Published in final edited form as:
Behav Res Ther. 2009 December ; 47(12): 1012–1017. doi:10.1016/j.brat.2009.07.014.

Does prior exposure to interpersonal violence increase risk of PTSD following subsequent exposure?

Jesse R. Cougle,
Department of Psychology, Florida State University

Heidi Resnick, and
National Crime Victims Research and Treatment Center, Medical University of South Carolina

Dean G. Kilpatrick
National Crime Victims Research and Treatment Center, Medical University of South Carolina

Abstract

Research has generally found a "dose relationship" between potentially traumatic events (PTEs) and the likelihood of developing PTSD, with greater number of events associated with greater likelihood. Most of these studies have been cross-sectional, however. A recent prospective study (Breslau, Peterson, & Schultz, 2008) found that PTSD response to prior potentially traumatic event (PTE) exposure, rather than prior PTE exposure itself, acts as a risk factor for PTSD in response to subsequent PTE; however, this analysis combined many different types of events, and the unique contribution of specific events (e.g., assault) that may be associated with differential risk of PTSD was indeterminable. The present study examined the effects of cumulative PTE exposure prospectively using a two-wave design in the National Survey of Adolescents (*N*=1,703). History of assault and witnessing serious violence were the focal PTEs examined. Wave I assault without PTSD was found to predict PTSD at Wave II following exposure to new assault or witnessed violence; however, among those without prior PTSD, Wave I witnessed violence did not increase risk of subsequent PTSD following exposure.

Keywords

posttraumatic stress disorder; revictimization; assault; risk factors for PTSD; sensitization hypothesis

Epidemiological studies estimate that 40–80% of people will experience at least one traumatic or life-threatening event during their lifetimes (Breslau, Davis, Andreski, & Peterson, 1991; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Individuals exposed to one potentially traumatic event (PTE) are at increased risk for subsequent exposure, possibly due to the existence of social and/or psychological variables affecting their safety (Stein, Jang, Taylor, Vernon, & Livesley, 2002). Posttraumatic stress disorder (PTSD) is one possible consequence of exposure to PTEs, and evidence has accumulated suggesting a dose relationship between such events and the likelihood of developing PTSD. This relationship has been explained by

Correspondence concerning the manuscript may be addressed to, Jesse R. Cougle, Department of Psychology, Florida State University, P.O. Box, 3064301, Tallahassee, FL 32306, or Dean G. Kilpatrick, National Crime Victims, Research and Treatment Center, Medical University of South Carolina, P.O. Box, 250852, 165 Cannon Street, Charleston, SC 29425.

the "sensitization" hypothesis, which posits that prior exposure sensitizes people to respond more intensely to subsequent stressors (e.g., Resnick, Yehuda, Pitman, & Foy, 1995; Yehuda, Kahana, Schmeidler, et al., 1995).

A few different mechanisms could account for the relationship between cumulative PTE exposure and increased risk of PTSD. An "inflation effect" may occur whereby exposure to a more severe stressor could enhance the conditioned response associated with a moderately stressful event (Rescorla, 1974). This suggests that someone who developed a minor fear of cars following a crash might develop a more intense driving phobia after experiencing rape or physical assault (Mineka & Zinbarg, 2006). In addition, research using male rats has found that prior exposure to multiple shocks (i.e., a "trauma") enhances the subsequent learning of conditioned fear (Rau, DeCola, & Fanselow, 2005).

Strong evidence for the sensitization hypothesis comes from studies of military veterans. Using data from the National Vietnam Veterans Readjustment Study (NVVRS) and military service records, investigators found rates of lifetime PTSD among veterans with low, moderate, and high combat exposure that were 10.3%, 24.9%, and 38.6%, respectively (Dohrenwend, Turner, Turse et al., 2006). A similar relationship between frequency of combat exposure and PTSD was found among Israeli soldiers (Solomon & Flum, 1988) and Gulf War veterans (Stein, Tran, Lund, et al., 2005). In addition, analyses of the NVVRS indicated that pre-war trauma history predicted combat-related PTSD among men (King, King, Foy & Gudanowski, 1996) and post-war stressful life events were associated with an increase in PTSD symptoms (King, King, Fairbank, Keane, & Adams, 1998). However, these findings relate to particular type of trauma in a unique population and may not be generalizable to civilian populations.

