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Posttraumatic Stress Disorder and Depression Symptomatology in a Sample of Gulf War Veterans: A Prospective Analysis

Darin J. Erickson
Veterans Affairs Boston Healthcare System

Jessica Wolfe, Daniel W. King, Lynda A. King, and Erica J. Sharkansky
Veterans Affairs Boston Healthcare System and Boston University School of Medicine

The authors examined the relationship over time of posttraumatic stress disorder (PTSD) and depression symptoms in a sample of Gulf War veterans. A large sample (N = 2,949) of Gulf War veterans was assessed immediately following their return from the Gulf region and 18–24 months later. Participants completed a number of self-report questionnaires including the Mississippi Scale for Combat-Related PTSD (T. M. Keane, J. M. Caddell, & K. L. Taylor, 1988) and the Brief Symptom Inventory (L. R. Derogatis & N. Melisaratos, 1983) at both time points and an extended and updated version of the Laufer Combat Scale (M. Gallops, R. S. Laufer, & T. Yager, 1981) at the initial assessment. A latent-variable, cross-lag panel model found evidence for a reciprocal relation between PTSD and Depression. Follow-up models examining reexperiencing, avoidance–numbing, and hyperarousal symptoms separately showed that for reexperiencing and avoidance–numbing symptoms, the overall reciprocal relation held. For hyperarousal symptoms, however, the association was from early hyperarousal to later depression symptoms only.

Posttraumatic stress disorder (PTSD) and depression are highly related. Studies using both clinical and community samples have shown that between one third and two thirds of those diagnosed with PTSD will at some point also be diagnosed with major depressive disorder (e.g., Blanchard, Buckley, Hickling, & Taylor, 1998; Engdahl, Speed, Eberly, & Schwartz, 1991; Helzer, Robins, & McEvoy, 1987; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). This represents a two- to fourfold increase in the risk of major depressive disorder for those with, versus those without, PTSD. A possible explanation for this relationship is that one of the two conditions may represent a risk or vulnerability factor for developing the other condition. Examining the temporal sequencing of PTSD and depression symptomatology, then, represents a first step in understanding their possible causal relation. The goal of the current study was to document the association between PTSD and depression symptomatology in a large sample of Gulf War veterans by using a cross-lag panel analysis.

Darin J. Erickson, National Center for Posttraumatic Stress Disorder, Veterans Affairs Boston Healthcare System, Jamaica Plain Campus; Jessica Wolfe, Daniel W. King, Lynda A. King, and Erica J. Sharkansky, Veterans Affairs Boston Healthcare System, Jamaica Plain Campus, and Department of Psychiatry, Boston University School of Medicine.

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Correspondence concerning this article should be addressed to Darin J. Erickson, who is now at the Alcohol Epidemiology Program, University of Minnesota, Twin Cities Campus, 1300 South Second Street, Suite 300, Minneapolis, Minnesota 55454. Electronic mail may be sent to erickson_d@epi.umn.edu.

A number of possible explanations have been proposed for the reliable correlation between PTSD and depression. As mentioned above, either PTSD or depression may represent risk or vulnerability factors for the other condition. This risk could occur in a number of forms: PTSD may be a risk factor for depression, depression may be a risk factor for PTSD, or both (the conditions are reciprocally related). The specific causal mechanism(s) through which this risk is conveyed could be neurochemical, behavioral, cognitive or any combination of the three. Second, a common third variable may predispose people to both disorders. A specific genetic liability that predisposes people to the development of depression, for example, could also represent a risk factor for PTSD. Conversely, a common situational influence might be present. Stressor exposure, for instance, a hallmark etiologic event for PTSD, may predispose people to the development of depression (see Kessler, 1997, for a review). Finally, the association between the two conditions may be inflated because of nonspecificity, both in terms of the conditions themselves as well as in terms of the measurement criteria. Symptom overlap, for example, may be driving the relation. PTSD, particularly as evidenced by the avoidance–numbing subcomponent (*Diagnostic and Statistical Manual of Mental Disorders, 4th ed.; DSM–IV;* American Psychiatric Association, 1994), is composed of emotional distress symptoms that may overlap with depression symptomatology and inflate the apparent relationship.

One of the first steps in beginning to distinguish between the possible explanations for the association between PTSD and depression is to understand their temporal relation. Studies have shown that the majority of those reporting both PTSD and depression say that PTSD came first (Breslau, Davis, Peterson, & Schultz, 1997; Kessler et al., 1995; Kovacs, Gatsonis, Paulauskas, & Richards, 1989; McFarlane & Papay, 1992; Skodol et al., 1996).

However, all of these studies were cross-sectional and relied on retrospective reports of symptom onset, the validity of which is debatable (e.g., Brewin, Andrews, & Gotlib, 1993). Whereas age-of-onset data may be suggestive of a temporal relation, it is not an explicit test of whether symptoms at one time reliably predict increases later on. Prospective data, in combination with an appropriate analytic model, provide a much stronger test of a direct temporal relation between disorders or symptoms.

Building on this prior work, our primary goal for the current study was to examine the temporal relation between PTSD and depression symptomatology in a large sample of Gulf War veterans. Specifically, the extent to which initial PTSD symptoms predict increases in depression symptoms and the extent to which initial depression symptoms predict increases in PTSD symptoms (over a 2-year period) were assessed. A latent variable, cross-lag panel design was used. Our secondary goal was to examine the differential relations that may exist between the three well-recognized components of PTSD as specified in the *DSM–IV* (i.e., reexperiencing, avoidance–numbing, and hyperarousal; American Psychiatric Association, 1994) and depression symptoms. For instance, any temporal relation found between PTSD and depression symptomatology may be due to the relation between depression symptoms and only one of the components of PTSD. Examining reexperiencing, avoidance–numbing, and hyperarousal separately is in keeping with recent interest in how the primary components of PTSD might differentially relate to the larger construct and to other clinical outcomes (e.g., King, Leskin, King, & Weathers, 1998).

