ORIGINAL ARTICLE

A Second Look at Prior Trauma and the Posttraumatic Stress Disorder Effects of Subsequent Trauma

A Prospective Epidemiological Study

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Context: Previous studies showed increased probability of a posttraumatic stress disorder (PTSD) effect of trauma in persons who had experienced prior trauma. The evidence comes chiefly from retrospective data on earlier events, obtained from trauma-exposed persons with and without PTSD. A generally overlooked major limitation is the failure to assess the PTSD response to the prior trauma.

Objective: To estimate the risk of PTSD after traumas experienced during follow-up periods in relation to respondents' prior traumatic events and PTSD.

Design: A cohort study of young adults interviewed initially in 1989, with repeated assessments during a 10-year follow-up.

Setting and Participants: The sample was randomly selected from a large health maintenance organization in Southeast Michigan, representing the geographic area.

Main Outcome Measures: The relative risk of PTSD precipitated by traumatic events occurring during follow-up periods in relation to prior exposure and PTSD that had occurred during preceding periods, estimated by general estimating equations (n=990).

Results: The conditional risk of PTSD during the follow-up periods was significantly higher among trauma-exposed persons who had experienced prior PTSD, relative to those with no prior trauma (odds ratio, 3.01; 95% confidence interval, 1.52-5.97). After adjustment for sex, race, education, and preexisting major depression and anxiety disorders, the estimates were only marginally revised. In contrast, the conditional risk of PTSD during follow-up among trauma-exposed persons who had experienced prior traumatic events but not PTSD was not significantly elevated, relative to trauma-exposed persons with no prior trauma. The difference between the 2 estimates was significant ($P=.005$).

Conclusions: Prior trauma increases the risk of PTSD after a subsequent trauma only among persons who developed PTSD in response to the prior trauma. The findings suggest that preexisting susceptibility to a pathological response to stressors may account for the PTSD response to the prior trauma and the subsequent trauma.

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EPIDEMIOLOGICAL SURVEYS have documented that most community residents in the United States have experienced traumatic events.[1-3] Considerable proportions of those who experienced any traumatic event have experienced more than 1 such event.[1-4] Previous studies have reported elevated rates of prior traumatic events in adults with posttraumatic stress disorder (PTSD).[5-10] The finding has been interpreted as supporting a "sensitization" process, that is, greater responsiveness to subsequent stressors.[11] Although other psychiatric disorders (eg, major depression or generalized anxiety disorder) also are associated with exposure to stressors,[12-17] and although the occurrence of those disorders might be influenced by repeated exposure to stressors, the relationship between prior stressors and the increased risk of psychiatric disturbance after a subsequent stressor has been examined primarily in relation to PTSD. Posttraumatic stress disorder is the signature psychiatric disorder in persons who experience extreme stressors, and an etiologic link with an identifiable stressor is an essential feature in the definition of PTSD.

The evidence supporting the influence of prior trauma on the PTSD effects of a subsequent trauma comes primarily from cross-sectional studies in which retrospective data on earlier events are obtained from trauma-exposed persons with and without PTSD. Retrospective accounts of traumatic events are subject to

recall bias, a limitation that threatens the validity of causal inferences. Persons with psychiatric disturbances might be more likely to recall negative experiences, whereas those with no psychiatric disturbance might be more likely to forget and less likely to attribute causal meaning to objectively similar events.[12,18,19] A reporting bias associated with psychiatric status is likely to influence reports of prior trauma and lead to an apparent (but spurious) association between PTSD and prior trauma. The limitations of retrospective data are well recognized and are acknowledged in studies that seek to identify causal pathways based on cross-sectional data. A generally overlooked major limitation of studies on the effects of prior trauma is their failure to assess how persons had responded to the prior trauma, specifically, whether or not they had developed PTSD in response to the prior trauma. Consequently, it is unclear whether prior trauma alone or prior PTSD amplifies the risk of PTSD after a subsequent trauma. Evidence that previously exposed persons are at increased risk of PTSD only if their prior trauma resulted in PTSD would not support the hypothesis that exposure to traumatic events increases the risk of (ie, sensitizes them to) the PTSD effects of a subsequent trauma, transforming persons with normal reactions to stressors into persons susceptible to PTSD. It might suggest the possibility that trauma precipitates PTSD in persons with preexisting susceptibility (that had already been present before the prior trauma occurred). A predisposition to a pathological response to stressors might account for the PTSD response to the prior trauma as well as to the subsequent trauma. Evidence that personal vulnerabilities, chiefly neuroticism, a history of major depression and anxiety disorders, and family history of psychiatric disorders, increase the risk of PTSD has been consistently reported.[20-28] There also is evidence that personal vulnerabilities are stronger predictors of a psychiatric response to traumatic events than is trauma severity, especially in civilian samples.[28,29]

