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Reappraising the Link Between Peritraumatic Dissociation and PTSD Symptom Severity: Evidence From a Longitudinal Study of Community Violence Survivors

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RAND

Cross-lagged panel analysis of longitudinal data collected from young adult survivors of community violence was used to examine the relationship between recall of peritraumatic dissociation and posttraumatic stress disorder (PTSD) symptom severity. Recollections of peritraumatic dissociation assessed within days of exposure differed from recollections measured at 3- and 12-month follow-up interviews. Peritraumatic dissociation was highly correlated with PTSD symptoms within each wave of data collection. Baseline recollections of peritraumatic dissociation were not predictive of follow-up PTSD symptom severity after controlling for baseline PTSD symptom severity. This pattern of results replicates previous work demonstrating a correlation between peritraumatic dissociation and subsequent symptom severity. However, findings are not consistent with the prevailing view that peritraumatic dissociation leads to increased PTSD symptom severity.

Why do some people seem adept at negotiating life’s hardships whereas others do not? The psychological aftermath of exposure to traumatic life experiences has long been recognized as highly variable, with some persons adjusting well—albeit often with marked initial distress—and others incurring significant adverse emotional and psychological consequences of considerable duration. Contemporary models of the course of adjustment to trauma include an array of personal, social, and environmental factors that may confer heightened risk or resilience for psychopathology (Davis & Breslau, 1994; Foy, Osato, Houskamp, & Neumann, 1992; Green, 1994; McFarlane, 2000). Although adjustment to trauma is likely to be multiply determined, in recent years growing interest has been directed at dissociative experiences that take place during a traumatic event as a predictor of future adjustment.

So-called peritraumatic dissociation appears to foreshadow subsequent maladaptation to psychological trauma (van der Kolk & van der Hart, 1989). In particular, peritraumatic dissociation has been linked to the emergence of posttraumatic stress disorder (PTSD) and PTSD symptom severity in various traumatized samples, including combat veterans (Marmar et al., 1994; O’Toole, Marshall, Schureck, & Dobson, 1999; Tichenor, Marmar, Weiss, Metzler, & Ronfeldt, 1996), natural disaster survivors (Koopman, Classen, & Spiegel, 1994), emergency services personnel (Marmar, Weiss, Metzler, Ronfeldt, & Foreman, 1996), victims of motor vehicle crashes (Ursano et al., 1999), and physical trauma survivors (Alexander, Kemp, Klein, & Forrester, 2001; Michaels, Michaels, Moon, et al., 1999; Shalev, Peri, Canetti, & Schreiber, 1996). For example, in their prospective study of 51 persons hospitalized for injuries stemming from physical trauma, Shalev et al. (1996) reported that peritraumatic dissociation was independently associated with subsequent severity of PTSD symptoms ($\beta = 0.49$), even after adjusting for other predictors.

Theoretical accounts of dissociation, which can be traced to Janet’s (1907) writings on the topic, emphasize its utility as a means of coping with traumatic experiences. In essence, dissociation serves to protect individuals from experiencing highly aversive emotions that may occur during or immediately after a traumatic event (e.g., van der Kolk, 1987). Thus, experiencing a sense of detachment from oneself, a perception that one’s surroundings are unreal, altered pain perception, and similar dissociative phenomena divert conscious attention away from even more threatening feelings of helplessness and horror. The benefits of diverted or restricted awareness are short lasting, however, as trauma theorists have posited that peritraumatic dissociation potentiates PTSD symptoms by interfering with normal information processing (van der Kolk & Fisler, 1995; van der Kolk & van der Hart, 1989).

The construct of peritraumatic dissociation offers the promise of yielding important insights into the etiology of PTSD and may serve as a key marker of increased risk for posttrauma maladjustment. Yet, several issues require further examination. First, much research examining peritraumatic dissociation has used cross-sectional designs using retrospective self-reports of dissociative experiences. In such instances, participants are typically asked to recall intrapsychic experiences that took place months—or even years—after the traumatic event had occurred (e.g., Johnson, Pike,
loss of personal autonomy were also assessed within days of exposure. Dissociative symptoms emerged as a significant predictor of PTSD symptom severity assessed 7–9 months after exposure, even after adjusting for the impact of other predictor variables. Notably, however, PTSD symptom severity was not assessed at baseline, precluding determination of whether dissociation would have predicted subsequent PTSD symptom severity independently of initial symptom severity. In short, a more definitive examination of the causal relationship between peritraumatic dissociation and PTSD symptom severity would use a cross-lagged panel design in which measures of both constructs are assessed with synchronicity at multiple points in time (Kenny, 1979; Kessler & Greenberg, 1981).

The broad aim of the current study was to examine the relationship between symptoms of PTSD and recollections of peritraumatic dissociative experiences in a sample of survivors of community violence who had been hospitalized for treatment of physical injuries. Community violence exacts an enormous societal toll (Cook, Lawrence, Ludwig, & Miller, 1999) and individual toll (e.g., Brewin, Andrews, Rose, & Kirk, 1999; Davis & Breslau, 1994; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993). By way of illustration, a recent study of crime survivors—the majority of whom were not physically injured—reported that 20% of participants met criteria for PTSD 6 months following victimization (Brewin et al., 1999). Yet, comparatively little research has examined the impact of community violence resulting in physical injury on subsequent psychopathology and, to our knowledge, no prior research has evaluated the link between peritraumatic dissociation and subsequent symptoms of PTSD in this population.