Method

Design and Procedure

The Fort Devens Operation Desert Storm Reunion Survey was designed to measure war stressors and their effects following the end of the Gulf War. Within 5 days of their return to the United States in 1991 (Time 1), we surveyed 2,949 U.S. Army personnel at Fort Devens, MA, using a 45-min paper-and-pencil questionnaire (see Wolfe, Brown, & Kelley, 1993, for a full description of the survey). Members of this cohort represented a broad array of Army personnel, including those deployed from active duty (n = 823), as well as those called from the Reserves (n = 587) and the National Guard (n = 1,505). The entire cohort represented approximately 60% of the military personnel deployed from Fort Devens to the Persian Gulf region. Those not surveyed were primarily in units that were unavailable for participation because of general administrative (e.g., debriefing, checkups, termination from active duty) purposes (Wolfe, Proctor, Davis, Borgos, & Friedman, 1998). We recontacted the full cohort for a follow-up survey (Time 2) in 1992–1993 (18–24 months following the initial survey). Of the 2,313 participants who completed the Time 2 survey (response rate of 78%), 922 (40%) completed the survey during face-to-face unit meetings. Those unavailable at unit meetings were contacted by mail, and 1,086 (47%) completed the survey in this manner. The remaining 304 participants (13%) did not return mailed surveys and were contacted and completed surveys during phone interviews. Informed consent was obtained after providing a complete description of the study to the participants.

Participants

At Time 1, participants included 2,702 men and 240 women. Their mean age was approximately 30 years, with the men significantly older than the women, 30.3 versus 28.1 years, respectively, $t(2878) = 3.98$, $p < .05$. The average education level was just over 13 years, with the women slightly more educated than the men, 13.6 versus 13.1 years, respectively, $t(2929) = 3.72$, $p < .05$. The majority of the cohort (82%) was Caucasian, with 9% African Americans, 4% Hispanic, and 5% designated as "other." Women in the sample were more likely to be members of an ethnic minority (27%) than were men (16%), $\chi^2(1, N = 2{,}941) = 18.5$, $p < .05$. More than half were married (59%), whereas only about a third of the women were married (35%), $\chi^2(1, N = 2{,}926) = 55.5$, $p < .05$. Almost three fourths (72%) of the cohort were called from the Reserves or National Guard, and most were enlisted personnel (92%). In terms of PTSD and depression symptomatology, the sample was quite healthy, with mean Mississippi Scale for Combat-Related PTSD (Keane, Caddell, & Taylor, 1988) scores of 61.9 (SD = 13.4) and 66.8 (SD = 17.5) at Times 1 and 2, respectively, and mean Brief Symptom Inventory (BSI) Depression (Derogatis & Melisaratos, 1983) scores of .52 (SD = .63) and .63 (SD = .78) at Times 1 and 2, respectively. Using a Mississippi Scale cut-score of 94 for presumptive PTSD (Zatzick et al., 1997), 3% at Time 1 and 8% at Time 2 met criteria for presumptive PTSD.

Time 2 participants included 2,119 men and 194 women. Those responding at follow-up were compared with nonresponders on a number of variables collected at Time 1: gender, age, education level, race, marital status, military status, military rank, prior combat experience, Gulf War combat exposure, and PTSD. Nonresponders were more likely to be younger (mean age of responders = 30.6 years, mean age of nonresponders = 28.7 years, $t[1194] = 5.74$, $p < .01$); a member of a minority group (15% of responders identified as minority vs. 27% of nonresponders, $\chi^2[1, N = 2{,}948] = 57.4$, $p < .01$); and deployed from active duty (21% of responders were deployed from active duty; 53% of nonresponders were deployed from active duty, $\chi^2[1, N = 2{,}914] = 254.6$, $p < .01$). No other significant differences were found (all ps > .10).

Similarly, the three survey administration techniques (in-person, mail, phone) were compared on a number of variables collected at Time 2. Those who responded by mail were more likely to be female (11%) than were those who responded by phone (7%) or in person (7%), $\chi^2(2, N = 2{,}312) = 14.1$, $p < .05$, and were less likely to be enlisted personnel (90%) than were those who responded by phone (93%) or in person (94%), $\chi^2(2, N = 2{,}284) = 8.4$, $p < .05$. Those who responded by phone were less likely to have previous combat service (9%) than those who responded by mail (14%), $\chi^2(1, N = 1{,}353) = 4.7$, $p < .05$ and were less likely to be married (50%) than those responding by mail (60%) or in person (58%), $\chi^2(2, N = 2{,}302) = 9.6$, $p < .05$. Those responding by phone were also more likely to be in the Reserves or National Guard (91%) than those who responded by mail (82%), who in turn were more likely than those who responded in person (71%), $\chi^2(2, N = 2{,}290) = 69.2$, $p < .05$. Those who responded by mail were older (31.8 years) than those who responded by phone (29.1 years) or in person (29.6 years), $F(2, 2280) = 19.8$, $p < .05$. Those who responded by mail had more years of education (13.3) than those who responded in person (13.0), $F(2, 2303) = 7.4$, $p < .05$. Those who responded by mail reported higher levels of PTSD symptomatology (mean Mississippi Scale score = 68.2) than those who responded by phone (mean Mississippi Scale score = 64.9), $F(2, 2299) = 6.9$, $p < .05$. Similarly, those who responded by mail reported higher levels of depression (mean BSI Depression score = .74) than those who responded by phone (.45) or in person (.56), $F(2, 2305) = 23.7$, $p < .05$. Because of these differences, survey technique was included as a covariate in all structural models.

Measures

Gulf War combat exposure. We assessed Gulf War combat exposure with the Laufer Combat Scale (Gallops et al., 1981), augmented with items that described distinctive Gulf War experiences (e.g., being on alert for SCUD missile or biochemical attack; Rosenheck, 1992). This self-report measure, designed to assess a range of combat experiences, contains 33 items, each scored using a 3-point Likert response format, anchored at 0

(*never*), 1 (*once or twice*), and 2 (*three or more times*). The reliability and validity of the Laufer scale has been well-established (Gallops et al., 1981); in the current study, coefficient alpha was .73. All 33 items were summed to create a total Gulf War combat exposure score.

