

Analysis of Risk Factors for Posttraumatic Stress Disorder in  
Trauma

Brewin, Chris; Andrews, Bernice; Valentine, John D.

Abstract

Meta-analyses were conducted on 14 separate risk factors for posttraumatic stress disorder (PTSD), and the moderating effects of various sample and study characteristics, including civilian/military status, were examined. Three categories of risk factor emerged: Factors such as gender, age at trauma, and race that predicted PTSD in some populations but not in others; factors such as education, previous trauma, and general childhood adversity that predicted PTSD more consistently but to a varying extent according to the populations studied and the methods used; and factors such as psychiatric history, reported childhood abuse, and family psychiatric history that had more uniform predictive effects. Individually, the effect size of all the risk factors was modest, but factors operating during or after the trauma, such as trauma severity, lack of social support, and additional life stress, had somewhat stronger effects than pretrauma factors.

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Originally, posttraumatic stress disorder (PTSD) was conceptualized as a normal response to overwhelming psychic trauma. Partly on the basis of accumulating evidence for wide variation in the prevalence of PTSD following exposure to different kinds of stressor, there is increasing acceptance of the idea that exposure to a trauma may not always be sufficient to explain the development of PTSD and that individual vulnerability factors have a role to play in understanding this condition (e.g., Yehuda, 1999; Yehuda & McFarlane, 1995). The impact on later PTSD of trauma intensity, pretrauma risk factors, and other aspects of vulnerability has been reviewed by, among others, Shalev (1996). Shalev summarized the results of 38 studies and identified numerous risk factors for PTSD, including pretrauma vulnerability (e.g., family history of mental disorders, gender, genetic and neuroendocrine factors, personality traits, early traumatization, negative parenting experiences, and lower education), the magnitude of the stressor, preparation for the event, immediate reactions to the trauma (e.g., dissociation and coping responses), and posttrauma factors (e.g., emerging symptoms, social support, and other life stress).

Much of the evidence that has formed the basis of previous reviews has derived from studies of combat veterans and may not be generalizable to civilian samples. Moreover, the studies are extremely heterogeneous (see discussion below) and results for individual risk factors have not always been consistent

(e.g., Bremner, Southwick, & Charney, 1995). In this situation, with a relatively large number of relevant studies varying simultaneously along several dimensions, an accurate summary may best be given using meta-analytic techniques. Previous meta-analyses in the trauma field have examined the general psychological impact of military service in Vietnam (Kaylor, King, & King, 1987), disasters (Rubonis & Bickman, 1991), or interpersonal violence (Weaver & Clum, 1995). Neither risk factors nor the specific diagnosis of PTSD have yet been studied in this way.

Given the current debate concerning the relative importance of the trauma itself versus other vulnerability factors in the development of PTSD, and the need to build comprehensive models of PTSD that take into account the rapidly growing numbers of empirical studies, it would be valuable to have more broadly based quantitative estimates of the absolute and relative effect sizes associated with the various possible risk factors. In this article, we present a series of meta-analyses of currently available studies yielding effect sizes for a variety of risk factors for PTSD and examine whether these effect sizes are homogeneous or whether they are affected by characteristics of the study or the sample.

In attempting to summarize the literature on risk factors for PTSD, several sources of variability should be acknowledged at the outset. These can be considered under the headings of design, sampling, and measurement. First, there have been relatively few prospective studies of PTSD that have measured risk factors before the traumatic event (one exception is Macklin et al., 1998), although others have measured risk factors posttrauma but prior to the onset of PTSD (e.g., Andrews, Brewin, Rose, & Kirk, 2000). Much research has attempted to infer what were risk factors by comparing a group of people who have already contracted PTSD with a control group (e.g., Kelly et al., 1998). This raises the issue of whether the presence of PTSD biases estimates of premorbid personality and intelligence or affects retrospective reports of childhood history or trauma severity. Although there is reassuring evidence that some types of psychopathology (such as depression) do not typically bias reports of childhood experience (Brewin, Andrews, & Gotlib, 1993), little is known about the possible impact of PTSD on such reports. It will be important to investigate, where possible, whether prospective and retrospective designs yield similar estimates of effect size.

Studies vary on several dimensions other than the longitudinal-retrospective one. The modal strategy adopted by investigators has been to examine the relation of risk factors to PTSD in a population of people all exposed to trauma. However, some studies have reported risk factors for PTSD in trauma survivors based on comparisons with controls who may not have been exposed to trauma, an approach that seeks to predict the joint risk of trauma exposure and the development of subsequent PTSD (e.g., Wolfe et al., 1998). Other studies have compared risk factors for PTSD versus other clinical conditions in a

trauma-exposed sample, thereby attempting to predict what is unique about posttraumatic versus other symptoms of psychopathology (e.g., Watson, Davenport, Anderson, Mendez, & Gearhart, 1998). Still other studies have reported predictors of symptom severity within a sample who all have either PTSD or some other psychiatric disorder, thereby focusing on gradations of disorder within a clinical population (e.g., McFall, Mackay, & Donovan, 1991). Thus, investigators have addressed a number of equally valid but quite separate questions, and it cannot be assumed that the risk factors identified will be consistent across these different designs. In practice, there are only enough studies using the first, modal strategy to justify a meta-analysis.