In assessing the relationship between peritraumatic dissociation and symptoms of PTSD, data were collected within days of the assault, at 3-month follow-up, and at 12-month follow-up. The specific goals were to evaluate alternative models of the relationship between peritraumatic dissociation and symptoms of PTSD, using a cross-lagged panel design. This design allows the investigation of a variety of causal models relating these two constructs, including the possibilities that (a) peritraumatic dissociation may cause subsequent PTSD symptom severity, (b) initial PTSD symptom severity may influence subsequent recall of peritraumatic dissociation, (c) a reciprocal influence exists in which initial levels of each construct may influence subsequent levels of the other, (d) the association between the two constructs may be primarily attributable to covariation within assessment waves.

To address these research aims, we also included baseline measures of injury severity and neuroticism in our models. Prior research has evaluated the link between peritraumatic dissociation and PTSD symptom severity only at follow-up (e.g., Michaels, Michaels, Moon, et al., 1999a; Shalev et al., 1996; Ursano et al., 1999). Using this approach, it is not possible to determine the extent to which dissociation at baseline actually causes the variability in subsequent PTSD symptom severity. For example, if peritraumatic dissociation and baseline levels of PTSD-related distress share a common cause, then the two constructs would be correlated at baseline even without any causal link between them. To the extent that PTSD symptom severity remains stable over time, baseline dissociation would be expected to predict future PTSD symptoms even without any causal relation.

In perhaps the most rigorous study of this type, Koopman et al. (1994) examined the relationship between dissociative experiences reported in the immediate aftermath of exposure to a fire and subsequent PTSD symptom severity. Symptoms of anxiety and loss of personal autonomy were also assessed within days of exposure. Dissociative symptoms emerged as a significant predictor of PTSD symptom severity assessed 7–9 months after exposure, even after adjusting for the impact of other predictor variables. Notably, however, PTSD symptom severity was not assessed at baseline, precluding determination of whether dissociation would have predicted subsequent PTSD symptom severity independently of initial symptom severity. In short, a more definitive examination of the causal relationship between peritraumatic dissociation and PTSD symptom severity would use a cross-lagged panel design in which measures of both constructs are assessed with synchronicity at multiple points in time (Kenny, 1979; Kessler & Greenberg, 1981).

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research has implicated neuroticism—the disposition to experience negative moods and cognitions—as a predictor of both peritraumatic dissociation (e.g., Marmar, Weiss, Metzler, & Delucci, 1996) and PTSD symptoms following trauma exposure (e.g., Fauerbach, Lawrence, Schmidt, Munster, & Costa, 2000). Although empirical research linking physical injury severity to peritraumatic dissociation and PTSD symptom severity is equivocal (e.g., Fullerton et al., 2000; Jeavons, 2000; Michaels, Michaels, Zimmerman, et al., 1999; Shalev et al., 1996), we elected to include injury severity as a possible source of covariation between peritraumatic dissociation and PTSD symptom severity.

Method

Participants and Procedure

Data were collected as part of a larger NIMH-funded study focusing expressly on the mental health consequences of young adult exposure to community violence resulting in significant physical injury. Although injuries stemming from other causes—including domestic violence—are important topics of study in their own right, comparatively little research has examined the psychological impact of injuries attributable to community violence. Because national studies concur in finding that victims of community violence tend disproportionately to be young men of minority descent (e.g., Annest, Mercy, Gibson, & Ryan, 1995), participants were young adults between the ages of 18 and 35. All participants were recruited following admission to a large Level I trauma facility for treatment of physical injuries stemming from community violence.

Between October 1998 and June 2000, the research staff attempted to screen for eligibility all consecutively hospitalized persons between 18 and 35 who were hospitalized for treatment of blunt or penetrating trauma. Persons eligible for the study had sustained an injury inflicted by a person other than a family member or a former intimate sexual partner. Individuals whose injuries stemmed from motor vehicle crashes, accidents, domestic violence, and other incidents not attributable to community violence were ineligible. Participants were required to communicate fluently in either English or Spanish. Participants were approached to complete a short interview to screen for eligibility only after they were judged medically capable of being interviewed as determined by discussions with medical staff. Multiple attempts were made to monitor, screen, and interview persons who were either not initially available or who appeared to be cognitively impaired or insufficiently alert. Respondents were offered token compensation of $25 for each interview in which they participated.

Of 653 persons screened, 423 were eligible for the study. Of these, 413 (98%) completed an interview. Only 2 eligible persons declined to participate; 5 participants chose to terminate the interview prior to completion. A total of 230 persons were determined to be ineligible for the study. An additional 584 persons could not be screened for eligibility. The primary reason for failure to screen was discharge before an approach could be made. Finally, 5 persons chose not to participate in a screening interview. In all, approximately 60% of age-eligible persons were screened.