PTSD. We evaluated PTSD symptoms at both Time 1 and Time 2 by using an extended version of the Mississippi Scale. The measure is composed of 39 items scored using a 5-point (1 to 5) Likert scale. In the present study, minor wording changes were made in the Mississippi Scale to reference the Gulf War context (Wolfe et al., 1993). The Mississippi Scale has been found to be reliable and valid as a self-report measure of PTSD and has demonstrated excellent sensitivity (.93) and specificity (.89) with clinical diagnoses of PTSD (Keane et al., 1988). For the current study, a rational approach was used to identify a subset of Mississippi Scale items that correspond most closely to the 17 PTSD symptoms identified in the *DSM-IV*. Four PhD-level clinicians with extensive experience in research and treatment of PTSD rated the 39 items of the extended Mississippi Scale on the extent to which the items corresponded to the three symptom clusters (reexperiencing, avoidance–numbing, and hyperarousal) in the *DSM-IV*. The highest rated items within each of the three criteria were then evaluated for the extent to which they directly corresponded to a specific symptom, retaining the highest rated items for each symptom. On the basis of these ratings, 15 items were then identified as most saturated with content representing the three clusters, with 5 items for each. This subset of items measured all but one *DSM-IV* symptom, namely "sense of foreshortened future (e.g., does not expect to have a career, marriage, children, or normal lifespan)." Internal consistency reliability for the 15 items was quite good, with coefficient alphas of .83 and .87 at Times 1 and 2, respectively. These 15 items correlate highly with the total score of the full Mississippi Scale (.89 and .92 at Times 1 and 2, respectively). The retained items were used to create 3 first-order latent variables representing reexperiencing, avoidance–numbing, and hyperarousal symptoms and, from these, a higher order latent variable representing PTSD symptoms.

Depression. We measured symptoms at both Time 1 and Time 2 using the Depression subscale of the BSI. The BSI is a 53-item self-report questionnaire in which people respond to a number of phrases by using a 5-point, Likert-type format anchored at *not at all* and *extremely*. Previous factor analyses (Derogatis, 1993) have identified seven items (thoughts of ending your life, feeling lonely, feeling blue, feeling no interest in things, feeling hopeless about the future, feelings of worthlessness, your feelings being easily hurt) hypothesized to load on a depression factor. In the current sample, reliability of the set of depression items was quite good, with coefficient alphas of .81 and .86 at Times 1 and 2, respectively. These seven items were used to create a latent variable representing depression symptoms.

Overview of Analyses

After first examining levels of depression and posttraumatic stress symptoms across time and gender by using repeated measures analyses of variance (ANOVAs), we assessed the temporal relation between PTSD and depression symptoms by using a latent variable, cross-lag panel analysis (conducted with the Mplus program; Muthen & Muthen, 1998). To control for the effects of potential third variable explanations on the association between PTSD and depression, we included gender, combat exposure, and type of survey administration (in-person, mail, or phone) as covariates in all structural models. A second-order factor representing PTSD symptoms and a factor representing depression symptoms were extracted at each of the two time points. We estimated a structural equation model using a multistep technique similar to that proposed by Anderson and Gerbing (1988). First, we evaluated the fit of the measurement model and made any necessary modifications. After establishing a measurement model that adequately fit the data, the structural model was evaluated in a number of stages. Initially, the fit of a base model was established. This base model estimated paths from combat exposure, gender, and survey administration to the 4 latent factors (PTSD and Depression symptoms at both Times 1 and 2) and both autoregressive paths (a path from Time 1 to Time 2 PTSD symptoms and from Time 1 to Time 2 Depression symptoms). Next, the two cross-lag paths were systematically freed and the change in fit was evaluated. In Model 1a, the cross-lag path from Time 1 PTSD symptoms to Time 2 Depression symptoms was freed, and the path from Time 1 Depression symptoms to Time 2 PTSD symptoms was constrained at zero. The opposite cross-lag path was evaluated in Model 1b, with the path from Time 1 Depression symptoms to Time 2 PTSD symptoms freed while the path from Time 1 PTSD symptoms to Time 2 Depression symptoms was constrained at zero. Finally, both cross-lag paths were estimated simultaneously in Model 2.

To assess comparability across the three *DSM-IV* PTSD symptom clusters, separate models examining the relation over time of depression and each of the 3 first-order factors (representing reexperiencing, avoidance–numbing, and hyperarousal symptoms) were estimated. The multistep approach of first establishing the fit of the measurement model and then testing the structural model was replicated for each of the three symptom clusters.

Results

Changes in PTSD and Depression Symptoms Over Time

One-way repeated measures ANOVAs were conducted for both PTSD and depression symptoms to examine differences across gender and the 2 time points. PTSD and depression total scores were created for each time point by summing across the 15 retained Mississippi Scale items and the 7 BSI Depression items, respectively. In addition, separate summed scores were created representing reexperiencing, avoidance and numbing, and hyperarousal symptoms.

For depression, women were more symptomatic than men, $F(1, 2122) = 38.62$, $p < .01$, and symptomatology increased over the two time points, $F(1, 2122) = 15.41$, $p < .01$. The interaction between gender and time was not significant. An identical pattern was seen for PTSD symptomatology, with women more symptomatic than men, $F(1, 2122) = 34.52$, $p < .01$, and symptomatology increasing over the two time points, $F(1, 2122) = 181.18$, $p < .01$.

For each of the three symptom clusters (reexperiencing, avoidance–numbing, and hyperarousal), women were more symptomatic than men were and symptomatology increased over the two time points (all $ps < .01$). For reexperiencing, the interaction between gender and time was also significant, $F(1, 2122) = 7.00$, $p < .01$, with the women showing a slightly larger increase in reexperiencing symptomatology from Time 1 to Time 2 than the men.