The samples in which PTSD has been studied are varied, including the general population, combat veterans, assault and rape victims, victims of motor vehicle and other types of accidents, victims of natural disasters, and people suffering from life-threatening medical conditions. By far the largest group studied is male combat veterans; in contrast, civilian studies have frequently focused on female trauma victims or have used samples in which both genders were well represented (e.g., Nishith, Mechanic, & Resick, 2000; Ursano et al., 1999). It is important to investigate whether risk factors discovered in combat veteran studies, or in male samples, generalize to civilian and female samples.

Apart from demographic factors that are routinely measured, there has been little consistency in the risk factors investigated in the different studies or in the measures used to assess them. Measures of trauma severity are reasonably consistent in studies of combat veterans, which have increasingly tended to focus on specific factors, such as high levels of perceived threat, exposure to atrocities or episodes of extraordinarily abusive violence, and on a general milieu of a harsh or malevolent environment (e.g., King, King, Gudanowski, & Vreven, 1995). In contrast, measures of trauma severity in the civilian studies vary widely, both between different types of trauma and also within trauma types. This necessary heterogeneity is likely to decrease the reliability of effect size estimates, although the finding of consistent associations across different trauma types would tend to increase confidence in their validity.

Another source of variability is the measure of PTSD used in the different studies. This typically consists of a dichotomous variable reflecting whether or not the person meets formal diagnostic criteria for the disorder laid down in the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (3rd ed. [DSM-III; American Psychiatric Association, 1980]; 3rd ed., rev. [DSM-III-R; American Psychiatric Association, 1987]; 4th ed. [DSM-IV; American Psychiatric Association, 1994]). Although remaining largely consistent, the diagnosis has changed somewhat in emphasis with succeeding versions of the DSM, particularly with the importance given to the impairment criterion in the DSM-IV. It is possible that these changes will impact on risk factor estimates, although too few studies have used DSM-IV criteria to test this empirically. Occasionally, some investigators (e.g.,

Solkoff, Gray, & Keill, 1986) have included together in their analyses participants meeting full diagnostic criteria for PTSD with those suffering from partial PTSD, and again there is no guarantee at present that the risk factors for the two conditions are the same.

Diagnoses may also be current or lifetime. It is conceivable that studies requiring a current diagnosis will contain a higher proportion of chronic cases and that risk factors for chronic PTSD are somewhat different to those for brief episodes of the disorder. A comparison of effect sizes for studies using current and lifetime diagnoses would be helpful in this regard. However, in practice there are two limitations to this strategy. First, studies of current or lifetime PTSD only rarely report the length of time symptoms have been present. Second, many of the most important studies assessing lifetime PTSD in adults have used large epidemiological samples, have assessed a variety of different trauma types, and have not distinguished PTSD arising as a result of traumas in childhood and in adulthood (e.g., Breslau, Davis, Andreski, & Peterson, 1991; Breslau et al., 1998). This subgroup differs in these respects from most of the other studies included in this analysis, which have smaller sample sizes and focus on a single type of trauma occurring in adulthood. Because of the size of these studies, and their consequent weighting in the meta-analysis, it is only feasible to compare this subgroup with the remaining studies rather than examine the current-lifetime distinction.

Another sizeable group of studies (e.g., Koopman, Classen, & Spiegel, 1994) used continuous measures covering the range of PTSD symptoms, either correlating these measures with risk factors or using cutoff scores to divide their samples into groups with and without PTSD. Measures such as the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder (Keane, Caddell, & Taylor, 1988), the Posttraumatic Symptom Scale (Foa, Riggs, Dancu, & Rothbaum, 1993), or the Impact of Events Scale-Revised (IES-R; Weiss & Marmar, 1996) are suitable for this purpose. However, another frequently used measure of individual PTSD symptoms, the original IES (Horowitz, Wilner, & Alvarez, 1979), is less suitable because it does not include arousal symptoms. Thus, although it is often highly correlated with a PTSD diagnosis, there is no guarantee that its relationship to potential risk factors will be the same. Finally, it should be considered that both diagnoses and continuous measures of PTSD symptoms may be based on interviews or questionnaires. It would be reassuring to know that this study characteristic did not affect risk factor estimates.

The study characteristics reviewed above place a number of constraints on the extent of any meta-analysis that can be undertaken. To begin with, it was necessary to identify subsets of studies, each subset having in common the measurement of a specific risk factor and each requiring its own meta-analysis. Each analysis was based on a different group of studies that overlapped to differing degrees with studies appearing in analyses of other risk factors. The

measures used to assess each risk factor may be different and have differing (and usually unknown) reliability and validity. Caution must therefore be exercised in interpreting the results of the meta-analyses. On the positive side, aggregation across studies produces substantial sample sizes and thereby increases reliability. Moreover, studies typically measure and report results for a number of risk factors, some showing a significant association with outcome and others showing no association. In contrast to the typical treatment trial, where only the effect of treatment is of interest, risk factor studies are therefore substantially less prone to the "file drawer" problem (the tendency for positive findings to be more likely to be published than negative findings).

