Persistent Posttraumatic Stress Disorder Following September 11 in Patients With Bipolar Disorder

Mark H. Pollack, M.D.; Naomi M. Simon, M.D., M.Sc.; Andrea Fagiolini, M.D.; Roger Pitman, M.D.; Richard J. McNally, Ph.D.; Andrew A. Nierenberg, M.D.; Sachicko Miyahara, Ph.D.; Gary S. Sachs, M.D.; Carol Perlman, Ph.D.; S. Nassir Ghaemi, M.D.; Michael E. Thase, M.D.; and Michael W. Otto, Ph.D.

Objective: We examined the development of posttraumatic stress disorder (PTSD) following indirect exposure to the September 11, 2001, terrorist attacks in a cohort at high risk for adverse trauma-related sequelae as a result of having bipolar disorder.

Method: Subjects (N = 137) were participants in the ongoing, naturalistic, longitudinal study Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD) prior to September 11, 2001. The present study examined prospectively collected pre-event information about bipolar disorder and other potential predictors of PTSD, along with assessment of the level of indirect trauma exposure (i.e., via media) and peritraumatic distress in the aftermath of September 11, and their association with 9/11-related, new-onset PTSD as assessed by a self-report measure, the Posttraumatic Stress Diagnostic Scale.

Results: Posttrauma assessments were completed a mean ± SD of 430.6 ± 78.7 days (range, 0.5–1.5 years) after September 11. Twenty percent (N = 27) of patients reported development of new-onset PTSD in response to the September 11 attacks. Rates of PTSD were significantly associated with the presence of a hypomanic, manic, or mixed mood state at the time of trauma ($\chi^2 = 4.25$; $p < .05$); 62% of patients in these states developed PTSD. Mania/hypomania remained a significant predictor of PTSD in response to the September 11 attacks after controlling for peritraumatic exposure and distress variables, suggestive of a substantial increase in risk compared with those in recovery (OR = 17; 95% CI = 2.6 to 115.6; p = .0034).

Conclusions: Rates of persistent new-onset PTSD among bipolar patients were elevated in the aftermath of the September 11 attacks. Our findings suggest that the presence of a manic state may be the most critical risk factor for adverse sequelae following indirect traumatic exposure in bipolar individuals.

(J Clin Psychiatry 2006;67:394–399)

Received July 8, 2005; accepted Dec. 21, 2005. From the Department of Psychiatry, Massachusetts General Hospital, Boston, Mass. (Drs. Pollack, Simon, Pitman, Nierenberg, Sachs, and Perlman); the Department of Psychiatry, Western Psychiatric Institute, Pittsburgh, Pa. (Drs. Fagiolini, Miyahara, and Thase); the Department of Psychology, Harvard University, Cambridge, Mass. (Dr. McNally); the Department of Psychiatry, Emory University, Atlanta, Ga. (Dr. Ghaemi); and the Department of Psychology, Boston University, Boston, Mass. (Dr. Otto).

This study was supported by the National Institute of Mental Health, Bethesda, Md. (5 R01 MH 663901; PI: M. H. Pollack).

Dr. Nierenberg is a consultant for Eli Lilly, Shire, GlaxoSmithKline, Innapharma, Genaissance, and Sepracor; has received grant support from Eli Lilly, Wyeth, GlaxoSmithKline, Bristol-Myers Squibb, Cyberonics, Lichtwer, Pfizer, Cederroth, Forest, and Janssen; and has received honoraria from Eli Lilly, Wyeth, and GlaxoSmithKline. Drs. Pollack, Simon, Fagiolini, Pitman, McNally, Miyahara, Sachs, Perlman, Ghaemi, Thase, and Otto report no other significant commercial relationships relevant to the study.

Corresponding author and reprints: Mark H. Pollack, M.D., Director, Center for Anxiety and Traumatic Stress Disorders, Massachusetts General Hospital, Simches Research Building, 2nd Floor, 185 Cambridge St., Boston, MA 02114 (e-mail: mpollack@partners.org).