Data from non-military civilian populations also provide evidence for the cumulative effects of trauma on increasing risk of PTSD. For example, Kilpatrick and Saunders (1999) found a lifetime prevalence of PTSD among youth to be 7.3% for those experiencing one PTE, though those who were multiply victimized had rates that were three times as high (24.6%). Prospective research has also found childhood abuse to be predictive of PTSD in response to PTEs experienced as an adult, with higher frequency of previous trauma implicating greater risk of subsequent PTSD (Breslau, Chilcoat, Kessler, & Davis, 1999).

Recently, Breslau, Peterson, and Schultz (2008) noted some potential limitations of research demonstrating a dose effect of PTEs on risk of PTSD. They argued that many reports are retrospective and suffer from problems associated with recall bias. That is, people who report PTSD may be biased towards the recollection of a greater number of PTEs. In addition, they noted that studies have not assessed the potential role of PTSD in response to prior traumatic events, which may be more important rather than prior PTE exposure in increasing risk for PTSD response to subsequent PTE. Certain individuals may be more prone to the experience of both PTE and PTSD, and underlying psychological vulnerabilities rather than frequency of PTEs may account for the apparent dose relationship.

To address these limitations, Breslau and colleagues (2008) used longitudinal data with repeated assessments over a 10-year period. They found conditional risk of PTSD during the follow-up periods to be much higher among traumatized individuals with PTSD than among those with no prior PTE experience. In addition, they found those with prior exposure without baseline PTSD to be at no greater risk of developing PTSD in the follow-up periods in response to a subsequent PTE than those without prior exposure. They concluded that these results contradicted the sensitization hypothesis and argued that psychological vulnerabilities rather than frequency of PTEs were important in increasing risk for PTSD.

In conducting their analyses, Breslau et al. did not distinguish between different kinds of traumatic events. More severe events, such as rape and physical assault, were grouped with

accidents and witnessed violence, events associated with lower rates of PTSD (Breslau, Kessler, Chilcoat et al., 1998; Kessler et al., 1995). Research on emergency healthcare workers found that those who experienced a distressing work event involving direct threat had different and in some ways more severe symptom profiles than those who witnessed an event that was threatening to patients (Alden, Regambal, & Laposa, 2008). A meta-analysis of risk factors for PTSD also found that prior traumatic event exposure was more strongly related to the development of PTSD when it involved interpersonal violence rather than an accident (Ozer, Best, Lipsey, & Weiss, 2003). Thus, the effects of more severe PTEs in increasing risk of PTSD in response to subsequent PTEs may have been diluted through this categorization scheme.

In the present study, we sought to expand on the investigation by Breslau and colleagues (2008) by examining the effects of physical and sexual assault on risk of PTSD in response to subsequent PTEs. We also assessed the effects of a more indirect PTE, prior witnessed violence, on increasing subsequent PTSD risk. Using longitudinal data with two assessment points covering an eight-year span, we predicted that among those without PTSD at Wave I, prior assault but not witnessed violence would be predictive of PTSD in response to a new PTE reported at Wave II.

Methods

Participants

Schulman, Ronca and Bucavala, Inc. (SRBI; a New York-based national survey research firm) conducted follow-up interviews with a national probability sample of 3,161 adolescents and a central city oversample of 862 adolescents who had completed Wave I interviews as part of the National Survey of Adolescents (NSA) approximately 7 to 8 years before. A more detailed description of the sample and methodology for the original NSA is provided in Kilpatrick, Acierno, Saunders et al. (2000). The data presented here are drawn from the 1,725 participants who completed both Wave I of the data collection in 1995 and Wave II of data collection in 2004. The majority of the sample was Caucasian non-Hispanic (74.1%), 12.1% was African-American, non-Hispanic, 8.4% was Hispanic, 2.7% was Asian-American, and 2.7% was Native American. Age at Wave I ranged from 12–17 (Mean = 14.57) but age at Wave II ranged from 18–26 (Mean = 22.21). Only 5.6% had less than high school education at Wave II.

Procedures

A highly structured interview was conducted via telephone using a Computer Assisted Telephone Interviewing (CATI) technology such that questions appeared on a computer screen to be read verbatim by highly trained interviewers. This format facilitated complex skip patterns, reduced data entry errors, and insured that questions were asked as written. Participants received a $10 incentive by mail for completing the interview.