Higher Order PTSD Symptomatology

Measurement model. In the measurement model, one loading for each latent variable was constrained at unity, factor loadings were constrained to be equal over time, errors were allowed to covary over time, and the PTSD and Depression symptom latent variables were correlated. The disturbances for two of the first-order PTSD symptoms factors (Time 1 Hyperarousal and Time 2 Avoidance–Numbing) were constrained at zero to prevent Heywood cases (Dillon, Kumar, & Mulani, 1987). Using maximum-likelihood estimation, the fit of the base-measurement model was acceptable, $\chi^2(1050, N = 2, 109) = 5{,}519.1$, $p < .01$, and root-mean-square error of approximation (RMSEA; Steiger, 1990) was

.045. The 90% confidence interval for RMSEA ranged from .044 to .046, with an associated probability of close fit of 1.00.

With the exception of the suicidal ideation item (standardized factor loading of .43 at both times) the standardized factor loadings for the depression symptoms were above .60 (between .62 and .90). Two factor loadings for Avoidance–Numbing and one loading for Hyperarousal were below .40. The factor loadings from the first-order latent variables to the second-order PTSD Symptom latent variable were all above .90. The latent variables were all highly correlated. Time 1 PTSD correlated .67 with Time 2 PTSD, whereas Time 1 Depression correlated .51 with Time 2 Depression. Not surprisingly, PTSD and Depression were also highly correlated with each other, with Time 1 PTSD correlated .65 with Time 1 Depression and Time 2 PTSD correlated .74 with Time 2 Depression. Across time PTSD–Depression correlations were smaller, with Time 1 Depression and Time 2 PTSD correlated .50 and Time 1 PTSD and Time 2 Depression correlated .45. Combat exposure was significantly correlated with both PTSD and Depression symptoms at both time points (ranging from .15 to .34). Gender was approximately equally correlated (correlations between .13 and .15) with both PTSD and Depression at both time points. Responding to the mailed survey was positively associated with both PTSD and Depression at both times (correlations between .08 and .15).

Structural model. The initial structural model included (a) paths from gender, combat exposure, and the two dummy-coded survey administration variables to the PTSD and Depression symptom factors at both time points, and (b) autoregressive paths for both the PTSD and Depression symptoms factors. This "autoregressive only" model was used as a base model against which the cross-lag paths were tested for significance (see Table 1).

The two cross-lag paths, from Time 1 PTSD to Time 2 Depression and Time 1 Depression to Time 2 PTSD, were tested separately. As seen in Table 1, freeing the path from Time 1 Depression to Time 2 PTSD significantly improved the fit of the base model (using both a chi-square difference test and the sample-size adjusted Bayesian information criterion). Similarly, freeing the path from Time 1 PTSD to Time 2 Depression also significantly improved the fit of the base model. Furthermore, estimating both cross-lag paths simultaneously improved model fit over these two single cross-lag models. Thus, there is support for the inclusion of both the cross-lag path from Depression to PTSD and the cross-lag path from PTSD to Depression. A final test was then conducted to test whether these two cross-lag paths differed in magnitude. As seen in the bottom row of Table 1, constraining the two cross-lag paths to be equal decreased the fit of the model, suggesting that the two paths, although both statistically significant, differ from each other in magnitude. The final structural model (with standardized parameter estimates) is shown in Figure 1. The autoregressive paths for PTSD and Depression were moderate, with standardized estimates of .55 and .36, respectively. The cross-lag path from Time 1 PTSD to Time 2 Depression was .21, with higher PTSD symptoms at Time 1 associated with increased depression symptoms at Time 2 (relative to Time 1). Although significant, the cross-lag path from Time 1 Depression to Time 2 PTSD was smaller (.11), with higher depression symptoms at Time 1 associated with increased PTSD symptoms at Time 2 (relative to Time 1).

Reexperiencing Symptomatology

Measurement model. The initial measurement model, including covaried errors, one factor loading for each latent variable constrained to unity, factorial invariance over time, and fully covaried factors, had an adequate model fit, $\chi^2(314, N = 2,147) = 2,417.7$, $p < .01$, and RMSEA = .056 (90% confidence interval ranging from .054 to .058). The standardized factor loadings for Reexperiencing ranged from .58 to .76, and, except for the suicidality item (.32), the factor loadings for Depression were above .50 (between .52 and .90). The correlations among the factors ranged from .38 to .62.

Structural model. The base structural model replaced the correlated factors of the measurement model with direct paths from combat exposure, gender, and survey technique to the four latent variables and autoregressive paths for Reexperiencing and Depression symptoms. This base structural model fit the data adequately (see Table 2). Freeing either one or the other cross-lag improved the fit of the base model, and simultaneously estimating both cross-lags further incremented model fit. Furthermore, adding an equality constraint to the two cross-lags did not significantly reduce fit, suggesting that the magnitudes of the two cross-lags are

Table 1
Fit Indices for the Overall Structural Equation Model Relating PTSD and Depression Symptomatology Over a 2-Year Period

Model	χ^2	*df*	RMSEA (90%CI)	AIC	SBIC	$\Delta\chi^2(1, N = 2,109)$
Base model	5,597.16	1052	.045 (.044, .046)	217,850	218,157	—
Depression → PTSD	5,557.18	1051	.045 (.044, .046)	217,812	218,122	39.98*
PTSD → depression	5,531.64	1051	.045 (.044, .046)	217,786	218,096	65.52*
Both cross-lags	**5,519.08**	**1050**	**.045 (.044, .046)**	**217,776**	**218,088**	**38.10,* 12.56***
Cross-lags constrained	5,547.53	1051	.045 (.044, .046)	217,802	218,112	28.45*

Note. Chi-square difference tests are the difference in chi-square values between the model and the immediately prior model with one degree of freedom difference. For the model with both cross-lags freely estimated, $\Delta\chi^2$ are given for both single cross-lag models. The model in bold typeface was accepted as the final model. PTSD = posttraumatic stress disorder; CI = confidence interval; RMSEA = root-mean-square error of approximation; AIC = Akaike information criterion; SBIC = sample-size adjusted Bayesian information criterion.
* $p < .05$.