In this longitudinal epidemiological study of young adults with repeated assessments during a 10-year follow-up period, we estimate the risk of PTSD associated with stressors experienced during the follow-up periods in relation to the respondents' prior traumatic events and PTSD that had occurred during preceding periods. The study addresses the limitation due to potential recall bias in previous cross-sectional studies. Information on prior trauma (the predictor) collected in earlier assessments is uncontaminated by the respondents' PTSD associated with new exposure during subsequent periods collected in subsequent assessments (the dependent variable). Furthermore, the availability of data on the PTSD effects of the prior traumas provides an opportunity to examine the following question: Does earlier trauma per se or only earlier trauma that culminated in PTSD predict the PTSD response to a subsequent trauma?

METHODS

SAMPLE

A sample of 1200 persons was randomly selected in 1989 from all 21- to 30-year-old members of a large health maintenance organization in southeast Michigan. The health maintenance organization membership represented the population of the geographic area, with the exception of the extremes of the socioeconomic range, at the time of the 1990 US census. Personal interviews were conducted in 1989 with 1007 respondents, 83.9% of the sample. Follow-up interviews were conducted in 1992, 1994, and 1999 to 2001 at 3, 5, and 10 years after baseline, respectively. Complete data from all 4 assessments are available on 899 participants, and 990 of the 1007 participants (98.3%) had at least 1 follow-up interview. Detailed information on the sample and the population was reported previously.[23,30]

ASSESSMENT

The National Institute of Mental Health Diagnostic Interview Schedule, III (NIMH-DIS)[31] for *DSM-III-R* was used to diagnose PTSD and other psychiatric disorders. The baseline interview inquired about lifetime history of the disorders, and each follow-up assessment inquired about disorders occurring during the interval since the previous assessment. The diagnosis of PTSD in *DSM-III-R* requires exposure to a qualifying traumatic event and the presence of a configuration of criterion symptoms that are linked to the traumatic event. The NIMH-DIS opens with a list of qualifying events, using the examples in the *DSM-III-R* text. The definition of qualifying events in *DSM-III-R* is considerably narrower than that in *DSM-IV*.[32] Among respondents who report 2 or more traumatic events, the NIMH-DIS inquires first about PTSD criterion symptoms in relation to an event designated by the respondent as their worst (ie, most distressing) event and continues up to 3 events.[31] The worst-event method, which has been used in large-scale epidemiological studies, is an efficient alternative to assessing PTSD for all traumatic events.[1,4,32,33] It has also been used in clinical trials.[34] A previous epidemiological study, which inquired about *DSM-III-R* PTSD in relation to second and third events (in addition to the worst event), reported that only a few respondents who did not develop PTSD after the worst event developed PTSD after another traumatic event.[33] Two studies[35,36] reported high concordance between the diagnosis of PTSD by lay interviewers using structured interviews based on the NIMH-DIS and independent clinical reinterviews.

STATISTICAL ANALYSIS

To estimate prospectively the risk of the occurrence of PTSD associated with prior trauma, we used multinomial logistic regression, applying generalized estimating equations (GEE).[37,38] Data come from baseline and 3 follow-up assessments 3, 5, and 10 years after baseline. Data from the 3-year follow-up assessment were combined with data from the 5-year follow-up assessment to increase the stability of the results, given the expected low conditional probability of PTSD. In this analysis, the outcome (no new exposure, exposure/no PTSD, or exposure/PTSD) is set in a clear temporal sequence in relation to the predictor (no prior exposure, prior exposure/no PTSD, or prior exposure/PTSD) across the repeated assessments. Exposure to traumatic events with and without PTSD (vs no exposure), measured at baseline and at the 5-year assessment, are used to predict new exposure and PTSD during the respective subsequent periods, from baseline to the 5-year assessment and from baseline and the 5-year assessment to the 10-year assessment. Compared with a standard logistic regression in which the cumulative incidence of PTSD during the period from baseline to the last assessment is estimated, our approach has the advantage of putting to use all the prospectively gathered information, taking into account changes in the predictors (prior