In this article, we examined studies that have investigated populations exposed to trauma in adulthood in order to detect risk factors for PTSD. Fourteen risk factors were included: gender, age at trauma, socioeconomic status (SES), education, intelligence, race, previous psychiatric history, reported abuse in childhood, reports of other previous traumatization, reports of other adverse childhood factors (excluding abuse), family history of psychiatric disorder, trauma severity, posttrauma life stress, and posttrauma social support. Although it has been sometimes claimed that female gender is a risk factor for PTSD, this conclusion partly rests on studies of women who have been raped or sexually assaulted, traumas carrying a particularly high risk of PTSD. Following the recommendations of Wolfe and Kimerling (1997), we therefore compared rates of PTSD for men and women similarly exposed to a variety of stressors. Finally, we examined the impact of the following six sample and study characteristics on the effect sizes obtained for the 14 risk factors: (a) type of trauma (military vs. civilian), (b) gender of participants, (c) retrospective versus prospective design, (d) analyses based on presence or absence of diagnosis versus continuous symptom scores, (e) PTSD assessed with interview or questionnaire, and (f) traumas that may have occurred in childhood or adulthood versus in adulthood only.

## Method

### Selection of Articles

English-language articles published since 1980 and including data on predictive or risk factors for PTSD (defined in a way consistent with the DSM-III, the DSM-III-R, or the DSM-IV) were located from bibliographic databases, recent journal issues, and secondary sources such as review articles and book chapters. The Social Science Citation Index and the PILOTS database managed by the National Center for PTSD were searched using the terms PTSD and risk, predictor or prediction, or predisposition. Articles were excluded on any of the following grounds: (a) The study did not specifically assess the reexperiencing, avoidance, and arousal symptoms of PTSD as described in the above diagnostic criteria; (b) the study included in the same comparison group

individuals meeting full diagnostic criteria for PTSD and individuals with less severe posttraumatic symptoms or partial PTSD and contrasted them with a non-PTSD group; (c) the study population consisted entirely of individuals already suffering from PTSD or from other psychiatric disorders; (d) the study did not report analyses on individuals who had been all exposed to a similar trauma in adulthood (but note that studies that assessed several different types of trauma simultaneously were included); or (e) the study did not provide sufficient data to calculate relevant effect sizes. Two raters independently coded 20 articles to check on the reliability of inclusion judgments and were in agreement on 95% of occasions ( $\kappa = .90, p < .001$ ).

As noted above, we found 10 articles based on large epidemiological samples of adults that permitted the index trauma to have occurred either in childhood or in adulthood (Acierno, Resnick, Kilpatrick, Saunders, & Best, 1999; Breslau, Chilcoat, Kessler, & Davis, 1999; Breslau et al., 1991, 1998; Bromet, Sonnega, & Kessler, 1998; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Kilpatrick et al., 1989; North & Smith, 1992; Resnick, Kilpatrick, Best, & Kramer, 1992; Widom, 1999). Given the importance of these studies, they were included in the meta-analysis, but we report subsidiary analyses comparing their conclusions with those of the remaining studies. We also found that 11 articles reported only multivariate estimates of effect size, typically derived from logistic or multiple regressions, use of which could result in the impact of risk factors being underestimated. In all of these cases, authors were requested to supply corresponding univariate data, and these were used in the analyses when available. Four studies for which univariate data were unavailable were omitted from the analysis.

These procedures left a group of 77 articles. Six articles (Acierno et al., 1999; Bromet et al., 1998; King, King, Foy, Keane, & Fairbank, 1999; North & Smith, 1992; North, Smith, & Spitznagel, 1994; Steinglass & Gerrity, 1990) reported results for men and women separately, or for separate subsamples, and as a result there are a total of 85 separate data sets in the meta-analysis.

## Procedure

### Risk factor estimates.

Risk factors were included in the meta-analysis if they had been assessed in at least four of the selected articles: The 14 factors are shown in Table 1. Whenever the same risk factors had been assessed within a study using more than one measure (e.g., several separate aspects of previous psychiatric history might have been assessed), effect sizes were averaged across these measures. Different articles reporting analyses from the same data set were included so long as they provided estimates for different risk factors and were checked to ensure there was no duplication of data. Where data were duplicated, estimates

from the largest sample or from the most comprehensive article were used. In studies using matched groups, demographic matching variables were not included in the meta-analysis. Additional risk factors that occurred rarely (e.g., neuroticism), or that overlapped significantly with the measurement of PTSD (e.g., early symptoms and acute stress disorder), were not included in the meta-analysis but are discussed later in this article.

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Table 1 Summary of Risk Factors Predicting PTSD  
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Studies using group designs reported *t*, *F*, chi-square, and Mann-Whitney *U* statistics, whereas studies using correlational designs reported Pearson's *r* or Spearman's  $\rho$ . Following the procedures described by Rosenthal (1991), all of the statistics were converted to the correlation coefficient *r* in order to yield a common measure of effect size. These *r*s were then combined using Fisher's *zr* transformation and were weighted by their degrees of freedom ( $n - 3$ ) in order to take into account the differential precision of estimate associated with larger and smaller studies. Finally, the Fisher's *zr* values were converted back to *r* to yield a weighted average effect size for each risk factor. Higher values of *r* indicate a stronger positive association with PTSD.