Measures

The interviews at Waves I and II were designed to assess several domains including demographics, interpersonal violence exposure, family alcohol and drug problems, and psychopathology. These are described in more detail below.

Demographics

Standard biographical variables were assessed including gender, racial/ethnic status, age, educational achievement and residential location.

Interpersonal Violence Exposure

Sexual assault, physical assault and witnessing violence history were assessed via behaviorally specific questions to ensure precise communication of the types of events under consideration, thereby increasing the likelihood of valid self-report of victimization (Koss, 1993). Other measures were taken to encourage accurate reporting, including the use of introductory statements to orient participants to the topic at hand. In the present study, sexual assault was defined as forced (a) vaginal or anal penetrating by an object, finger or penis; (b) oral sex; (c) touching of the participant's breasts or genitals; or (d) participant's touching of another person's genitalia. Physical assault was defined as (a) attacked or threatened with a gun, knife, or other weapon; (b) attacked by another person with perceived intent to kill or seriously injure; or (c) physically abusive punishment (see Kilpatrick et al., 2000 for the actual questions asked). An affirmative answer to questions in the sexual or physical assault module at Wave I corresponded to victim status at Wave I. New sexual or physical assault between Wave I and II corresponded to an affirmative answer to the questions that were reported at Wave II and happened at an age older than their age at Wave I assessment.

Witnessing violence was defined as directly observing someone being shot, stabbed, sexually assaulted, mugged, robbed or threatened with a weapon. Presence of witnessed serious violence for Wave I and II was coded similarly to assault history.

Psychopathology

Major Depressive Disorder – Lifetime Wave I (MDD). The depression module at Wave I data collection assessed whether participants have ever had a period of two weeks or longer during which they experienced symptoms corresponding to the MDD criteria in the DSM-IV (APA, 1994). Participants were assigned a diagnosis of MDD if they endorsed the required 5 or more symptoms. Cronbach's alpha coefficient for the depression module for this sample was .85 at Wave I, indicating good internal consistency (Kilpatrick, Ruggiero, Acierno et al., 2003).

Post-Traumatic Stress Disorder - Lifetime Waves I and II and Past Year Wave II (PTSD). A modified version of the National Women's Study (NWS) PTSD module (Kilpatrick, Resnick, Saunders & Best, 1989) was used to assess lifetime PTSD at Waves I and II and past year PTSD at Wave II. Unfortunately, age of onset was not evaluated in the Wave II lifetime PTSD assessment. This variable, then, was used to conduct a rough analysis of any PTSD onset between Waves I and II based on the assumption that those who reported lifetime PTSD history at Wave II but not at Wave I experienced the onset of PTSD since Wave I. The NWS PTSD module assesses symptoms corresponding to DSM-IV criteria for PTSD (APA, 1994) and has demonstrated good convergent validity with the Structured Clinical Interview for DSM-III-R (Spitzer, Williams, & Gibbon, 1987). Cronbach's alpha for the PTSD module for this sample at Wave I was .87, indicating good internal consistency (Kilpatrick et al., 2003).

Alcohol and Drug abuse/dependence – Lifetime Wave I. A structured clinical interview (see Kilpatrick et al., 2000 for the actual questions asked) was used to diagnose Alcohol abuse/dependence and Drug abuse/dependence, and reflected exact DSM-IV criteria for abuse or dependence for each type of substances (alcohol, marijuana, and other hard drugs). Participants were assigned a diagnosis of Alcohol abuse/dependence if they endorsed the requisite symptoms for either abuse or dependence over the past 12 months. Cronbach's alpha for the alcohol abuse/dependence module for this sample at Wave I was .95, indicating good internal consistency (Kilpatrick et al., 2003). Participants were assigned a diagnosis of Drug abuse/dependence if they endorsed the requisite symptoms for either abuse or dependence over their lifetime. Cronbach's alpha for the drug abuse/dependence module for this sample at Wave I was .84, indicating good internal consistency (Kilpatrick et al., 2003).