Table 1. Exposure to Trauma and Conditional Probability of PTSD During the 2 Follow-up Intervals Across Subgroups

	First Follow-up			Second Follow-up		
	No. of Respondents[a]	Exposed, %	PTSD Among Exposed, %	No. of Respondents[a]	Exposed, %	PTSD Among Exposed, %
Prior exposure/PTSD	92	42.4	18.0	105	60.0	19.1
Prior exposure/no PTSD	294	33.3	12.2	386	41.5	6.3
No prior exposure	604	24.0	8.3	419	27.4	6.1
Sex						
Female	613	28.4	13.8	575	38.1	11.0
Male	377	28.7	6.5	338	35.5	4.2
Race						
White	797	25.2	9.0	740	34.1	7.9
Black	193	42.0	16.1	173	50.3	10.3
Education						
College	289	22.2	9.4	269	34.2	7.6
< College	701	31.1	11.5	644	38.4	8.9
Preexisting disorders						
MDD	112	42.9	20.8	163	46.6	15.8
No MDD	877	26.7	9.0	745	35.2	6.5
Anxiety disorders	237	32.9	19.2	300	41.3	12.1
No anxiety	753	27.1	7.8	610	35.3	6.5
SUD	261	28.0	15.1	277	36.5	9.9
No SUD	729	28.7	9.6	636	37.4	8.0
Total	**990**	28.5	11.0	**913**	37.1	8.6

Abbreviations: MDD, major depressive disorder; PTSD, posttraumatic stress disorder; SUD, substance use disorder.
[a] Some variables do not add to the total because of missing data.

exposure and prior PTSD) that occurred between the assessments. Major depression and any anxiety disorder were used as covariates to control for their effects, except if their onset was after the first occurrence of PTSD. Logistic regression using GEE models yields estimates of odds ratios, adjusted for other variables in the equation. The outcome (Y) in the model has the following 3 nominal scaled categories: no new exposure (the reference), 0; new exposure/no PTSD, 1; and new exposure/PTSD, 2. The model has the following 2 logit functions: Y = 1 vs Y = 0 and Y = 2 vs Y = 0. The logit for comparing Y = 2 vs Y = 1, which estimates the risk of new exposure/PTSD vs new exposure/no PTSD, the key outcome of interest, can be obtained as the difference between the 2 logit functions.[39] We used SAS software, release 9.2 (Proc Surveylogistic) for the statistical analyses.[40]

RESULTS

EXPOSURE TO TRAUMATIC EVENTS AND PTSD DURING FOLLOW-UP

Analyses included 990 respondents, 98.3% of the initial panel. This subset had at least 1 follow-up assessment. Of this sample, 63.2% were women, 80.0% were white, 46.1% were married, and 29.0% had completed college. The initial sample is described in detail by Breslau et al.[23] The total number of respondents exposed to 1 or more *DSM-III-R* traumatic events during the 10 years from baseline to the last follow-up was 498. A total of 52 respondents met *DSM-III-R* criteria for PTSD associated with exposure to traumatic events during the 10-year follow-up. The most common traumatic events reported at baseline and follow-up assessments were physical assault, severe injury or accident, witnessing violence, and a family member or a close friend severely injured or killed. Approximately 80% of respondents with at least 1 event at each assessment reported 1 of those event types.

Of the respondents who reported traumatic events at baseline, 30.8% referred to an event in childhood (< 16 years of age) as the worst (or only). The risk of PTSD was 23.2% if the worst event occurred in childhood and 23.9% if it occurred at an older age. Of all respondents with prior PTSD who were identified at the baseline and first assessments, 24.7% had experienced symptoms at the time of the prior assessments, which marks the start of subsequent periods. The conditional probability of PTSD associated with subsequent exposure among them was 22.2%. The conditional probability of PTSD associated with subsequent exposure among prior PTSD cases whose symptoms had remitted at the time of the prior assessment was 15.6%.