#### Sample and study characteristics.

The six characteristics examined are listed above at the end of the introduction. Interrater reliability was checked using a further sample of 20 articles and was found to be high for all judgments (agreement = 90%–100%,  $[\kappa] = .69 - 1.00$ ). Moderator effects were evaluated in the following way. The relevant subsets of studies (e.g., studies investigating the effect of social support in military vs. civilian samples) were combined, once again using Fisher's *zr* transformation and converting back to yield a weighted average *r*. These were then compared using two-tailed *t* tests, which employed large-sample standard errors for the two *r*s and Satterthwaite's approximation via weighted degrees of freedom to the distribution of *t* when these two standard errors differed and so had to be separately estimated. As the sample and study characteristics were all dichotomous, correlations between them were assessed using the phi coefficient.

## Results

### Characteristics of the Articles

Sample size varied from 25 to 4,127, with a median of 119. Of the 77 articles included in the meta-analysis, 28 were based on military samples of service personnel enduring war (presence in a war zone, combat, or imprisonment) and 49 articles were based on civilian samples: 13 on crime victims (1 article

contained two separate subsamples), 9 on disaster victims (1 article contained two separate subsamples), 4 on motor vehicle accident (MVA) victims, 6 on burns victims, 7 on other specific groups such as victims of terrorist attacks, and 10 on victims of mixed traumas. Twenty-six articles were based on samples of men, 12 articles were based on samples of women, and 40 articles were based on mixed-gender samples, for which 4 articles reported results for men and women separately. Inspection of the Appendix shows that the age span of the samples was wide, with all ages from 18 upward represented. Fifty-four percent of the articles reported on the race of their respondents, and in this group the median level of representation of minority groups was 25% (range = 0%–75%). Fifty-nine articles were purely retrospective, and 18 were at least partly longitudinal (measuring either pretrauma variables or trauma variables that preceded the development of PTSD). Risk factor analysis was based on the presence or absence of a PTSD diagnosis in 53 articles and a cutoff score on a continuous measure in 5 articles; 19 articles conducted analyses using the full range of scores on a continuous measure. Fifty articles measured PTSD with an interview, 26 measured PTSD with a questionnaire, and 1 combined both methods. Finally, in 19 articles assessment of PTSD was based on the DSM-III, in 48 articles assessment of PTSD was based on the DSM-III-R, in 4 articles assessment of PTSD was based on the DSM-IV, and in the remaining 6 articles no specific DSM diagnosis was specified.

### Risk Factor Estimates

Effect sizes were combined using the methods described above within each subset of studies to provide estimates of the strength of each predictive effect. For each risk factor, Table 1 shows the number of studies, the total number of individuals involved, the range of the observed effect sizes, the combined effect size  $r$ , an overall test of significance based on the standard normal deviate  $z$ , and a chi-square test of heterogeneity. Table 1 shows that all of the predictors were highly significant statistically but varied considerably in their effect size. The three factors relating to events during and after the trauma (i.e., greater trauma severity, lack of social support, and more subsequent life stress) convey the strongest risk of PTSD, with effect sizes that are individually small to moderate in Cohen's (1988) terms. Next, there was a group of demographic and prior history variables that had similar effect sizes in the range of .10 to .19, including female gender; lower SES; less education; lower intelligence; a positive psychiatric history; a reported history of abuse, other trauma, or childhood adversity; and a family psychiatric history. These individual effect sizes are generally regarded as small, according to Cohen. Finally, two variables, younger age at trauma and race (minority status), had effect sizes that were weaker still, although highly significant because of the large numbers involved.

A method has been recommended by Rosenthal (1991) to assess the robustness of effect sizes against sampling bias introduced by the tendency to selectively publish positive results and leave negative results in researchers' file drawers. This method involves calculating the number of unpublished studies with null results that would be necessary to reduce the observed effect size to a level where it was statistically marginal. Rosenthal suggested that if this value is greater than  $5k + 10$  (where  $k$  is the number of studies in the meta-analysis), it can be assumed that the result is unlikely to be overturned by the possible existence of studies with contrary findings. Applying this method to the above effect sizes indicated that all the findings were robust, with one exception. In the case of age at trauma, 103 studies would be needed to overturn the result, which failed to exceed the value of  $5k + 10$  (155).

Finally, it should be noted that the heterogeneity statistics given in Table 1 indicate that the majority of the effect sizes were not uniform but varied significantly across studies. In other words, the risk factors do not always predict equally well. This variability may be random or systematic, and we examined whether it was explicable in terms of sample and study characteristics (see below). The only significant predictors to show relatively homogeneous effects were psychiatric history, childhood abuse, and family psychiatric history.

#### Gender as a Risk Factor Across Trauma Types

In view of the possible artifactual reasons previously discussed for female gender being a risk factor for PTSD, we examined the combined effect size for gender, weighted by study degrees of freedom, within the different populations studied. These analyses, conducted on the subset of studies that included both men and women, are shown in Table 2. It is evident that the effect size for the two military samples ( $r = .00$ ) was not significant ( $z = .04$ ,  $p \geq .05$ ). It was also considerably smaller than in the civilian samples combined ( $r = .13$ ), and Table 3 shows that this difference was significant. Although there was a particularly strong effect size for victims of other specific traumas, all groups of civilian victims showed a consistent pattern for women to be at higher risk than men.