Statistical Analyses

In order to prospectively test predictors of PTSD following exposure to subsequent violence only participants reporting exposure to new assault or witnessed serious violence at Wave II (Weighted N = 733) were used. Wave I MDD and SUD and relevant demographic variables, including gender, ethnicity, and Wave II education status and age, were included as covariates in each multivariate regression analysis.

Participants were categorized for analyses based on their status at Wave I. In order to assess whether PTSD in response to prior trauma was predictive of PTSD in response to subsequent trauma, those with prior exposure to violence (assault and/or witnessed serious violence) and PTSD at Wave I were compared to those without such exposure and no PTSD. Given the low sample size in this prior exposure/PTSD group (Unweighted N = 100), these analyses were considered to be exploratory. In order to examine the sensitization hypothesis of traumatic event exposure, participants with prior violence without PTSD were compared to those without prior violence exposure and no PTSD. The specificity of this hypothesis by trauma type was assessed by comparing respondents with prior assault/no PTSD to those with no prior assault/no PTSD and by comparing participants with prior witnessed serious violence/no PTSD to those with no prior witnessed serious violence/no PTSD. For the last two comparisons, the analysis was conducted with both Wave I assault history and witnessed serious violence entered simultaneously in the model. This was done to assess the uniqueness of each predictor in increasing risk of PTSD following subsequent exposure.

Results

Unless otherwise noted, results reflect weighting of sample data on the basis of age, gender, and race estimates for the adolescent population of the United States in 1995 as previously described. Logistic regression analyses were conducted using the *Statistical Analysis Software (SAS) version 9.1* statistical software package.

Attrition

Data analyses were conducted to examine the extent to which attrition impacted the final sample. Of the 4,023 youth who completed Wave I of data collection, 43.5% (n = 1753) completed follow-up interviews approximately 8 years later (Wave II). Data were not collected from the remainder of the sample at Wave II for the following reasons: 1516 (66.7%) could not be located, 267 (11.7%) were located but either could not be reached or were not successfully scheduled during the assessment period, 449 (19.8%) refused to participate or terminated the interview, and 40 (1.8%) were deceased or had health problems that precluded their participation. In conclusion, difficulty locating and scheduling participants, rather than participant refusal, accounted for the majority of the observed attrition.

In order to identify attrition bias (Miller & Wright, 1995), Wave II completers and noncompleters were compared with respect to selected demographic characteristics, victimization and mental health outcome variables as measured at Wave I with weighting of the sample for representativeness to the 1995 U.S. population. When considering demographics, a greater proportion of female (46.7%) than male (40.3%) participants were completers, $\chi^2(2,N = 4023) = 16.73$, $p < .001$. Nonhispanic Caucasians (47.0%) were also more likely than ethnic minorities (35.8%) to be completers at Wave II, $\chi^2(2,N = 4023) = 41.45$, $p < .001$.

With regard to interpersonal violence, a greater proportion of those participants who did not have history of physical assault/abusive punishment at Wave I (45.0%) than those who were victims (38.2%) were completers, $\chi^2(2,N = 4023) = 13.42$, $p < .002$. Similarly, participants

who had not witnessed violence had a higher completion rate (46.3%) than those who had (39.3%), $\chi^2(2,N = 4023) = 19.35$, $p < .002$). There were no observed differences in attrition with regard to sexual assault ($\chi^2(2,N = 4023) = .30$, $p > .30$).

Finally, considering mental health outcomes, there was no difference in completion rate between those who experienced a major depressive episode (MDE) or PTSD 6 months prior to Wave I data collection and those who did not (MDD: $\chi^2(2,N = 4023) = .12$, $p > .70$; PTSD: $\chi^2(2,N = 4023) = .02$, $p > .90$). However, while there was no difference in completion rate between those who admitted to alcohol abuse/dependence in the past 12 months at Wave I data collection and those who did not ($\chi^2(2,N = 4023) = 2.26$, $p > .10$), fewer of those who admitted to drug abuse/dependence at Wave I (31.1%) were completers compared to those who did not (44.0%), $\chi^2(2,N = 4023) = 10.83$, $p < . 002$.

Eleven participants were excluded from the sample due to not answering screening questions about sexual abuse history. Seventeen participants were also missing complete data. Thus, the final unweighted sample included 1,725 participants.