The terrorist attacks of September 11, 2001, were an unprecedented event in the history of the United States and resulted in high rates of distress in the general population.[1–3] For example, Schuster and colleagues[1] reported results of a nationwide telephone survey 3 to 5 days after the attacks of September 11 in which 44% of adults reported significant symptoms of distress related to the event. However, for most individuals, the increase in affective and anxiety symptoms, including the development of acute posttraumatic stress disorder (PTSD), was transient and generally resolved over a period of weeks or months. For example, although a random phone survey of New York City residents conducted within 2 months of the attacks found rates of current PTSD related to the attacks of 7.5%,[3] only 0.6% still had PTSD at 6 months after the event.[4]

Identification of individuals at greatest risk for the development of persistent PTSD is an area of critical public health concern as it may permit targeting of resources for the treatment of those most vulnerable to the aversive effects of trauma. Individuals with bipolar disorder appear to be a group with particular vulnerability to the development of PTSD. Patients with bipolar disorder tend to report elevated rates of PTSD; evidence from 8 studies rep-

resenting 1214 individuals with bipolar disorder indicates an overall prevalence of PTSD of 16%, roughly double the expected rate.[5] In addition to trauma exposure,[6–8] a number of characteristics identified as risk factors for PTSD are common in individuals with bipolar disorder, including depression and hypomania,[9] comorbid psychiatric disorders such as anxiety disorders,[10–13] and heightened neuroticism.[9,11,14,15]

Relatively little is known about the effects of indirect exposure on the development of PTSD or associated symptoms, although studies of earlier national traumas suggest that media exposure, both in the acute aftermath and ongoing at the time of assessment, may be a significant contributor to postevent distress in children.[16] In this study, we examined the emergence of PTSD following indirect exposure (i.e., via media) to the September 11 attacks in a cohort of patients who were at high risk for adverse trauma-related sequelae as a result of having bipolar disorder. Because participants had been enrolled in an ongoing naturalistic study, detailed prospectively collected pre-event data regarding potential predictors of PTSD were available.

Assessment of PTSD symptoms began approximately 6 months after September 11, 2001, by which time, as data from Galea and colleagues[4] suggest, many of the transient cases of PTSD would already have abated, thus providing an opportunity to examine individuals with more persistent symptoms.

We hypothesized that rates of emergent PTSD after September 11 would be elevated among adults with bipolar disorder relative to the general population, and that rates of PTSD would be highest among patients meeting criteria for depression or mania/hypomania on September 11.

METHOD

Study Overview

Participants were recruited from enrolled participants in an ongoing, naturalistic study of bipolar disorder: the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD; NIMH-98-DS-0003).[17] STEP-BD is a multicenter National Institute of Mental Health–funded project designed to evaluate the longitudinal outcome of patients with bipolar disorder.[17] To enter STEP-BD, patients were required to be at least 15 years of age and to meet *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, (DSM-IV)[18] criteria for bipolar I disorder, bipolar II disorder, cyclothymia, bipolar disorder not otherwise specified, or schizoaffective disorder manic or bipolar subtypes. Exclusion criteria were limited to unwillingness or inability to comply with study assessments, or inability to give informed consent. Written informed consent was obtained before study participation. Patients entering STEP-BD completed diagnostic interviews for bipolar disorder using 2 interview guides: a standardized affective disorder evaluation (ADE)[19] and the Mini-International Neuropsychiatric Interview Plus, Version 5.0 (MINI).[20] Further detail regarding study methodology has been published elsewhere.[17]

Pre-event data, already collected in STEP-BD about current affective symptoms, comprised clinician-rated DSM-IV–based categories of full criteria for a manic/hypomanic/mixed state (“mania”), a depressive episode (“depression”), recovering or recovered (relative euthymia defined as ≤ 2 moderate symptoms for at least a week or a full 8 weeks, respectively), and roughening (subsyndromal symptoms defined as ≥ 3 moderate symptoms, but not full criteria for a mood episode) from the closest clinician assessments (ADE) prior to 9/11. These bipolar state categories and interrater reliability training are further discussed by Sachs et al.[17]

Diagnostic status for comorbid conditions, bipolar subtype (bipolar I vs. II), and age at onset of bipolar disorder were available from structured clinician interviews with the MINI[20] administered at the yearly assessment most closely preceding 9/11 in STEP-BD. Within the class of anxiety disorders, we also examined, separately, current and past-but-not-current panic disorder with or without agoraphobia, agoraphobia without panic disorder, social anxiety disorder, obsessive-compulsive disorder, posttraumatic stress disorder, or generalized anxiety disorder. We defined “any anxiety disorder” as having met DSM-IV criteria for at least 1 of these 6 disorder classifications.

STEP-BD participants with a baseline evaluation before September 11, 2001, were eligible for study entry. To reduce potential variability associated with examination of varying numbers of patients at different sites across the country, we studied individuals only at 2 of the top-enrolling sites for STEP-BD for whom data were available prior to September 11: the Massachusetts General Hospital in Boston (N = 98), the site of the hijacking of 2 planes used in the September 11 attacks, and Western Psychiatric Institute in Pittsburgh, Pa. (N = 42). The human studies committees at both sites approved this study, and participants received and signed written informed consent. Subjects received compensation for completing the questionnaires.