Table 1 presents descriptive data on exposure to traumatic events and PTSD during the 2 consecutive 5-year follow-up periods in subsets of the sample, classified by prior exposure and prior PTSD, sex, race, education, and preexisting psychiatric disorders. For the first follow-up interval, the classification of respondents into categories of prior PTSD, prior exposure/no PTSD, and no prior trauma is based on data from the baseline interview. For the second follow-up interval, the classification is based on the combined data from the assessments at baseline and the 5-year follow-up. The occurrence of PTSD during the follow-up periods among respondents who had developed PTSD from a prior exposure was higher than among those who had experienced prior trauma but had not developed PTSD, as well as among those with no prior exposure. The occurrence

Table 2. Relative Risk of PTSD After Subsequent Traumas Associated With Prior PTSD, Prior Trauma/No PTSD, and Covariates From a Series of Bivariate Models and a Multivariable GEE Multinomial Model[a]

Variable	Bivariate Associations OR (95% CI)	Multivariable Model Adjusted OR (95% CI)
Prior exposure/PTSD vs no prior exposure	3.01 (1.52-5.97)[b]	2.68 (1.33-5.41)[b]
Prior exposure/no PTSD vs no prior exposure	1.24 (0.65-2.36)	1.22 (0.64-2.34)
Female vs male	2.51 (1.25-5.06)[b]	1.94 (0.93-4.07)
White vs black	0.61 (0.33-1.12)	0.60 (0.32-1.11)
College education vs less than college	0.81 (0.41-1.59)	1.00 (0.51-1.96)
Preexisting MDD	2.72 (1.49-4.99)[b]	2.09 (1.71-3.75)[b]
Preexisting anxiety disorder	2.35 (1.32-4.20)[b]	1.65 (0.91-2.97)

Abbreviations: CI, confidence interval; GEE; generalized estimating equation; MDD, major depressive disorder; OR, odds ratio; PTSD, posttraumatic stress disorder.

[a] Each of the bivariate models and the multivariable model includes a term for the 2 time intervals (suppressed). The outcome (Y) in these models has the following 3 nominal scaled categories: 0, no exposure (the reference); 1, exposure/no PTSD; and 2, exposure/PTSD. The logit for comparing Y = 2 vs Y = 1, which estimates the risk of new exposure/PTSD vs exposure/no PTSD and is presented in the table, is obtained by the difference between the 2 logit functions, Y = 1 vs Y = 0 and Y = 2 vs Y = 0. The differences between prior trauma/PTSD and prior trauma/no PTSD are statistically significant in both models (in the multivariable model, adjusted OR, 2.20; 95% CI, 1.10-4.39) ($P = .005$).

[b] $P < .05$.

of PTSD during the follow-up periods in respondents with prior exposure/no PTSD was intermediate between that of respondents with prior exposure/PTSD and those with no prior exposure in the first follow-up interval, but it was indistinguishable from the reference category (no prior exposure) at the second follow-up interval. We also observed in these data the well-established sex difference in the conditional probability of PTSD and the elevated risk of PTSD in persons with preexisting major depression and anxiety disorders. The observed associations of these factors with PTSD were consistent across the 2 follow-up intervals. Table 1 also presents descriptive data on exposure to trauma during follow-up, discussed in "Estimating the Risk of Subsequent Exposure to Traumatic Events" subsection.

ESTIMATING THE RISK OF PTSD IN RELATION TO PRIOR EXPOSURE WITH AND WITHOUT PTSD

Table 2 displays estimates of the relative risk of PTSD from the GEE models. The unadjusted (bivariate) and adjusted odds ratios (from a multivariable model) in the first and second columns, respectively, estimate the conditional risk of PTSD during the follow-up intervals in relation to prior exposure to trauma and PTSD that had occurred during the preceding periods. The estimated relative risk of PTSD after new exposure during follow-up periods was 3-fold higher among respondents who had experienced prior PTSD (prior exposure/PTSD), compared with respondents with no prior exposure (odds ratio, 3.01; 95% confidence interval [CI], 1.52-5.97). In contrast, the conditional risk of PTSD during the follow-up periods among trauma-exposed respondents who had previously experienced traumatic events but not PTSD (exposure/no PTSD) was not significantly elevated, compared with that of respondents with no prior trauma. After adjustment for sex, race, education, and preexisting major depression and anxiety disorders in the multivariable analysis, these estimates were only marginally attenuated. Examination of the effects of covariates showed that women's higher risk of PTSD and the elevated risk associated with preexisting major depression and anxiety disorders were significant in the bivariate GEE models. However, only preexisting major depression was sustained in the multivariable model as an independent predictor of PTSD.