Descriptive Data

Table 1 presents data that describe percentage of individuals exposed to interpersonal violence victimization among key comparison groups and past year prevalence of PTSD among those exposed. Since the focus of our analyses was on groups of individuals who had either been assaulted or witnessed serious violence, those who reported PTSD from other traumatic stressors who had not been assaulted or witnessed serious violence (Unweighted $N = 22$) were excluded from the exposure categories presented in this table and from our main analyses. The remainder of the descriptive data presented here and in Table 1 excluded these cases. Overall, the weighted data indicated that 24.2% reported an assault at Wave I, and of these 3.7% reported sexual assault, 16.3% reported physical assault, and 4.1% reported both. In addition, 37.7% witnessed serious episodes of violence at Wave I. Among this sample, 15.4% were assaulted and witnessed serious violence, while 22.3% witnessed serous violence but were not assaulted and 8.8% were assaulted but did not witness serious violence. Altogether, 46.5% reported an assault or witnessed violence at Wave I.

Further, 20.8% of the sample reported exposure to assault at Wave II that occurred since Wave I. Among these, 4.7% reported a sexual assault, 14.2% reported a physical assault, and 1.8% reported both. New witnessed violence was also reported by 35.5% of the sample. Overall, 7.2% of the sample were assaulted but did not witness serious violence, 22.0% witnessed serious violence but were not assaulted, and 13.5% reported both. In sum, 42.7% reported new assault or witnessed violence at Wave II.

Prediction of PTSD after subsequent exposure

The findings of the regression analyses are presented in Table 2. Overall, the results were consistent with our hypotheses. Prior assault in the absence of prior PTSD diagnosis predicted PTSD following exposure to new Wave II PTE. In contrast, prior witnessed violence in the absence of prior PTSD did not display similar effects. Further, PTSD in response to prior trauma did not predict PTSD in response to a subsequent stressor, though this is likely due to the low sample size for this group (Unweighted $N = 100$).

Some participants may have developed PTSD since Wave I and recovered prior to Wave II, so we also wished to consider them in our analyses. Lifetime PTSD diagnosis at Wave II was used to assess whether those who did not report PTSD at Wave I had experienced it since then. We were not able to examine conditional probability of subsequent PTSD among the prior violence/PTSD group using this method, since lifetime PTSD at Wave II would have also

included PTSD reported at Wave I. When this broader analysis of subsequent PTSD was conducted for those reporting no PTSD at Wave I, the findings were nearly identical. Among those exposed to subsequent violence, those with prior assault/no PTSD were more likely to report lifetime PTSD at Wave II than those without such histories, OR=1.93, 95% *CI* = 1.13 – 3.29, $p < .05$. However, prior witnessed serious violence/no PTSD was not predictive of lifetime PTSD reported at Wave II.

Lastly, to assess whether new trauma type influenced the findings, we also added variables on Wave II assault and witnessed serious violence to the regression models. These additions did not change the directions or significance of the main findings. Prior assault without PTSD was still found to increase risk of subsequent PTSD following new exposure when controlling for new trauma type.

Discussion

The findings of the present study suggest that prior exposure to assault increases the risk of PTSD following subsequent exposure. After entering a range of covariates, including demographic variables, prior substance use disorder, and depression, we found prior assault to be predictive of PTSD following exposure to interpersonal violence. Further, the experience of prior witnessed violence was not found to predict PTSD following new interpersonal violence exposure within a sample of individuals who had not met prior lifetime criteria for PTSD. These findings indicate that all PTEs are not the same and raise the possibility that Breslau and colleagues' (2008) findings showing no effect of prior exposure may be attributable to a diminished potency of assault history in their analyses by its classification with witnessed violence and/or other less severe events. Our data indicate that witnessed violence is very common (affecting 37.2% at Wave I) and the probability of PTSD following interpersonal violence at Wave II among those with prior witnessed violence (15.5%) is similar to that of the entire sample (with or without prior witnessed violence) following exposure to interpersonal violence (15.8%).

In the present study, PTSD in response to prior trauma was not predictive of PTSD following subsequent trauma. These findings, thus, ran counter to Breslau and colleagues' analysis. However, this discrepancy is likely due in part to low sample size among this group.