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Table 2 Effect of Female Gender on Risk of PTSD by Different Trauma Types  
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Table 3 Comparison of Effect Sizes for Military and Civilian Studies  
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Effects of Sample and Study Characteristics

We investigated whether the effect sizes associated with the risk factors shown in Table 1 differed depending on the characteristics for which studies had been coded (i.e., whether these characteristics operated as moderator variables).

#### Military versus civilian samples.

This distinction represents the single largest and most important division of the literature on the prediction of PTSD, and it accounted for the greatest number of significant moderator effects. A detailed breakdown of effect sizes according to whether studies had been conducted with military or civilian samples is therefore shown in Table 3. Whereas, as noted above, female gender was a less important risk factor in military samples, younger age at trauma, lack of education, race (minority status), childhood adversity, trauma severity, and lack of social support were all significantly more important in this group. The only risk factor that was no longer significant, other than gender in the military samples, was age at trauma in the civilian samples ( $z = .72$ ,  $p \leq .05$ ). Heterogeneity statistics reported in Table 3 also indicate that the effect of lack of education is homogeneous once military versus civilian status is taken into account. It is apparent that the effects of a number of demographic variables, such as female gender, lower SES, and race (minority status), are more likely to be homogeneous across military studies than across civilian studies.

#### Gender.

We next examined the subset of studies conducted exclusively on male or female samples to test whether there was any evidence for gender moderating the effect of the other risk factors. The effect size for younger age at trauma was significantly greater among men than among women ( $r = .17$  vs.  $.07$ ),  $t(2195) = 2.61$ ,  $p \leq .01$ . This was also true of the effect size for lower SES ( $r = .14$  vs.  $.05$ ),  $t(3317) = 2.92$ ,  $p \leq .01$ , and for race (minority status;  $r = .11$  vs.  $.00$ ),  $t(2636) = 2.91$ ,  $p \leq .01$ . There were no other effects of gender (largest  $t = 1.59$ ,  $p \leq .05$ ). All of the risk factor effects remained significant, with the exception of race in female samples ( $z = .04$ ,  $p \leq .05$ ). It should be noted that in the meta-analysis all-male samples were much more likely to have been features in military studies than in civilian studies ( $\phi = .69$ ,  $p \leq .001$ ).

#### Prospective versus retrospective design.

We next examined the moderating effect of prospective versus retrospective designs on the set of risk factors. Retrospective designs were associated with a weaker effect size for female gender ( $r = .11$  vs.  $.16$ ),  $t(4306) = 2.07$ ,  $p \leq .05$ , but with a stronger effect size for younger age at trauma ( $r = .07$  vs.  $.45$ ),  $t(1127) = 2.62$ ,  $p \leq .01$ , and trauma severity ( $r = .24$  vs.  $.17$ ),  $t(2626) = 2.85$ ,  $p \leq .01$ . There were no other effects of design (largest  $t =$

1.81,  $p \leq .05$ ). All of the risk factor effects remained significant, with the exception of age at trauma in prospective studies ( $z = .63$ ,  $p \leq .05$ ). In the meta-analysis, studies using retrospective designs were more likely to have been conducted on military samples ( $[\phi] = .25$ ,  $p \leq .05$ ) and on all-male samples ( $[\phi] = .37$ ,  $p \leq .001$ ).

Analysis by diagnostic category versus continuous score.

For these analyses, the small number of studies using a cutoff score on a continuous measure were omitted. Continuous measures were associated with a weaker effect size for female gender ( $r = .04$  vs.  $.13$ ),  $t(911) = 2.29$ ,  $p \leq .05$ . Continuous measures were, however, associated with stronger effect sizes for younger age at trauma ( $r = .17$  vs.  $.02$ ),  $t(4223) = 5.72$ ,  $p \leq .001$ ; lack of education ( $r = .27$  vs.  $.09$ ),  $t(619) = 4.20$ ,  $p \leq .001$ ; previous trauma ( $r = .18$  vs.  $.09$ ),  $t(4310) = 3.05$ ,  $p \leq .01$ ; childhood adversity ( $r = .32$  vs.  $.13$ ),  $t(3767) = 7.32$ ,  $p \leq .001$ ; and trauma severity ( $r = .40$  vs.  $.16$ ),  $t(6531) = 12.89$ ,  $p \leq .001$ . There were no other effects of diagnosis versus continuous measures (largest  $t = 1.81$ ,  $p \leq .05$ ). All of the risk factor effects remained significant, with the exception of gender in studies using continuous measures ( $z = 1.18$ ,  $p \leq .05$ ) and age at trauma in studies based on diagnostic categories ( $z = 1.50$ ,  $p \leq .05$ ). In the meta-analysis, this variable was unrelated to any of the other moderator variables examined (largest  $[\phi] = .19$ ,  $p \leq .05$ ), except for use of interviews versus questionnaires ( $[\phi] = .51$ ,  $p \leq .001$ ).

Assessment of PTSD by interview versus questionnaire.