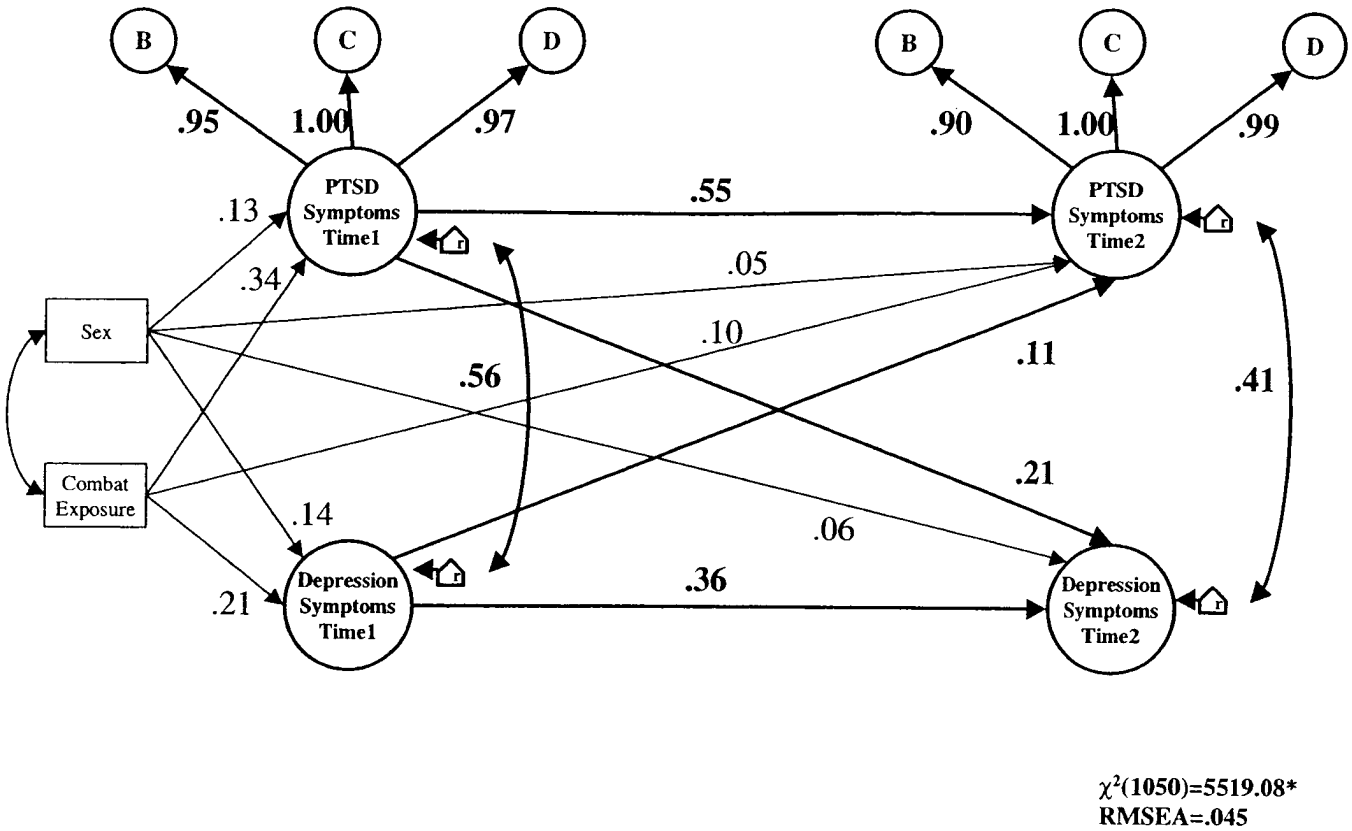

Figure 1. Model relating posttraumatic stress disorder (PTSD) and depression symptomatology. Although not shown in the diagram, mail survey is significantly associated with PTSD at Time 1 and Time 2 (both standardized paths = .05) and depression symptomatology at Time 1 and Time 2 (standardized paths = .07 and .08, respectively). Phone survey is significantly associated with depression symptomatology at Time 2 (standardized path = −.04). r = residual; RMSEA = root-mean-square error of approximation; B = reexperiencing symptoms; C = avoidance and numbing symptoms; D = hyperarousal symptoms. * $p < .05$.

equal. Thus, against the backdrop of moderate stability of Depression and Reexperiencing symptoms (standardized autoregressive paths of .45 and .46, respectively), higher Depression symptoms at Time 1 were associated with higher Reexperiencing symptoms at Time 2 (controlling for Reexperiencing symptoms at Time 1; standardized coefficient of .16) and higher Reexperiencing symptoms at Time 1 were associated with higher Depression symptoms at Time 2 (standardized coefficient of .07). This model is shown in Figure 2.

Avoidance–Numbing Symptomatology

Measurement model. The initial measurement model for Avoidance–Numbing (identical in form to that used for Reexperiencing symptoms) also fit the data adequately, $\chi^2(324, N = 2,152) = 2{,}637.7$, $p < .01$, and RMSEA = .058 (90% confidence interval ranging from .056 to .060). All standardized factor loadings were between .38 and .91, and the correlations among the factors ranged from .45 to .77.

Structural model. Using a technique identical to that used for Reexperiencing symptoms, support was found for both cross-lag paths, although the equality of these two cross-lag paths was not supported (see Table 2). As seen in Figure 3, higher Avoidance–Numbing symptoms at Time 1 were associated with higher Depression symptoms at Time 2 (relative to Time 1 Depression symptom levels; standardized coefficient of .20). Similarly, higher Depression symptoms at Time 1 were associated with higher Avoidance–Numbing symptoms at Time 2 (relative to Time 1 Avoidance–Numbing symptom levels; standardized coefficient of .11).

Hyperarousal Symptomatology

Measurement model. As with the two previous models, the measurement model for the Hyperarousal factor fit the data adequately, $\chi^2(324, N = 2,173) = 2{,}803.1$, $p < .01$, and RMSEA = .059 (90% confidence interval ranging from .057 to .061). All standardized factor loadings were between .26 and .90, and the correlations among the factors ranged from .52 to .83.

Structural model. As seen in the bottom of Table 2, although freeing each cross-lag improved fit over the base model, freeing the path from Time 1 Depression to Time 2 Hyperarousal did not increment fit beyond that which was due to freeing the path from Time 1 Hyperarousal to Time 2 Depression symptoms. Higher levels of Hyperarousal at Time 1 were associated with higher Hyperarousal and Depression symptoms at Time 2 (see Figure 4).