Our study differed from Breslau and colleagues in a few ways, one of the most important being the way in which PTE history was assessed and categorized. Our sample was nationally representative, whereas theirs was found in a specific region (southeast Michigan). They included four different assessment points, the first occurring between ages 21 and 30. Our sample was assessed first at an earlier age (12 – 17) and again at an average of seven to eight years later. Though we included many of the same covariates as Breslau et al., we were not able to control for history of prior anxiety disorder. It should be noted that preexisting anxiety disorder was not predictive of subsequent PTSD in their multivariate analysis. In addition, since such events were not assessed at Wave II, we did not consider presence of other traumatic stressors (e.g., accidents) in our analyses. There is little reason to suspect that the inclusion of such events would have influenced our main findings, since there were very few participants with PTSD at Wave I who had been exposed only to these other events and prior studies by our group (e.g. Resnick, Kilpatrick, Dansky, Saunders & Best, 1993) and others have consistently demonstrated higher risk of PTSD associated with interpersonal violence compared to these events. However, future studies would do well to examine whether other traumatic stressors sensitizes individuals to experience PTSD in response to subsequent interpersonal violence and whether prior assault sensitizes individuals to experience PTSD in response to other traumatic stressors. In addition, given the low sample size among those with PTSD at Wave I who were exposed to a subsequent PTE, we were only able to adequately

evaluate the possible sensitization hypothesis related to prior exposure to direct or indirect violence exposure among who did not meet prior lifetime criteria for PTSD. Therefore this study is a partial test of Breslau et al.'s findings that focuses specifically on the impact of varying types of event exposure versus non-exposure. Lastly, past year PTSD at Wave II was the focal dependent variable in our analyses, which may have only captured the more chronic PTSD cases that had their onset in the intervening years. However, additional analyses using a rough approximation of PTSD occurring since Wave I revealed nearly identical findings. To summarize, the differences between our findings and those of Breslau et al. may be due to differences in the number of assessment points, sample age, regional demographics, PTEs under consideration, or the absence of the additional preexisting anxiety disorder covariate in our analysis.

Both our and Breslau and colleagues' analyses could be considered overly conservative tests of the sensitization hypothesis. The key analysis from both studies compared those with prior PTE exposure and no PTSD to those without prior exposure in terms of whether they differed in their response to a subsequent PTE. Even though most people do not develop PTSD following exposure, the prior exposure/no PTSD group is by definition "resilient" in that they did not develop PTSD following exposure. Classifying them accordingly could be considered "stacking the deck" in comparative analyses of traumatic stress reactions. Evidence that this group is not more likely to develop PTSD following an additional PTE does not necessarily suggest that prior PTE does not act as a risk factor for PTSD in response to a subsequent PTE; it may, instead, suggest that this group possesses certain characteristics such as hardiness that help buffer the effects of PTEs (Waysman, Schwarzwald, & Solomon, 2001). Indeed, one might expect this resilient group to be less likely to develop PTSD following new interpersonal violence exposure. Some individuals in this group are unlikely to ever develop PTSD following a PTE, while others who are sensitized may require a subsequent PTE in order to develop PTSD. The fact that those with prior assault history and no PTSD in our study were more likely to develop PTSD in response to new interpersonal violence implicates a sensitization effect that is strong enough to appear in a group that has also been shown to be resilient.

A better test of this hypothesis might use prospective data and participants in environments in which PTE exposure is more uniformly distributed. For example, if the prevalence of PTSD symptoms is found to increase gradually among soldiers the longer they are in the field, and one could demonstrate this using multiple assessment points (baseline, in theatre, and following deployment), this would provide more methodologically sound support for the sensitization hypothesis. Similar studies could be carried out with police officers, firefighters, or other populations likely to be exposed to PTEs.

The findings of the present study have theoretical and clinical implications. The fact that prior assault was found to predict PTSD response to subsequent violence suggests that researchers and clinicians seeking to prevent revictimization and PTSD should pay close attention to assault history with and without posttraumatic stress response when identifying those at risk. In addition, they suggest that cumulative exposure to specific types of PTEs put people at greater risk of developing PTSD, though the exact mechanisms by which individuals could be sensitized to subsequent PTEs is not yet clear.

Acknowledgments

This research was supported by NIJ Grant No. 93-IJ-CX-0023 and CDC Grant No. R49-CCR-419810. The preparation of this paper was also partially supported by NIMH Grant No. MH18869–20, Dean G. Kilpatrick, Principal Investigator.