The majority of respondents were male (94%). Participants averaged 24.3 years of age (SD = 5.60). With respect to race/ethnicity, 78% self-identified as Hispanic, 12% as Black, 3% as non-Hispanic Caucasian, 3% as Asian, and 4% as Native American, multiracial, or other. Sixty percent of participants had not completed high school or its equivalent; 36% had completed high school or its equivalent, and less than 4% had attended some college. With respect to income, 35% reported receiving less than $500 of pretax income in the 30 days prior to their assault, 26% had received between $500 and $1,000, and 18% reported receiving between $1,000 and $1,500; the remainder reported having received more than $1,500. Sixty-two percent of the sample had never been married, 30% were either married or cohabiting, and 7% were separated or divorced. Fifty-nine percent had sustained injuries stemming from gunshot, and the remaining 41% had received injuries from other penetrating or blunt objects.

All participants completed a face-to-face structured interview conducted by trained lay interviewers within several days of hospital admission (M = 9.55 days, Min = 1, mode = 1, SD = 9.84). Participants also completed face-to-face interviews again at 3 months and 12 months after hospital admission. Of the 386 participants with complete data at baseline, 275 (71.2%) had complete data from the 3-month interview and 250 (64.8%) had complete data from the 12-month interview. The pattern of participant attrition was not significantly related to any of the measures used in the current study, as determined by chi-square tests for categorical variables and t tests for continuous variables.

Measures

Data on injury mechanism, injury site, injury severity, and degree of consciousness within 24 hr of hospital admission were extracted from computerized medical records. All other data—including measures of PTSD symptom severity and peritraumatic dissociation—were obtained by means of face-to-face structured interviews conducted by a highly trained team of lay interviewers. All psychological measures were included at all waves of data collection.

Level of consciousness. Level of consciousness was assessing using Glasgow Coma Score (GCS) data obtained from computerized medical records (Teasdale & Jennett, 1974). GCS ratings were made within 24 hr of admission. GCS scores range from 3 to 15, with lower scores reflecting greater cognitive impairment. In our sample, most respondents were fully alert at testing (M = 14.67; SD = 1.30). Eighty-eight percent of participants received a GCS of 15, reflective of no detectable impairment; 7% received a score of 14; and 5% received a score lower than 14.

Participants were not administered a formal mental status examination to screen for cognitive impairment. Nonetheless, the eligibility screener and survey afforded interviewers the opportunity to observe respondents’ answers to a range of questions. These included questions that could be independently verified by the interviewer, using hospital records. Although we acknowledge that this process is not a substitute for a mental status exam, it did enable interviewers to make judgments about respondent lucidity. Interviewers were trained to recognize some signs of compromised mental acuity and to postpone contact to a later date in instances in which they judged that the validity of a completed interview would be compromised.

We used lay interviewers—rather than relying on self-administered questionnaires—to reduce respondent burden, maximize data quality, and enhance participant motivation. On the basis of our experience with this population and other difficult-to-assess populations (e.g., Marshall, Burnham, Koegel, Sullivan, & Benjamín, 1996), we have found lay-administered structured interviews to be superior to self-administered questionnaires in obtaining complete data of high quality.

All interviewers received at least 8 full days of training prior to conducting interviews and were required to successfully complete a series of mock interviews before being allowed to interview study participants. Ongoing training and supervision were also provided throughout the study. The interview was fully structured, providing interviewers with little or no discretion in coding responses. In essence, interviewers were restricted to reading aloud questions, which would otherwise have been read by respondents themselves, and noting respondents’ answers.

Researchers have become increasingly attentive to the possibility that dissociative experiences can follow from medical conditions in general, and head injury in particular (e.g., Harvey & Bryant, 1998). In our study, only 12% of participants had received an injury to the head or neck. We conducted a sensitivity analysis to determine whether study findings were influenced by the presence of any detected impairment in consciousness within 24 hr of admission, irrespective of injury site. Reanalysis after removal of data from participants with GCS scores less than 15 resulted in a virtually identical pattern of findings.
**Peritraumatic dissociation.** Peritraumatic dissociation was assessed using a modified version of the PDEQ (Marshall et al., 2002) of the self-administered form of the Peritraumatic Dissociative Experiences Questionnaire (PDEQ; Marmar, Weiss, & Metzler, 1997). The original self-administered version of the PDEQ consists of 10 items measuring the extent to which participants remember specific dissociative phenomena that occurred during or immediately after trauma exposure. Each PDEQ item consists of a statement indicating a dissociative experience and a 5-point response scale anchored by 1 (not at all true) and 5 (extremely true).

A modified 7-item version of the PDEQ was developed and validated for the purposes of this study after our pilot interviews revealed that the original PDEQ appeared ill suited for administration to a largely minority, less-educated sample of modest means. With few exceptions (e.g., Griffin, Resick, & Mechanic, 1997), use of the PDEQ has been limited to samples composed primarily of middle-class Caucasians with comparatively high educational achievement (e.g., Fullerton et al., 2000; Marmar, Weiss, Metzler, Ronfeldt, & Foreman, 1996; O'Toole et al., 1999). As described in Marshall et al. (2002), modifications to the PDEQ were limited to reducing item length, clarifying the wording of items that were poorly understood or foreign to the cultural experiences of this sample, and omitting 3 items. Two items were dropped because of psychometric considerations. An additional omitted item, which referred to feeling trapped in the presence of others, was dropped because respondents did not regard it as germane. Notably, the latter item has been dropped by other researchers on an ad hoc basis (e.g., Griffin et al., 1997; Shalev et al., 1996).

