean (standard deviation) is reported for continuous variables

$p < .05$
*

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TABLE 2

Type of Trauma Recalled

	Frequency (%)
Sexual Assault	11 (28.2)
Physical Assault	8 (20.5)
Mugging/Robbery	1 (2.6)
Death/illness of loved one	4 (10.3)
Serious illness/accident	6 (15.4)
Other	9 (23.1)

Note: Other traumas included verbal abuse/harassment, witnessing a death, witnessing physical assault and natural disaster