References

Alden LE, Regambal MJ, Laposa JM. The effects of direct versus witnessed threat on emergency department healthcare workers: Implications for PTSD Criterion A. Journal of Anxiety Disorders 2008;22:1337–1346. [PubMed: 18325730]

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. Vol. 4. Washington, DC: Author; 1994.

Breslau N, Chilcoat HD, Kessler RC, Davis GC. Previous exposure to trauma and PTSD effects of subsequent trauma: Results from the Detroit Area Survey of Trauma. American Journal of Psychiatry 1999;156:902–907. [PubMed: 10360130]

Breslau N, Davis GC, Andreski P, Peterson E. Traumatic events and posttraumatic stress disorder in an urban population of young adults. Archives of General Psychiatry 1991;48:216–222. [PubMed: 1996917]

Breslau N, Kessler RC, Chilcoat HD, Schultz LR, Davis GC, Andreski P. Trauma and posttraumatic stress disorder in the community: The 1996 Detroit Area Survey of Trauma. Archives of General Psychiatry 1998;55(7):626–632. [PubMed: 9672053]

Breslau N, Peterson EL, Schultz LR. A second look at prior trauma and the posttraumatic disorder effects of subsequent trauma. Archives of General Psychiatry 2008;65(4):431–437. [PubMed: 18391131]

Dohrenwend BP, Turner JB, Turse NA, Adams BG, Koenen KC, Marshall R. The psychological risks of Vietnam for U.S. veterans: A revisit with new data and methods. Science 2006;313:979–982. [PubMed: 16917066]

Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. Archives of General Psychiatry 1995;52:1048–1060. [PubMed: 7492257]

Kilpatrick DG, Acierno R, Saunders B, Resnick HS, Best CL, Schnurr PP. Risk factors for adolescent substance abuse and dependence: Data from a national sample. Journal of Consulting & Clinical Psychology 2000;68:19–30. [PubMed: 10710837]

Kilpatrick, DG.; Resnick, HS.; Saunders, BE.; Best, CL. The National Women's Study PTSD module. Charleston, SC: Crime Victims Research and Treatment Center, Department of Psychiatry and Behavioral Sciences, Medical University of South Carolina; 1989. Unpublished instrument

Kilpatrick DG, Ruggiero KJ, Acierno R, Saunders BE, Resnick HS, Best CL. Violence and risk of PTSD, major depression, substance abuse/dependence, and comorbidity: Results from the National Survey of Adolescents. Journal of Consulting and Clinical Psychology 2003;71:692–700. [PubMed: 12924674]

Kilpatrick, DG.; Saunders, BE. Prevalence and consequences of child victimization: Results from the National Survey of Adolescents. Charleston: Medical University of South Carolina, National Crime Victims Center; 1999.

King LA, King DW, Fairbank JA, Keane TM, Adams GA. Resilience-recovery factors in post-traumatic stress disorder among female and male Vietnam veterans: Hardiness, postwar social support, and additional stressful life events. Journal of Personality and Social Psychology 1998;74(2):420–434. [PubMed: 9491585]

King DW, King LA, Foy DW, Gudanowski DM. Prewar factors in combat-related posttraumatic stress disorder: Structural equation modeling with a national sample of female and male Vietnam veterans. Journal of Consulting and Clinical Psychology 1996;64:520–531. [PubMed: 8698946]

Koss MP. Detecting the scope of rape: A review of prevalence research methods. Journal of Interpersonal Violence 1993;8:198–222.

Miller RB, Wright DW. Detecting and correcting attrition bias in longitudinal family research. Journal of Marriage and the Family 1995;57:921–929.

Mineka S, Zinbarg R. A contemporary learning theory perspective on the etiology of anxiety disorders: It's not what you thought it was. American Psychologist 2006;61:10–26. [PubMed: 16435973]

Ozer EJ, Best SR, Lipsey TL, Weiss DS. Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis. Psychological Bulletin 2003;129:52–73. [PubMed: 12555794]

Rau V, DeCola JP, Fanselow MS. Stress-induced enhancement of fear learning: An animal model of posttraumatic stress disorder. Neuroscience and Biobehavioral Reviews 2005;29:1207–1223. [PubMed: 16095698]

Rescorla RA. Effect of inflation of the unconditioned stimulus value following conditioning. Journal of Comparative and Physiological Psychology 1974;86(1):101–106.