Table 2
Fit Indices for the Structural Equation Models Relating Reexperiencing, Avoidance and Numbing, and Hyperarousal With Depression Symptomatology Over a 2-Year Period

Model	χ^2	*df*	RMSEA (90%CI)	AIC	SBIC	$\Delta\chi^2(1, N = 2{,}152)$
			Reexperiencing			
Base model	2,804.88	326	.060 (.057, .062)	115,781	115,981	—
Depression → reexperiencing	2,770.84	325	.059 (.057, .061)	115,749	115,951	34.0*
Reexp → depression	2,777.04	325	.059 (.057, .061)	115,755	115,958	27.8*
Both cross-lags	2,754.80	324	.059 (.057, .061)	115,735	115,940	16.0,* 22.2*
Cross-lags constrained	**2,757.79**	**325**	**.059 (.057, .061)**	**115,736**	**115,938**	**3.0**[a]
			Avoidance and numbing			
Base model	2,675.29	326	.058 (.056, .060)	130,071	130,271	—
Depression → avoidance	2,658.35	325	.058 (.056, .060)	130,056	130,259	16.9*
Avoidance → depression	2,654.65	325	.058 (.056, .060)	130,041	130,243	20.6*
Both cross-lags	**2,637.69**	**324**	**.058 (.056, .060)**	**130,038**	**130,243**	**20.7,* 17.0***
Cross-lags constrained	2,651.81	325	.058 (.056, .060)	130,050	130,252	14.1*
			Hyperarousal			
Base model	2,897.21	326	.060 (.058, .062)	137,444	137,645	—
Depression → hyperarousal	2,874.67	325	.060 (.058, .062)	137,424	137,627	22.5*
Hyperarousal → depression	**2,804.15**	**325**	**.059 (.057, .061)**	**137,353**	**137,556**	**93.1***
Both cross-lags	2,803.11	324	.059 (.057, .061)	137,354	137,560	71.6,* 1.0[a]

Note. Chi-square difference tests are the difference in chi-square values between the model and the immediately prior model with one degree of freedom difference. For the model with both cross-lags freely estimated, $\Delta\chi^2$ are given for both single cross-lag models. The model in bold typeface was accepted as the final model. CI = confidence interval; RMSEA = root-mean-square error of approximation; AIC = Akaike information criterion; SBIC = sample-size adjusted Bayesian information criterion.
[a] Nonsignificant at $\alpha = .05$.
* $p < .05$.

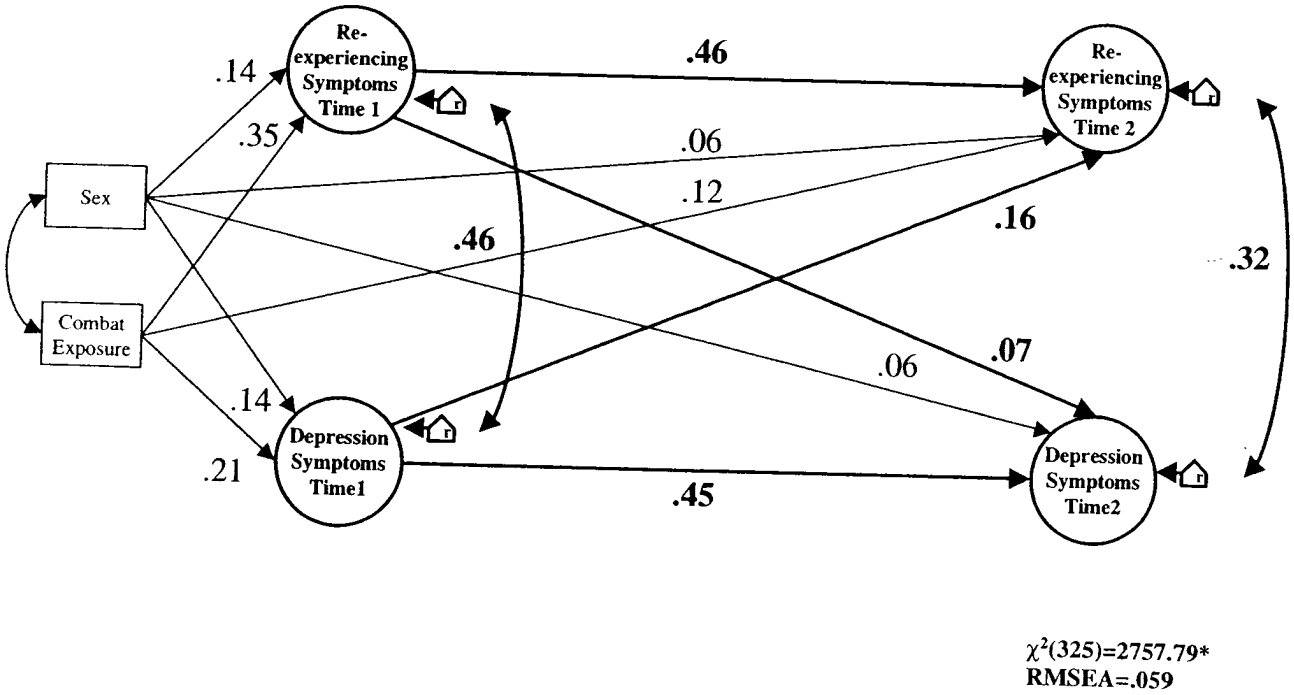

Figure 2. Model relating reexperiencing and depression symptomatology. Although not shown in the diagram, mail survey is significantly associated with reexperiencing at Time 1 (standardized path = .05) and depression symptomatology at Time 1 and Time 2 (standardized paths = .07 and .08, respectively). Phone survey is significantly associated with depression symptomatology at Time 2 (standardized path = −.05). r = residual; RMSEA = root-mean-square error of approximation. * $p < .05$.