ESTIMATING THE RISK OF SUBSEQUENT EXPOSURE TO TRAUMATIC EVENTS

Table 1 also presents descriptive data on exposure to traumatic events during each of the 2 follow-up intervals in subsets of the sample. Respondents who had developed PTSD from a prior trauma had the highest likelihood of exposure to traumatic events during the follow-up periods, and those who had experienced prior trauma but not PTSD had an intermediate likelihood, compared with respondents with no prior trauma as the reference group. This pattern was observed in both follow-up intervals. Another finding of interest was that preexisting major depression was associated with an elevated risk of exposure to traumatic events during the follow-up periods. In contrast, preexisting substance use disorders bore no relationship to exposure to traumatic events during subsequent years.

Results for the relative risk of subsequent exposure to traumatic events from the GEE models (data not displayed) showed that prior trauma exposure with and without PTSD was associated with an increased risk of subsequent exposure. The relative risk estimate for subsequent exposure adjusted for sex, race, education, and preexisting disorders associated with prior exposure/PTSD was 2.61 (95% CI, 1.86-3.66) and for prior exposure/no PTSD, 1.59 (95% CI, 1.26-1.99). The risk of subsequent exposure to traumatic events was lower in white than in nonwhite respondents (OR, 0.52; 95% CI, 0.40-0.67) and was increased in respondents with preexisting major depression (OR, 1.50; 95% CI, 1.09-2.07).

COMMENT

In this epidemiological prospective study, we found that prior exposure to trauma increased the risk of the PTSD effects of a subsequent trauma only among persons who had developed PTSD in response to the prior trauma. Those who had experienced a prior trauma but had not developed PTSD in response to the prior trauma were not at elevated risk of PTSD when they experienced a subsequent trauma. These prospective data do not support the suspected sensitization effect of

prior trauma. The studies that gave rise to the sensitization hypothesis, including the 2 studies that we conducted in samples of the general population,[6,41] did not obtain information on the PTSD response to the prior trauma. That information appears to be crucial. Without it, any observed difference in the PTSD risk between trauma-exposed persons with vs those without prior trauma is ambiguous as evidence concerning the role of prior trauma in PTSD. The information about the PTSD response to the prior trauma suggests the possibility that preexisting susceptibility might account for the PTSD response to the prior trauma and the PTSD response to the subsequent trauma.

Evidence that prior trauma alone (in the absence of prior PTSD) did not predict the PTSD response to a subsequent trauma, observed in this study, was foreshadowed by a 1987 Israeli study of acute combat stress reaction (CSR) among a sample of soldiers of the 1982 Lebanon War.[42] (Posttraumatic stress disorder was not examined.) In that study, data from military and mental health records were consulted to verify the soldiers' participation in previous wars and whether soldiers who had participated in a previous war had experienced CSR. The authors reported that CSR occurred more frequently among soldiers of the Lebanon war who had experienced CSR in a previous war but not among soldiers who had fought in a previous war but had not experienced CSR, compared with new recruits who had not fought in a previous war. The authors concluded that knowledge of the outcome of prior combat was essential for predicting soldiers' response to subsequent combat. Soldiers who experienced CSR in a previous war might have had preexisting vulnerability that also accounted for their increased risk of CSR during the subsequent war. Soldiers who had fought in a previous war but had not experienced CSR had a lower rate of CSR during the subsequent war than did new recruits who had no war experience. It is tempting to interpret this observation as evidence of inoculation. However, the higher rate of CSR among new recruits than among those who had fought in a previous war without experiencing CSR is not unexpected. The new recruits, an unselected group with respect to their psychiatric response to war, included soldiers who would have had CSR had they fought in a prior war. This undetected vulnerable subset would push up the rate of CSR in the group of new recruits as a whole.

A later cross-sectional study in a civilian sample of bus drivers in which symptoms of PTSD (but not the actual disorder) were measured reported no association between prior trauma alone and the psychiatric response to subsequent trauma.[43] Instead, only subjects who reported a high level of symptoms from the prior trauma were at increased risk of a high level of symptoms from the subsequent trauma. Preexisting vulnerability also might explain our findings. Posttraumatic stress disorder from a prior trauma might signify a preexisting susceptibility that continued to confer an elevated PTSD risk from subsequent traumas.