Interview methods were associated with stronger effect sizes for female gender ( $r = .14$  vs.  $.05$ ),  $t(3647) = 4.10$ ,  $p \leq .001$ ; younger age at trauma ( $r = .05$  vs.  $-.03$ ),  $t(3461) = 2.59$ ,  $p \leq .01$ ; and trauma severity ( $r = .26$  vs.  $.16$ ),  $t(7355) = 5.10$ ,  $p \leq .001$ . Questionnaire methods were associated with a stronger effect size for previous trauma ( $r = .24$  vs.  $.09$ ),  $t(851) = 3.42$ ,  $p \leq .01$ . There were no other effects of interview versus questionnaire assessment of PTSD (largest  $t = 1.83$ ,  $p \leq .05$ ). All of the risk factor effects remained significant, with the exception of age at trauma in questionnaire studies ( $z = 1.18$ ,  $p \leq .05$ ). In the meta-analysis, method of assessment was strongly associated with the use of diagnostic versus continuous measures in the analysis but was unrelated to all the other moderator variables examined (largest  $[\phi] = .19$ ,  $p \leq .05$ ).

Inclusion of adult and childhood traumas versus traumas in adulthood only.

The subset of epidemiological studies that included PTSD arising from events in childhood demonstrated stronger effect sizes for female gender ( $r = .15$  vs.  $.08$ ),  $t(9317) = 3.61$ ,  $p \leq .001$ , but weaker effect sizes for lack of education ( $r = .08$  vs.  $.14$ ),  $t(6824) = 3.06$ ,  $p \leq .01$ ; previous psychiatric

history ( $r = .09$  vs.  $.15$ ),  $t(5892) = 2.26$ ,  $p \leq .05$ ; previous trauma ( $r = .06$  vs.  $.16$ ),  $t(4004) = 3.30$ ,  $p \leq .01$ ; childhood adversity ( $r = .12$  vs.  $.27$ ),  $t(6400) = 6.16$ ,  $p \leq .001$ ; and trauma severity ( $r = .16$  vs.  $.24$ ),  $t(1669) = 2.81$ ,  $p \leq .01$ . There were no other effects of including childhood events (largest  $t = 1.58$ ,  $p \leq .05$ ). All of the risk factor effects shown in Table 1 remained significant. In the meta-analysis, inclusion of childhood events was positively associated with studying civilian rather than military samples ( $[\phi] = .32$ ,  $p \leq .01$ ), with analyses based on diagnosis ( $[\phi] = .28$ ,  $p \leq .01$ ) and with interview assessments of PTSD ( $[\phi] = .30$ ,  $p \leq .01$ ).

## Summary.

A summary of the effects of the different sample and study characteristics on each of the risk factors is given in Table 4. Table 4 divides the risk factors into three groups: those that were no longer significant in some subsets of studies, those that always remained significant but to varying degrees, and those with significant and homogeneous effects as reported in Table 1.

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Table 4 Summary of Whether Sample and Study Characteristics Impact on Risk Factor Effect Sizes  
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## Discussion

Our results identify several important trends emerging from the rapidly growing number of studies reported since 1980. The meta-analyses on the various risk factors included results from 77 articles and involved combined sample sizes ranging from 1,149 to over 11,000. These large samples provided confirmation that, when soldiers and civilians exposed to traumatic events are included together, the risk of PTSD is enhanced reliably, but to a relatively small extent, by the effects of female gender; greater social, educational, and intellectual disadvantage; psychiatric history; and various types of previous adversity, as well as by somewhat larger effects attributable to factors occurring during or after the trauma.

However, the data also caution that effects tend not to be uniform across studies, with the exception of those for psychiatric history, childhood abuse, and family psychiatric history. In most cases this heterogeneity is a matter of degree and does not affect the statistical significance of the effect. But for three demographic variables, gender, age at trauma, and race, the effect disappears completely in certain subsets of studies. This has two major implications for future research. First, it suggests that attempts to identify a common set of pretrauma predictors of PTSD that will be equally valid across different traumatized groups are premature. Second, to understand vulnerability to PTSD, it will be necessary to discover whether this heterogeneity has

substantive origins or whether it can be traced to methodological differences between studies. The meta-analysis has made a useful contribution to this process by investigating the moderating effects of a number of sample and study characteristics, although in some cases the number of studies involved in the comparisons is very limited.

There were numerous differences between studies based on military versus civilian samples. For example, the gender effect, substantial among civilians, was nonexistent among combat veterans. This could reflect a greater exposure to traumatic situations among male veterans than among female veterans, which counteracted women's greater vulnerability. Alternatively, there may be important demographic differences between male and female veterans. Data were only available from two military studies, however, and conclusions about gender effects in the military await further research. Interestingly, the National Vietnam Veterans Readjustment Study also reported lower rates of PTSD in female Vietnam War veterans than in male Vietnam War veterans, the female advantage being substantially greater for current diagnoses than for lifetime diagnoses (Weiss et al., 1992). This article was not included in the meta-analysis because the majority of female veterans were nurses, whereas the majority of male veterans were combat soldiers, so that their trauma exposure is likely to have been dissimilar.

In contrast, although there was significant heterogeneity in both military and civilian groups, younger age at trauma was only a risk factor in the military. Lack of education, childhood adversity, trauma severity, and lack of social support all demonstrated relatively stronger effect sizes in military studies. One reason could be that military studies are more likely to have samples with chronic PTSD, and these variables are good predictors of chronicity.