An initial validation study of the modified instrument was conducted with a sample of participants in the core study (n = 284), demonstrating that the revised instrument possessed good internal consistency reliability and divergent–convergent validity. The reliability, validity, and generalizability of the modified PDEQ was further documented in a second validation study of an independent sample of relatively well-educated, female survivors of sexual assault (N = 90). Of particular note, the magnitude of the correlation between the revised and original versions of the PDEQ in the latter was sample strong (r = .89), demonstrating that the two instruments assess highly similar constructs. Moreover, in the latter study, the pattern of relations of the two scales to external criteria was essentially identical (Marshall, Orlando, Jaycox, Foy, & Belzberg, 2002). Additional research using the modified version attests to its construct validity (Marshall & Orlando, 2002). Descriptive statistics for the PDEQ at each wave of data collection are displayed in Table 1.

**PTSD symptom severity.** PTSD symptom severity was assessed using the 17-item PTSD Checklist (PCL, Civilian Version; Weathers, Litz, Herman, Huska, & Keane, 1993). Respondents were instructed to indicate the degree to which they had been bothered by each symptom, using a 5-point scale ranging from 1 (not at all) to 5 (extremely). The PCL was designed for use with multiple time frames, depending on research needs (Weathers et al., 1993). At the initial interview, respondents were asked to respond with respect to symptoms experienced since the attack. At follow-up interviews, respondents were asked to respond with respect to the previous 7 days. We opted for a 7-day response framework for subsequent waves of data collection to optimize the comparability of baseline and follow-up assessments. The instructional set at each wave made specific mention of our interest in symptoms related to the focal attack. Responses at each wave of data collection were answered with respect to the specific assault in question (e.g., “how much have you been bothered by repeated, disturbing dreams of the attack,” “avoiding thinking or talking about the attack or avoiding having feelings related to it”).

The PCL has been used in various traumatized populations and has been found to possess solid psychometric properties and construct validity in diverse samples (e.g., Andrykowski, Cordova, Studts, & Miller, 1998; Asmundson et al., 2000; Blanchard, Jones-Alexander, Buckle, & Forneris, 1996; Gallagher, Riggs, Byrne, & Weathers, 1998). Previous analyses of data collected at baseline indicated that the scale is well fit by a confirmatory factor analytic model in which a single global domain reflecting overall PTSD symptoms subsumes 4 first-order factors corresponding to the dimensions of reexperiencing, avoidance, emotional numbing, and hyperarousal (Marshall, 2002). For analytic purposes, we summed the scale items within each symptom cluster to produce four symptom scores at each administration of the checklist. Descriptive statistics for the PCL, derived by summing across all 17 items at each wave of data collection are displayed in Table 1.

**Neuroticism.** Dispositional neuroticism, the tendency to experience negative moods and cognitions, was assessed using 5 items drawn from the 12-item Neuroticism scale of the NEO Five-Factor Inventory (Costa & McCrae, 1989). Each item is phrased as a statement, with respondents indicating extent of agreement on a 5-point scale ranging from 1 (strongly disagree) to 5 (strongly agree). The full set of items was not included due to respondent burden considerations. The 5 items were averaged to form a single scale (M = 2.72, SD = 0.84), with an internal consistency reliability estimate of .79. To help ensure that participant responses could be interpreted as pretrauma neuroticism, respondents were asked to provide answers with respect to “what you were generally like as a person before the attack, not how you have been feeling since the attack.” Further support for the view that this measure may reflect pretrauma neuroticism comes from a recent review of the literature that suggests substantial temporal stability in neuroticism scores (McCrae & Costa, 1994).

**Data Analysis.**

**Overview.** Covariance structure modeling, using the EQS software program (Bentler, 1995), constituted the primary method of data analysis. For the current purposes, we combined data from English- and Spanish-speaking participants. The measurement equivalence of English and Spanish versions of all instruments reported herein has been demonstrated in previous analyses using either covariance structure modeling or item

### Table 1

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Baseline</th>
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<tr>
<td></td>
<td>N</td>
<td>406</td>
<td>291</td>
</tr>
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</table>

**Note.** Scores were derived by summing across PCL and PDEQ items. PCL = Posttraumatic Symptom Checklist; PDEQ = Posttraumatic Dissociative Experiences Questionnaire.
response theory methods (Marshall & Orlando, 2002; Marshall, Orlando, Jaycox, Foy, & Belzberg, 2002; Orlando & Marshall, 2002; Updegraff & Marshall, 2002). Because our central interest is in the stability or change in initial recall of peritraumatic dissociation and its association with PTSD symptoms, we chose to examine both 3-month and 12-month assessment data with reference to the baseline assessment. In other words, we did not model data from all three waves simultaneously. Fitting a three-panel cross-lagged model would have required more parameters than could be reliably estimated from a sample of this size. Finally, because the majority of previous studies have required participants to recall dissociative experiences after an elapsed time of multiple months to years, we conducted primary model development on the baseline to 12-month data. These models were also fit to the 3-month data as a replication of the primary findings.