Resnick H, Kilpatrick D, Dansky B, Saunders B, Best C. Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. Journal of Consulting and Clinical Psychology 1993;61:984–991. [PubMed: 8113499]

Resnick HS, Yehuda R, Pitman RK, Foy DW. Effect of previous trauma on acute plasma cortisol level following rape. American Journal of Psychiatry 1995;152:1675–1677. [PubMed: 7485635]

Solomon Z, Flum H. Life events, combat stress reaction and post-traumatic stress disorder. Social Science and Medicine 1988;26(3):319–325. [PubMed: 3347854]

Spitzer, RL.; Williams, JB.; Gibbon, M. Structured Clinical Interview for DSM–III–R—Nonpatient version (SCID-NP-V). New York: New York State Psychiatric Institute, Biometrics Research Department; 1987.

Stein AL, Tran GQ, Lund LM, Haji U, Dashevsky BA, Baker DG. Correlates for posttraumatic stress disorder in gulf war veterans: a retrospective study of main and moderating effects. Journal of Anxiety Disorders 2005;19:861–76. [PubMed: 16243635]

Stein MB, Jang KL, Taylor S, Vernon PA, Livesley WJ. Genetic and environmental influences on trauma exposure and posttraumatic stress disorder symptoms: A twin study. American Journal of Psychiatry 2002;159:1675–1681. [PubMed: 12359672]

Waysman M, Schwarzwald J, Solomon Z. Hardiness: An examination of its relationship with positive and negative long term changes following trauma. Journal of Traumatic Stress 2001;14(3):513–548.

Yehuda R, Kahana B, Schmeidler J, Southwick SM, Wilson S, Giller EL. Impact of cumulative lifetime trauma and recent stress on current posttraumatic stress disorder symptoms in Holocaust survivors. American Journal of Psychiatry 1995;152:1815–1818. [PubMed: 8526254]

Table 1

Exposure to violence and conditional probability of past year PTSD at Wave II across subgroups.

	Unweighted N	**Weighted % exposed to assault or witnessed serious violence at Wave II**	**Weighted % past year PTSD among exposed**
Prior assault or witnessed violence/PTSD	100	62.4	26.4
Prior assault or witnessed violence/No PTSD	704	52.5	17.3
Prior assault/No PTSD	350	55.2	21.9
Prior witnessed violence/No PTSD	569	54.4	15.5
No prior assault or witnessed violence/No PTSD	899	33.3	11.2
Female	890	36.1	21.4
Male	813	49.2	11.3
White/non-Hispanic	1259	39.3	14.6
Non-white	444	51.5	17.5
W1 Lifetime MDD	292	48.9	27.7
W1 Lifetime SUD	142	62.7	17.7
Total sample	1703	42.7	15.4

PTSD, posttraumatic stress disorder; MDD, major depressive disorder; SUD, substance use disorder.

Table 2

Relative risk of past year PTSD at Wave II among those reporting exposure to new assault or witnessed violence since Wave I (n=733).

	Bivariate Associations OR (95% CI)	Multivariate Model Adjusted OR (95% CI)
Prior assault or witnessed violence/ PTSD vs no prior Exposure	2.69 (1.33 – 5.44)**	1.89 (0.68 – 5.26)
Prior assault or witnessed violence/ no PTSD vs no prior Exposure	1.64 (0.99 – 2.73)	1.71 (0.99 – 2.95)
Prior assault/no PTSD vs no prior assault/no PTSD	2.11 (1.25 – 3.56)**	2.03 (1.10 – 3.76)*[a]
Prior witnessed violence/no PTSD vs no prior witnessed violence/no PTSD	1.11 (0.67 – 1.85)	0.98 (0.54 – 1.79)[a]

OR=Odds Ratio; CI=Confidence Interval. Multivariate models were adjusted for gender, ethnicity, Wave I alcohol/drug abuse or dependence, Wave I lifetime depression, and Wave II age and education.

[a]Variables for both Wave I assault and witnessed serious violence were included as predictors in these regression models.

*$p < .05$,

**$p < .01$..