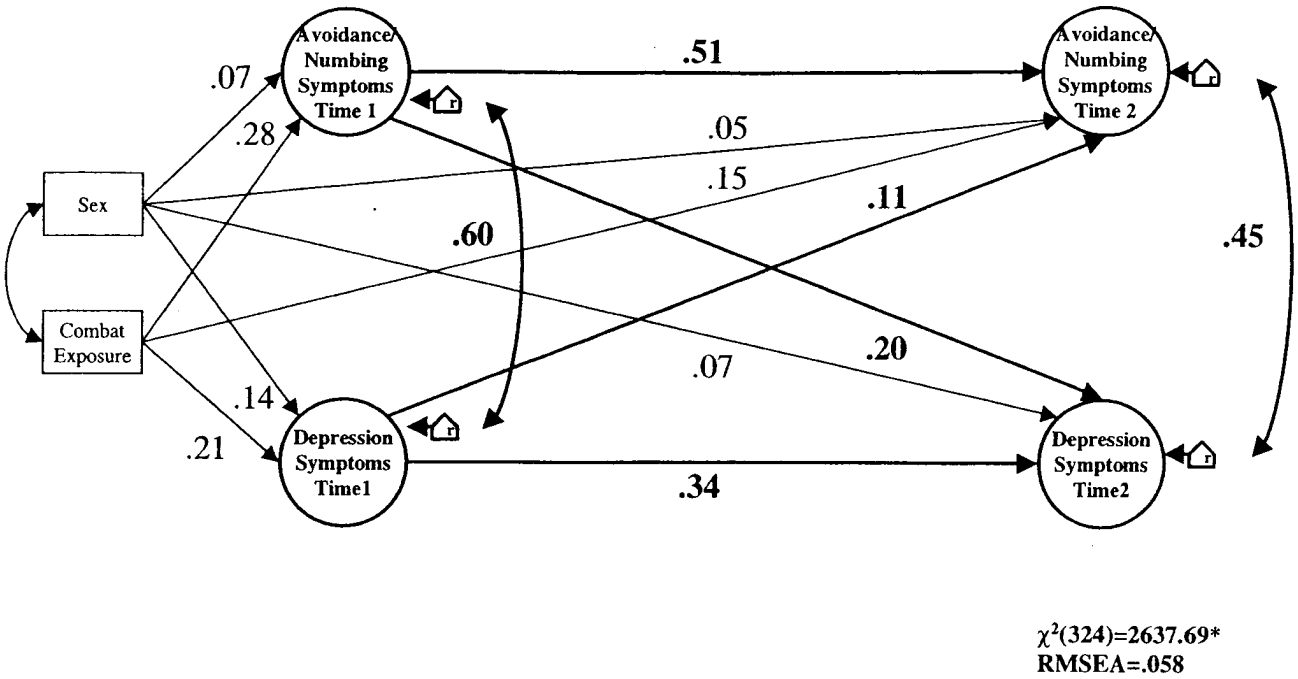

Figure 3. Model relating avoidance and numbing and depression symptomatology. Although not shown in the diagram, mail survey is significantly associated with avoidance and numbing at Time 1 (standardized path = .06) and depression symptomatology at Time 1 and Time 2 (standardized paths = .07 and .08, respectively). Phone survey is significantly associated with depression symptomatology at Time 2 (standardized path = −.05). r = residual; RMSEA = root-mean-square error of approximation. * $p < .05$.

Discussion

Our study reports a number of findings that potentially help explicate the relationship between PTSD and depression. We found support for a bidirectional relation over the 2-year study interval, although initial PTSD symptoms were more strongly predictive of later depression than vice versa. This prospective finding is consistent with previous retrospective research (e.g., Kessler et al., 1995) in which a majority of those with both disorders report that PTSD preceded depression. Because the path from Depression to later PTSD was also significant, however, these findings provide confirmation for results from retrospective research in which depression has been shown to precede PTSD. Breslau et al. (1997), for example, found that the likelihood of depression leading to PTSD in young women was nearly as strong as that for PTSD predicting later depression. Further, prior depression significantly increased the risk for subsequent traumatic exposure, irrespective of the PTSD diagnosis.

More important, our data suggest that the overall bidirectional relation between PTSD and depression is complex and potentially better understood when PTSD is disaggregated into its three symptom components. Symptoms of hyperarousal, for example, reliably preceded symptoms of depression in the current study. The importance of hyperarousal appearing early on, prior to the onset of later depression, is especially noteworthy for several reasons. Prior research with Gulf War veterans has indicated that hyperarousal symptoms are more likely to be seen immediately following trauma than are either reexperiencing or avoidance and numbing symptoms (Southwick et al., 1993), leading these researchers to speculate about the diagnostic and prognostic significance of hyperarousal immediately following trauma. The current results support the significance of early hyperarousal, and suggest that in addition to its association with PTSD, hyperarousal may impact other outcomes such as depression. In terms of comorbidity, however, Friedman and Yehuda (1995) discussed how psychophysiological reactivity (likely related to hyperarousal symptoms, but see Eifert & Wilson, 1991) differentiates those with PTSD from those with depression. Additional studies designed to examine hyperarousal more specifically, both in terms of how it relates to the course of PTSD as well as to other outcomes, are warranted.

Although there was support for a bidirectional relation between avoidance and numbing and depression, the magnitude of the standardized path from avoidance and numbing to depression was much larger. A decrement in positive interaction is one possible explanatory factor for this link. That is, symptoms of behavioral avoidance could, by definition, interfere with experiences involving positive interaction or reinforcement, and actively avoiding or withdrawing from situations may decrease positive interactions or experiences, which, in turn, may increase depressive symptoms like isolation and loneliness. A second possibility is that the affective dampening typically linked with avoidance and numbing could be responsible for the increased symptoms of depression (e.g., Litz et al., 1997). In the general sense, affective dampening may produce an attenuated overall emotional response that could lead to depression symptoms. Alternatively, affective dampening may have more specific effects, such as selectively blocking perceptions of positive feelings.