Following the two-step procedure advocated by Anderson and Gerbing (1988; see also Hays, Marshall, Wang, & Sherbourne, 1994), the measurement and structural portions of various models were examined in successive stages. In building multivariate models, we allowed for correlated error terms involving analogous items over time. Modeling errors in this way assumes that the sources of measurement error in individual questionnaire items may be stable over time. In addition, because invariance of factor loadings over time is an important criterion for meaningful comparisons in longitudinal data (Hoyle & Smith, 1994), we constrained analogous factor loadings to be equivalent across measurement occasions. We also evaluated the fit of more restrictive models of factorial invariance. In these models, the magnitude of each indicator’s residual was constrained to be equal over time. These models resulted in significantly worse fits ($p < .05$) for both PTSD symptoms and peritraumatic dissociation. It appears that the variances of the residuals are not constant over time for either the four indicators of PTSD or the seven indicators of peritraumatic dissociation. Thus, cross-time equality constraints imposed on residual variances for analogous variables were relaxed for all subsequent analyses.

Finally, model fit was evaluated using three common indices. These included the root-mean-square-error of approximation (RMSEA; Steiger, 1990), the Bentler–Bonett nonnormed fit index (NNFI; Bentler & Bonett, 1980), and the comparative fit index (CFI; Bentler, 1990). RMSEA values less than .05 are generally regarded as indicative of good model fit (Browne & Cudeck, 1993), whereas values exceeding .05 are indicative of good model fit as assessed by the NNFI and the CFI (Hu & Bentler, 1998). Chi-square difference tests and the consistent Akaike’s information criterion (CAIC; Bozdogan, 1987) were also used to evaluate the relative fit of alternative models.

**Results**

**Descriptive Analyses**

For descriptive—but not analytic—purposes, scores for PTSD symptom severity and peritraumatic dissociation were computed by summing across the 17 PTSD PCL items and the 7 PDEQ items to create separate PTSD and PDEQ indexes. The means and standard deviations for these measures are shown in Table 1 for the three interviews and all respondents. As would be expected, for the group of respondents with PTSD symptom severity data at baseline as well as follow-up assessments, there was a slight decrease in reported symptoms over time, with a 4.15-point drop from baseline to 3 months, $t(289) = 5.59, p < .001$, and a 1.68-point drop from the 3-month to the 12-month point, $t(226) = 2.15, p < .05$. For participants with peritraumatic dissociation data at both baseline and follow-up, there were no significant changes for the overall group in average level of recalled peritraumatic dissociation over time.

The intercorrelations among the measures of PTSD and peritraumatic dissociation, without disattenuation for measurement error, are presented in Table 2. Correlations among the three measures of PTSD were quite high, ranging from .49 to .67. Correlations among the measures of peritraumatic dissociation were similarly high, ranging from .46 to .58. Correlations across different measures given in the same interview (i.e., within wave) were nearly as large as the correlations within measures over time, ranging from .46 to .51. Of particular relevance, baseline peritraumatic dissociation was significantly correlated with subsequent PTSD symptom severity, thus replicating previous research findings (e.g., Michaels et al., 1999; Shalev et al., 1996; Ursano et al., 1999).

**Cross-Lagged Analyses**

A conventional cross-lagged panel analysis was conducted, with latent variables representing peritraumatic dissociation and PTSD symptoms repeated at two times (baseline and follow-up). Latent variables were identified by fixing the factor loading of a single item on each factor to 1.0. As noted previously, four subscale scores were used as measured indicators of PTSD symptom severity, whereas the seven individual items assessing peritraumatic dissociation served as indicators of the latter construct. In addition, baseline measures of neuroticism and objective injury severity were included as potential causes of both peritraumatic dissociation and PTSD symptom severity. The latter two variables were treated as measured, rather than latent, to reduce the number of parameters estimated in the model. As noted previously, two separate cross-lagged models were fit. The first model focused on the relationship between baseline and 12-month follow-up data,

<table>
<thead>
<tr>
<th>Instrument</th>
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<tr>
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<tr>
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<td>19.9</td>
<td>37.8</td>
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<td>19.1</td>
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<tr>
<td>3 months</td>
<td>.54</td>
<td>53.6</td>
<td>31.4</td>
<td>40.2</td>
<td>53.8</td>
<td>45.8</td>
</tr>
<tr>
<td>12 months</td>
<td>.46</td>
<td>.58</td>
<td>52.0</td>
<td>34.7</td>
<td>47.5</td>
<td>52.2</td>
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<tr>
<td>Baseline</td>
<td>.46</td>
<td>.40</td>
<td>.34</td>
<td>190.1</td>
<td>117.2</td>
<td>99.2</td>
</tr>
<tr>
<td>3 months</td>
<td>.34</td>
<td>.51</td>
<td>.45</td>
<td>.60</td>
<td>205.6</td>
<td>139.8</td>
</tr>
<tr>
<td>12 months</td>
<td>.22</td>
<td>.44</td>
<td>.50</td>
<td>.49</td>
<td>.67</td>
<td>213.2</td>
</tr>
</tbody>
</table>

Note. Covariances are in the upper triangle; variances on the diagonal are in boldface; correlations are in the lower triangle. Statistics are based on complete pairs; average pairwise $N = 305$. PDEQ = Posttraumatic Dissociative Experiences Questionnaire; PCL = Posttraumatic Symptom Checklist.
whereas the second examined the relationship between baseline and 3-month follow-up data.