Race (minority status) was one of the few demographic variables to be a weak predictor across all of the studies, but it should be noted that race was coded dichotomously (White majority vs. minority groups) and included a number of quite diverse groups in the minorities category. It would be inadvisable to draw any conclusions about the role of race given these severe limitations, but it is relevant that it was a significantly stronger predictor in military samples than in civilian samples and did not predict PTSD at all in female samples. Two studies in the meta-analysis that investigated the relative roles of race and combat exposure both found that minorities tended to have more exposure to war trauma and that the apparent higher risk of PTSD associated with minority status was no longer significant when exposure was controlled (B. L. Green, Grace, Lindy, & Leonard, 1990; MacDonald, Chamberlain, & Long, 1997). Similarly, there was a greater risk of PTSD associated with minority status in the Detroit Area Survey of Trauma (Breslau et al., 1998) that was no longer significant when other variables were controlled. Other studies have found similar effects (see Resick, in press, and Frueh, Brady, & de Arellano, 1998, for reviews). These results undermine further what very limited evidence

there is for race itself being a risk factor.

The impact of trauma severity was significantly greater among combat veterans than among civilian trauma victims. This finding should be treated with caution, bearing in mind that the military studies were unlike the civilian studies in being predominantly based on male samples and that measures of trauma severity were very different. In particular, measures of trauma severity were much more disparate in the civilian studies, and the effect size for trauma severity varied considerably. The effect size was in the range of .20 to .26 for crime, disaster, and MVA victims but only in the range .10 to .14 for the victims of burns and other specific traumas.

There was a general trend for many of the pretrauma variables to show more homogeneous and larger predictive effects in combat veterans as compared with civilian trauma victims. The data clearly warn against assuming that effects will be constant across military and civilian samples and that a single risk factor model for PTSD can be created. These effects are noteworthy because of the enormous amount of research in PTSD that is based on male combat veterans. These studies overwhelmingly used retrospective designs, so that in looking at the impact of gender and design as moderator variables, there will inevitably be confounding with military/civilian status.

The gender effects clearly answered one of the questions raised by Wolfe and Kimerling (1997) in that, when men and women were directly compared within the same study, women were more at risk of developing PTSD despite type of trauma being held constant. At this time, it is unclear whether in civilian samples gender constitutes a risk factor per se or whether this effect is a product of other characteristics such as levels of symptom reporting (women being more willing to report than men) or greater exposure to prior trauma. For example, it has been argued that women's greater exposure to child sexual abuse and other sexual assaults may account for their excess of PTSD in adulthood (Wolfe & Kimerling, 1997). Breslau, Davis, Andreski, Peterson, and Schultz (1997) investigated this possibility using data from their epidemiologic survey but found that the excess of PTSD among women could not be attributed to them experiencing more trauma or different kinds of trauma in childhood. Rather, after controlling for trauma exposure, Breslau et al. showed that women were more likely than men to develop PTSD following childhood trauma. Consistent with this finding, our meta-analysis indicated a greater effect size for female gender when studies included childhood traumas than when they focused exclusively on adult traumas.

When gender itself was investigated as a moderator variable, we found that studies of men yielded significantly larger effect sizes for younger age at trauma, lower SES, and race (minority status) than did studies of women. In the case of age at trauma and race, this is likely to be because of confounding between gender and military/civilian status.

As noted above, trauma intensity and posttrauma variables appeared to have somewhat larger effects on liability to PTSD than did pretrauma variables. A major concern is that retrospective reporting inflates the effects of these variables. This is difficult to establish conclusively because asking about trauma intensity too soon after the event, when individuals still feel numb and unable to appraise what has happened, might produce underestimates of trauma intensity. What can be stated is that effect sizes for trauma severity in retrospective studies are significantly greater than in prospective studies, a difference that clearly demands further investigation. The moderating effect of design could not be adequately tested for social support and life stress because of the small number of prospective studies (one and two studies, respectively).

Interestingly, although the number of prospective studies was again small, the use of prospective versus retrospective designs was not associated with different effect sizes for reports of childhood trauma and adversity. This is consistent with studies of depressed patients that have failed to find inflated accounts of adversity during the depressed state (Brewin et al., 1993). Another area of controversy concerns the role of intelligence as a risk factor. Prospective studies (Macklin et al., 1998; Pitman, Orr, Lowenhagen, Macklin, & Altman, 1991) have found that lower intelligence precedes rather than follows the development of PTSD, and in our meta-analysis the effect size for low intelligence based on prospective studies was somewhat larger than the effect size based on retrospective studies ( $r = .24$  vs.  $.16$ ),  $t(354) = 1.15$ ,  $p < .05$ . These findings were, however, all based on studies of combat veterans and remain to be confirmed within civilian samples. Finally, retrospective studies were associated with weaker effects for female gender and stronger effects for younger age at trauma, which again is likely to be because of confounding between design and military/civilian status.