Although we found that prior trauma alone did not predict the PTSD effects of a subsequent trauma, we have no direct evidence to explain how prior PTSD increased the risk of the PTSD response to a subsequent trauma. The possibility that preexisting vulnerabilities (ie, present before the prior trauma occurred) might account for the finding is supported by the body of evidence that personal susceptibilities have a key role in PTSD. Other factors should be considered. Among them are (1) PTSD cases identified at baseline might already have been sensitized by early childhood traumas, which were undetected in our study, and (2) PTSD from a subsequent trauma among respondents with prior PTSD might have been a continuation of chronic, unremitted PTSD. As to the first factor, traumatic events that occurred in childhood were not examined prospectively. However, we found no evidence that childhood events, when selected by the respondents as the worst (or only) events at baseline, were associated with an elevated PTSD risk. A recent prospective population study detected a very low conditional risk of PTSD in childhood.[44] As to the second factor, we found that PTSD from a new trauma, at the first or the second follow-up, could not be explained as the persistence of unremitted prior PTSD. Approximately 75% of cases of lifetime PTSD ascertained at the baseline assessment and the additional cases of PTSD that occurred after trauma experienced during the first follow-up period had remitted at the start of the respective follow-up periods. The risk of the PTSD effects of a subsequent exposure varied little between remitted and active cases.

We found that prior exposure to trauma increased the risk of subsequent exposure to traumatic events, a finding that is consistent with previous prospective and retrospective data.[45] Traumatic events are not randomly distributed. Personal predispositions, occupation and job conditions, and leisure-time activities might account for prior and subsequent exposure to traumatic events. The increased risk of exposure to traumatic events associated with preexisting major depression replicates prior reports and is in line with the evidence that persons with major depression are at increased risk of stressful life events.[46,47] Impairment in emotional, motivational, and cognitive capacities associated with major depression might increase the likelihood of exposure to a wide range of stressors.[48,49] The failure to find evidence that preexisting substance use disorders are associated prospectively with an elevated risk of exposure to traumatic events replicates some, but not all, earlier reports.[50-52]

The study was conducted on a sample of the general community during peacetime. Although research on PTSD has focused primarily on Vietnam veterans and war-related traumas, stressors that qualify for the PTSD diagnosis also occur among community residents who experience violence, disaster, serious accidents, and other severely traumatic events. The results of the present study may not apply to war-related traumas. In addition, the generalizability of the findings is limited by the characteristics of this epidemiological sample of young adults who were followed-up for 10 years.

The prospective nature of our data offers an important methodological advantage. Information on prior trauma (the predictor), collected in earlier assessments, is uncontaminated by PTSD associated with a new exposure during subsequent periods, gathered in subse-

quent assessments (the dependent variable). The potential for bias in studies that rely on retrospective reports of presumed etiologic events in relation to psychiatric disturbances has been examined most often in connection with depression.[12,18,19] Theoretical and empirical grounds for questioning the meaning of statistical association between depression and stressful life events based on retrospective data are summarized in Schraedley et al.[19] A leading cognitive theory of depression, as they noted, postulates that depressed persons are "characterized by negatively distorted perceptions of their world, and by retrieval biases that enhance the accessibility of sad or negative memories."[19(p308)] Their own findings show the instability of retrospective reports of traumatic events because depressed persons overreported and nondepressed persons underreported past traumatic events, including events in childhood. Similar findings have been reported in connection with combat or military stressors and PTSD.[53-55] The significance of recall bias in research on the relationship of PTSD with early or prior trauma cannot be overestimated because virtually all of the empirical evidence on the effects of past trauma and childhood trauma is based on the retrospective accounts of adults. The high costs of successfully completing longitudinal studies from childhood to adulthood have prohibited such studies. Our longitudinal study, which followed up young adults for 10 years, is limited by the lack of assessments during childhood. Despite this limitation, the study offers new and potentially useful insight into the role of prior trauma. Information on the occurrence of prior trauma is relevant but might be grossly insufficient, without knowledge of the PTSD effects of the prior trauma. If replicated in future studies, the findings would have implications for studies on risk factors and diathesis in PTSD. Among trauma-exposed persons who have experienced a prior trauma, the subset of persons with prior PTSD might be highly informative in identifying biological markers and predispositions to psychiatric responses to stressors. The essential information might be the PTSD response to prior trauma rather than the presence and characteristics of prior trauma.

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