One should note that our cross-lagged panel model differs from typical models insofar as the path from baseline to follow-up peritraumatic dissociation is a measure of the test–retest reliability of this measure. In other words, at all waves of data collection, respondents were asked to recall memories of their intrapsychic experience for the same traumatic event. Although somewhat unusual, this strategy is central to addressing key questions about the stability of recall over time and has been used in other recent cross-lagged panel studies investigating the interplay of memory for traumatic events and distress over time (e.g., King et al., 2000). Following convention, the path from baseline to follow-up PTSD symptom severity is an autoregressive path reflecting the stability of the phenomena over time.

Analyses on the baseline data demonstrated that neuroticism and injury severity were related to both PTSD symptoms and recall of peritraumatic dissociation, although neuroticism and injury severity are not significantly correlated (Jaycox, Marshall, & Orlando, 2002). These two context variables were included in the model as covariates because they may partially explain the observed relationship between PTSD symptoms and peritraumatic dissociation. It was not hypothesized a priori whether these variables would have a direct effect on peritraumatic dissociation and PTSD symptom severity at follow-up or whether their effects at follow-up would be mediated through baseline levels of peritraumatic dissociation and PTSD symptom severity. Models with direct paths from both neuroticism and injury severity to the follow-up measures of peritraumatic dissociation and PTSD symptom severity were tested. Models with direct paths did not have significantly better fits than those without: \( \Delta \chi^2(4) = 3.55, p > .10, \) for the 12-month follow-up models, and \( \Delta \chi^2(4) = 7.72, p > .10, \) for 3-month follow-up models. Therefore, subsequent model testing incorporated paths from neuroticism and injury severity to baseline measures of peritraumatic dissociation and PTSD symptom severity but not to follow-up measures.

A saturated model was constructed for an initial investigation of the cross-lagged components. In this model, the four possible paths from the baseline to follow-up measures of peritraumatic dissociation and PTSD symptom severity were estimated. The correlations between the residuals of these latent variables at baseline and at follow-up were also estimated. A series of more restrictive nested models were then tested in a sequential manner to investigate more precise hypotheses about the causal relationship between peritraumatic dissociation and PTSD symptom severity.

For the 12-month follow-up data, the full model fit acceptably well: \( \chi^2(242) = 336.55, p < .001, \) RMSEA = .040, 90% CI = .03—.05, NNFI = .95, CFI = .95 (see Figure 1). This finding of good fit was replicated in the 3-month follow-up model, \( \chi^2(242) = 305.89, p < .01, \) RMSEA = .031, 90% CI = .02—.04, NNFI = .97, CFI = .97.

As shown in Figure 1, the impact of baseline PTSD symptom severity and peritraumatic dissociation on their follow-up counterparts was large and statistically significant. Similarly, correlated residuals between PTSD symptom severity and peritraumatic dissociation within each assessment wave were also large and statistically significant. By contrast, cross-lagged paths involving PTSD symptom severity and subsequent recall of peritraumatic dissociation, as well as peritraumatic dissociation and subsequent PTSD symptom severity, were small and nonsignificant.

A common explanation for the within-wave correlation between PTSD symptoms and recall measures of peritraumatic dissociation is that dissociation at the time of trauma is one of the causes of PTSD symptom severity. A strict modeling interpretation of this explanation is that the correlation between PTSD symptoms and recall measures of peritraumatic dissociation at follow-up results entirely from sharing the common causes measured at baseline. This possibility can be tested by removing the correlation between

![Figure 1](image-url)

**Figure 1.** Standardized regression coefficients for the initial 12-month model, with 3-month coefficients in parentheses (measurement model omitted). PTSD = posttraumatic stress disorder. * \( p < .01; \) ** \( p < .001; \) unmarked \( p > .20. \)
the residuals of PTSD symptoms and peritraumatic dissociation in the follow-up panel while retaining all of the paths from the baseline to the follow-up latent variables. This respecification resulted in a significantly worse fit for both models: $\Delta \chi^2(1) = 45.10, p < .001$, for the 12-month follow-up, and $\Delta \chi^2(1) = 36.16, p < .001$, for the 3-month follow-up. Thus, the relationship between peritraumatic dissociation and PTSD severity at follow-up cannot be fully explained by sharing the common causes of baseline peritraumatic dissociation and PTSD symptoms.

A less strict interpretation is that some portion of the correlation between current PTSD symptoms and recall measures of peritraumatic dissociation occurs because they are both caused by dissociation at the time of trauma. This interpretation is plausible only if (a) the reliability path from baseline peritraumatic dissociation to follow-up peritraumatic dissociation and (b) the cross-lagged path from baseline peritraumatic dissociation to follow-up PTSD symptom severity are both nonzero. Models fixing the peritraumatic dissociation reliability path to zero fit significantly worse than the initial model: $\Delta \chi^2(1) = 25.81, p < .001$, for the 12-month follow up, and $\Delta \chi^2(1) = 35.31, p < .001$, for the 3-month follow-up. However, models eliminating the cross-lagged path from peritraumatic dissociation to PTSD symptom severity did not fit significantly worse than the initial model: $\Delta \chi^2(1) = .54, p > .20$, for the 12-month follow up, and $\Delta \chi^2(1) = 1.4, p > .50$, for the 3-month follow-up.