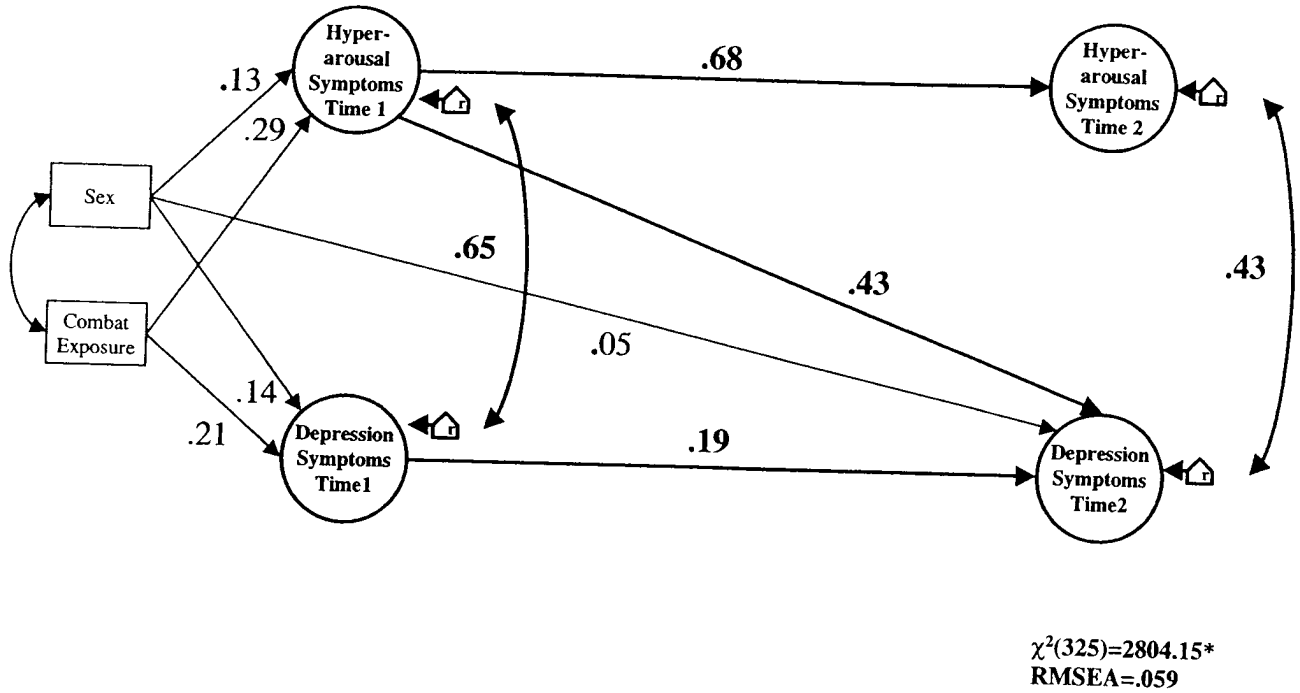

Figure 4. Model relating hyperarousal and depression symptomatology. Although not shown in the diagram, mail survey is significantly associated with hyperarousal at Time 2 (standardized path = .07) and depression symptomatology at Time 1 and Time 2 (standardized paths = .07 and .08, respectively). Phone survey is significantly associated with depression symptomatology at Time 2 (standardized path = −.04). r = residual; RMSEA = root-mean-square error of approximation. * $p < .05$.

Like the link between depression and avoidance–numbing, the temporal relation between reexperiencing and depression was bidirectional, although the path from depression to later reexperiencing appeared somewhat larger. An association between reexperiencing and depression is consistent with a growing body of research linking intrusive memories and depression. Studies have indicated that intrusive recollections of adverse events significantly predict the onset of depression, signaling that intrusion could mark the start of depression or, alternatively, a susceptibility for this disorder (e.g., Brewin, Hunter, Carroll, & Tata, 1996). Furthermore, Brewin and colleagues have directly compared the intrusive memories of patients with PTSD and depression and found that, although there are some distinctions, the memories are quite similar (Brewin, 1998; Reynolds & Brewin, 1999). Together, these findings suggest that regardless of origin, intrusive memories may act as a mediator or link between PTSD and depression.

The impact of comorbid psychiatric disorders on treatment and prevention has received much recent attention (Kendall & Clarkin, 1992; McLean, Woody, Taylor, & Koch, 1998). Although our findings are not tied strictly to diagnoses, results from the examination of the separate symptom clusters may provide important information for both the treatment and the prevention of PTSD and depression comorbidity. On the basis of the bidirectional relation between reexperiencing and depression, for instance, our results raise the possibility that treatments that directly address intrusive thoughts may prove effective for both PTSD and depression. A more efficacious application of these findings may lie in prevention. Understanding the strong relationship between certain PTSD symptoms, particularly hyperarousal, and later depression in trauma-exposed individuals may provide guidance on ways to curtail the development of these secondary disorders through the amelioration or attenuation of symptoms with early onset, particularly if causal links are confirmed.

The current study has certain limitations. Given differences in rates of PTSD and depression between men and women (e.g., Kessler et al., 1995), it is critical to examine whether gender is a factor. In this study we controlled for gender; there were not enough female participants for separate analyses. Future work might benefit from larger numbers of female participants and separate models for both genders (our results did not differ when models were limited to men only). Our measure of depression was less than ideal. A more thorough and comprehensive measure of depression symptomatology would strengthen the current results. Likewise, we used items taken from the Mississippi Scale to measure *DSM–IV* symptoms of PTSD. Because of this, we did not have an item specifically measuring the reexperiencing symptom of a sense of foreshortened future. This study also had no data related to participants' predeployment psychological status. Even though participants were presumably healthy at deployment, it is possible that some had preexisting psychological symptoms.

Finally, a few design limitations are worth noting. There were a number of differences between survey administration and, although we controlled for this statistically, they may influence results. We have two data points in the current set of analyses. Future work including additional waves of data could allow for stronger conclusions through the use of more sophisticated analytic techniques (e.g., state–trait modeling, latent growth curve modeling, survival analysis). Latent growth curve modeling, for

example, has the advantages of parsimoniously integrating multiple waves of data and modeling interindividual differences in intraindividual change. Related to this, categorizing subjects in certain ways (e.g., PTSD only, depression only, early PTSD and later depression, early depression and later PTSD) and analyzing those classes may prove to be quite useful.

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