In general, it would be expected that use of continuous scores would generate larger effect sizes because of the absence of the attenuating effect of grouping. This effect was found for younger age at trauma, lack of education, previous trauma, other childhood adversity, and trauma severity. Curiously, the effect size for gender was no longer significant when continuous measures of PTSD were used. This finding was based on only two studies using continuous measures and so may not be reliable. But the effect size for female gender was greater when respondents were interviewed rather than given questionnaires, a finding based on more studies and substantial numbers of respondents. There are a number of possible reasons for this; for example, interviewers may be biased toward assigning positive diagnoses of PTSD to women or men may be less willing to disclose symptoms in interviews. Interview methods of assessing PTSD also led to larger effect sizes for younger age at trauma and trauma severity, whereas questionnaire methods led to a larger effect size for previous trauma. The substantial effects of these moderator variables suggest that the

different ways PTSD is assessed are not all equivalent, and these methodological issues need to be taken into account when assessing research findings.

The subset of epidemiological studies that included PTSD arising from events in childhood demonstrated stronger effect sizes for female gender, as noted above, but weaker effect sizes for lack of education, previous psychiatric history, previous trauma, childhood adversity, and trauma severity. Inevitably there is confounding here with other moderator variables, such as civilian status, analyses based on diagnosis, current versus lifetime diagnoses, and interview assessments of PTSD, that cannot yet be teased apart because of the limited number of available studies. Nevertheless, it is noteworthy that comparing this group of studies with the rest tended only to affect the relative magnitude of what were still significant effect sizes rather than abolishing them completely, as was the case with some of the other moderator variables.

Although each of the risk factors identified in the meta-analysis accounts for relatively little variance, aggregating them could result in better levels of prediction. The sum of the pretrauma factors might then outweigh the apparently larger impact of trauma severity. Little is known, however, about whether such predictive effects would involve shared or unique variance. Clearly, education, SES, and intelligence are likely to be strongly interrelated. Further, childhood abuse and early trauma could be having effects both on intelligence (e.g., Perez & Widom, 1994), on child or adolescent psychiatric problems, and on later PTSD, with the result that the apparent predictive effects of intelligence or prior psychiatric disorder are already partly being measured by questions about early adverse environmental factors. Similarly, a family history of psychiatric disorder may be strongly related to an adverse family environment and the occurrence of abuse (Andrews, Brown, & Creasey, 1990).

Other reasons why pretrauma variables do not appear to be powerful predictors of PTSD include the possibility that their effects are mediated by later aspects of the trauma or of the person's response to the trauma. That is, their effects are distal rather than proximal, and their impact is diluted by one or more intervening variables. For example, it has been shown that the association between childhood abuse and later PTSD is mediated by the experience of shame (Andrews et al., 2000). Alternatively, several studies suggest that early trauma interacts with level of combat exposure to intensify the risk of PTSD (e.g., King, King, Foy, & Gudanowski, 1996). Together with the fact that the predictor variables so far identified are very general ones, these findings suggest that our understanding of vulnerability to PTSD is at an early stage.

Very few studies have examined other predictive factors measured pretrauma. O'Keefe, Toole, Marshall, Schureck, and Dobson (1998) reported that scores obtained at enlistment on the Australian Army Self-Description Inventory, which correlates highly with neuroticism, were predictive of later PTSD. Similar results were obtained by Schnurr, Friedman, and Rosenberg (1993) with various

subscales of the Minnesota Multiphasic Personality Inventory obtained preenlistment. In both cases, scores indicating greater psychopathology were weakly predictive of later PTSD. In general, more is known about potential predictors of PTSD occurring during or after exposure to a variety of traumas. Emotional responses such as shame and anger with others (Andrews et al., 2000), dissociation occurring during the traumatic event (Koopman et al., 1994; Shalev, Peri, Canetti, & Schreiber, 1996), and acute stress disorder (Brewin, Andrews, Rose, & Kirk, 1999; Classen, Koopman, Hales, & Spiegel, 1998; Harvey & Bryant, 1998) have been found to predict later PTSD in prospective studies. A recent study of victims of violent crime (Brewin et al., 1999) found that a simple threshold of three or more intrusive or arousal symptoms, assessed 3 weeks postcrime, was sufficient to predict later PTSD with a high degree of accuracy (approximately 80%; correct classification rate).

Despite the limitations imposed by the heterogeneity of the constituent studies in sampling, design, measurement, statistical analysis, and resulting effects, the meta-analysis succeeded in identifying weak effects of pretrauma risk factors together with slightly stronger effects for trauma intensity and other posttrauma risk factors. Although three variables had effects that were consistent in magnitude, there were ample indications that for most variables the degree of these predictive effects differs systematically according to sample and study characteristics. These moderator effects cannot yet be disentangled because of the high degree of confounding relative to the total number of studies. Additionally, there were substantial sectors of the population for whom gender, age at trauma, and race could not be shown to be risk factors at all. These findings clearly point up the heterogeneity of the disorder in different settings and warn against attempts to build a general vulnerability model for all cases of PTSD at this time. The data may be regarded as consistent with a model in which the impact of pretrauma factors on later PTSD is mediated by responses to the trauma or, alternatively, with a model in which pretrauma factors interact with trauma severity or trauma responses to increase the risk of PTSD. In either case, they suggest that it may be productive to investigate more proximal links in the causal chain, such as the association between pretrauma risk factors and immediate trauma responses.

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