A similar analysis can be performed to determine if some portion of the correlation between follow-up PTSD symptoms and recall measures of peritraumatic dissociation occurs because they are both caused by PTSD symptoms at baseline. This would be an unusual interpretation of the correlation because it assumes that psychological distress near the time of trauma creates memories of dissociation at some point in the future. This interpretation is plausible only if (a) the autoregressive path from baseline PTSD symptoms to follow-up PTSD symptom severity and (b) the cross-lagged path from baseline PTSD severity to follow-up peritraumatic dissociation are both nonzero. Models fixing the PTSD autoregressive path to zero fit significantly worse than the initial model: $\Delta \chi^2(1) = 30.74, p < .001$, for the 12-month follow up, and $\Delta \chi^2(1) = 46.45, p < .001$, for the 3-month follow-up. However, models eliminating the cross-lagged path from peritraumatic dissociation to PTSD did not fit significantly worse than the initial model: $\Delta \chi^2(1) = .536, p > .20$, for the 12-month follow up, and $\Delta \chi^2(1) = 1.12, p > .20$, for the 3-month follow-up.

Putting these results together, we find that both cross-lagged paths can be set to zero without a significant degradation in model fit: $\Delta \chi^2(2) = .586, p > .50$, for the 12-month follow up, and $\Delta \chi^2(2) = 1.53, p > .20$, for the 3-month follow-up. In fact, these reduced models are more parsimonious than the initial models.

In this final model (see Figure 2), the correlation between PTSD symptom severity and peritraumatic dissociation at follow-up, residualized for autoregressive effects, was .54 for the 12-month follow-up and .50 for the 3-month follow up. In other words, changes in respondents’ PTSD symptoms over time were very highly correlated with changes in their memories of peritraumatic dissociation. The strength of this relationship ($r^2 = .29$) was of approximately the same magnitude as the test–retest reliability of memories of peritraumatic dissociation from the baseline to 12-month follow-up ($r^2 = .34$).

**Figure 2.** Standardized regression coefficients for the final 12-month model, with 3-month coefficients in parentheses (measurement model omitted). PTSD = posttraumatic stress disorder. * $p < .01$; ** $p < .001$. 

**Discussion**

This study used covariance structure modeling, in the context of cross-lagged panel analysis, to examine the relationship between peritraumatic dissociation and PTSD symptom severity in a sample of young adult survivors of community violence. To our knowledge, this research constituted the first such examination. Two separate sets of models were developed. The first set of models examined the relationship between baseline and 12-month...
follow-up data, whereas the second examined the replicability of these findings in models linking baseline with 3-month follow-up assessment.

Several key findings emerged that were substantially similar in both models. First, subsequent memory for peritraumatic dissociation, assessed at 3 and 12 months following initial interview, was often not concordant with recollections made within days of trauma exposure. Although the average level of peritraumatic dissociation for the group stayed relatively constant over time in this sample, individual respondents’ recall of peritraumatic dissociative experiences within a few days of trauma differed markedly from recall at both subsequent assessments. The latter finding is consistent with research on memory for intrapsychic experiences (e.g., Henry et al., 1994), suggesting that recall of peritraumatic dissociative experiences that took place months following exposure may not constitute veridical assessments of an individual’s actual dissociation at the time of trauma. To the extent that these results provide an accurate picture, research findings predicated on the temporal stability of recalled peritraumatic dissociation should be reconsidered (e.g., Alexander et al., 2001; Johnson et al., 2001; Marmar et al., 1994; Marmar et al., 1996; O’Toole et al., 1999; Tichenor et al., 1996).

A second key finding is that peritraumatic dissociation at baseline did not emerge as an independent predictor of subsequent symptoms of PTSD as assessed at either follow-up wave. Although we were able to replicate the basic finding of a significant correlation between baseline dissociation and subsequent PTSD symptom severity, this relationship did not hold after controlling for initial PTSD symptom severity. Thus, these findings raise questions about the predictive utility of peritraumatic dissociation and run counter to the prevailing view of peritraumatic dissociation as an independent marker of persons at high risk of subsequent traumagenic distress (McFarlane, 2000). Stated differently, we found no evidence that peritraumatic dissociation served as a harbinger of latter PTSD symptom severity. Conversely, we also found no evidence that PTSD symptom severity contributed to subsequent recall of peritraumatic dissociation. That is, initial symptom severity did not serve as a significant predictor of follow-up recall of dissociation after adjusting for initial recall.

In short, baseline measures of PTSD symptom severity and peritraumatic dissociation were strongly correlated even after controlling for injury severity and pretrauma neuroticism. Moreover, follow-up measures of PTSD symptom severity and peritraumatic dissociation were strongly correlated, even after controlling for their baseline counterparts. In fact, these correlations were virtually as strong at the 3- and 12-month follow-ups as they were at the initial assessment that took place within days of trauma exposure. In essence, although baseline peritraumatic dissociation does not help to predict follow-up PTSD symptoms, a very strong relationship emerged between changes in PTSD-related distress and changes in memory for peritraumatic dissociation.

There are several possible explanations of the observed pattern of findings, each of which has important implications for the conceptualization of PTSD. First, both memories of peritraumatic dissociation and PTSD symptom severity might be caused by an unmeasured third variable that varies substantially over time. One such variable could be general psychological distress. Inasmuch as general distress is often indexed by neuroticism—a construct that was found to play little role as a predictor of either dissociation or PTSD symptoms—our findings provide few clues as to the nature of a potential third variable. Future research is needed to investigate factors that may be causing both PTSD-related distress and memories of peritraumatic dissociation to vary over time.

It is also possible that believing that one’s traumatic experience was accompanied by dissociation is one facet of the phenomenology of PTSD. This position would imply that the memory of a peritraumatic experience should be thought of as a separate symptom cluster of PTSD. As explicated elsewhere, this possibility is consistent with the position that dissociation is a component of PTSD (e.g., Brett, 1993; van der Kolk et al., 1996). This view does not necessarily hold that peritraumatic dissociation causes PTSD symptoms. Rather, dissociation is viewed as another manifestation of the underlying disturbance, along with reexperiencing, avoidance, emotional numbing, and hyperarousal. To the degree that this position vis-à-vis dissociation is accurate, a fundamental shift in the conceptualization of PTSD is required. Additional research is essential to assess the merits of such a reformulation.

Another possible interpretation of the observed results posits that the causal connection between peritraumatic dissociation and PTSD symptomatology is dynamic in a way that cannot be detected in a conventional, cross-lagged panel design. In a conventional causal model, the level of one variable at a given time point determines the level of another variable at some later time (e.g., high levels of dissociation lead to high distress). However, it is possible that changes in one construct over time, regardless of the level of that variable, determine the level of another variable. One such dynamic model could occur if memories of peritraumatic dissociation were created by changes in PTSD symptoms. Trauma victims may reconstruct their traumatic experience in a way that is consistent with the improvement or deterioration of their PTSD symptoms regardless of the severity of their symptoms. Although speculative, this possibility warrants further study.

The current data do not allow us to determine which of these interpretations is correct. Nonetheless, all of these possibilities pose theoretical problems for the construct of peritraumatic dissociation. Specifically, each assumes that measures of peritraumatic dissociation are substantially affected by factors other than actual dissociation experienced during trauma exposure. As such, each—in its own way—calls into question the validity of the retrospective measures of peritraumatic dissociation. On the whole, these interpretations support the view that retrospective measures of peritraumatic experiences may be substantially, albeit not entirely, based on information that is influenced by respondents’ current psychological state rather than merely retrieved from a memory trace created at the time of the incident (Henry, Moffitt, Caspi, Langley, & Silva, 1994; Loftus, 1997). The nature of memory for traumatic events is not well understood (Buckley, Blanchard, & Neill, 2000; Shobe & Kihlstrom, 1997), however, and much research remains to be conducted.

These results also have practical implications for how measures of peritraumatic dissociation should be used. Because recalled dissociation varies over time, researchers and clinicians must use caution in interpreting the meaning of information collected months or years after the traumatic event. Perhaps more important, these data suggest that there may be little, if any, value to be gained by using temporally proximal measures of peritraumatic dissociation as a marker of subsequent PTSD symptom severity. Simply put, the best predictor of future PTSD symptom severity is...
initial symptomatic distress. Measuring peritraumatic dissociation provides no incremental improvement in the prediction of subsequent PTSD symptom severity.

In arriving at these conclusions, it is important to bear in mind certain limitations of the current study. First, this research focused on a largely male sample of survivors of community violence. Additional research is needed to determine the degree to which these findings would generalize to female survivors of community violence as well as to persons exposed to other traumatic experiences. A related concern pertains to the generalizability of these results to other ethnic, racial, and socioeconomic groups inasmuch as our sample was predominantly Hispanic persons of quite modest socioeconomic and educational backgrounds. Thus, future studies that include a broader range of trauma survivors are needed to assess the generalizability of the current results. We should also underscore that our research focused solely on self-appraised symptom severity. Although the representation of clinical phenomena as continuous psychological constructs, rather than discrete psychiatric diagnoses, has certain advantages (Persons, 1986), it is nonetheless conceivable that different findings might have emerged had psychiatric interviews been used to determine diagnostic status. Finally, this research used a measure of neuroticism that was administered subsequent to trauma exposure. Although no significant association was found between degree of injury severity and neuroticism, insofar as our measure does not constitute a true index of pre-exposure neuroticism, additional research that uses a fully prospective design is warranted.

In sum, this longitudinal study of the relationship between peritraumatic dissociation and PTSD symptom severity used covariance structure modeling within a cross-lagged analytic framework, providing perhaps the most comprehensive study to date. The results of this research permit two broad conclusions about the construct of peritraumatic dissociation and its causal relation to PTSD symptoms. First, recall of peritraumatic dissociation is not stable over time. Thus, researchers and clinicians should be concerned about the accuracy of recollections of peritraumatic dissociation when they occur months or years after the traumatic event. Perhaps more significantly, no evidence was found that peritraumatic dissociation serves as a unique and independent predictor of PTSD symptom severity, even though recalled dissociation and PTSD symptoms were highly correlated within each wave. This finding is particularly notable inasmuch as dissociation was measured shortly after the traumatic event. This result is inconsistent with the typical causal interpretation of the correlation between recalled peritraumatic dissociation and PTSD symptom severity. At the very least, if a causal relationship does exist between these constructs, then it does not operate in the straightforward manner posited by previous researchers.

References


Fullerton, C. S., Ursano, R. J., Epstein, R. S., Crowley, B., Vance, K. L.,...


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