Participants completed an additional assessment battery consisting of self-rated questionnaires about their level of peritraumatic exposure and distress and about the development of PTSD symptoms specifically in response to the terrorist attacks on September 11. We used the Posttraumatic Stress Diagnostic Scale (PDS),[21] a 17-item self-report scale that includes items reflecting DSM-IV diagnostic criteria and has good internal reliability and convergence with other measures of PTSD, as the instrument to screen for PTSD in this study. In order to reflect that the diagnosis of PTSD derived with this instrument should be considered provisional pending structured clini-

cal assessment, we will refer to individuals meeting syndromal criteria assessed with the PDS as having "probable PTSD" throughout the article.

Peritraumatic exposure and distress were measured with an assessment instrument utilizing a Likert-type response format with questions about (1) the amount of television coverage of the attacks watched on September 11 and the amount viewed during the subsequent 10 days; (2) the degree to which the participant purposely avoided or sought out media coverage of the September 11 attacks (i.e., changed viewing habits) as an attempt to reduce anxiety; (3) the degree of overall distress experienced during the week after September 11; and (4) the level of identification with an individual who died in the attacks (for example, thinking "He or she was just like me," or "What would it have been like to be that person?"). Date of packet completion (relative to September 11) was recorded and controlled for in the data analysis.

Our overall data analytic strategy was to examine the individual relationships between the predictors and 9/11-related new-onset PTSD, using logistic regression to adjust for the number of days since 9/11, since the time of questionnaire completion varied for each subject. To examine the relationship between bipolar mood state and television exposure, the nonparametric Kruskal-Wallis test was used, followed by pairwise comparisons with the Wilcoxon 2-sample test.

Table 1. Characteristics of Bipolar Patients With Persistent New-Onset Posttraumatic Stress Disorder (PTSD) Following September 11

Characteristic	Patients (N = 137)
Age, mean ± SD, y	41.0 ± 11.3
Female, %	63
Marital status, %	
Never married	43.5
Married or living as married	37.4
Divorced or separated	16.8
Widowed	1.5
Unknown	0.8
Education, %	
College or graduate degree	57.6
Some college or technical school	25.2
High school diploma	13.7
Partial high school or less	3.8
Employment, %	
Full time	35.1
Part time	16.0
Disabled or unemployed	42.7
Homemaker	3.8
Retired	1.5
Unknown	0.8
Age at onset of bipolar disorder (N = 133), mean, y	18.1
Diagnoses, %	
Bipolar I (N = 137)	74
Current anxiety disorder (N = 132)	40
Current comorbid substance abuse (N = 132)	5
Lifetime PTSD prior to 9/11 (N = 131)	14

RESULTS

Subjects

Posttrauma assessments were completed by a total of 140 of 203 patients recruited on the basis of enrollment in STEP-BD at these sites prior to 9/11 and continuing at the time of this study (69% response rate; N = 98 in Boston and N = 42 in Pittsburgh). Assessments were completed a mean ± SD of 430.6 ± 78.7 days (range, 0.5–1.5 years) after September 11. As there were no significant differences between the 2 sites, data were combined for all analyses. Because of the focus on the impact of indirect trauma, 3 patients who personally knew someone who died during the attack were excluded; thus, data on 137 patients were examined. The demographics of the study sample are described in Table 1.

Of the 137 patients (74% bipolar I), 69 (50%) were in recovering or recovered affective status proximal to September 11; 33 (24%) were depressed; 13 (10%) were hypomanic, manic, or mixed; and 22 (16%) had subsyndromal mood symptomatology (referred to as "roughening" in STEP terminology).

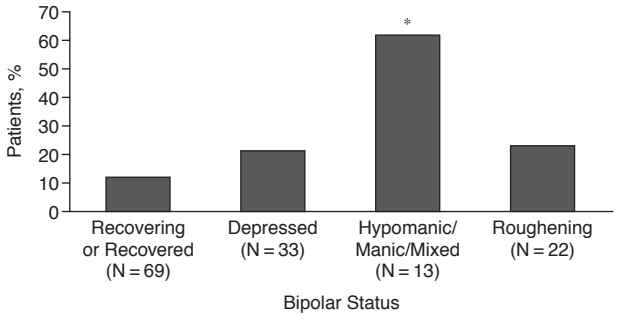
Rates of Probable PTSD

Twenty percent (N = 27; 95% CI = 13.4% to 27.4%) of patients reported the development of probable new-onset PTSD in response to September 11. We examined the association between the development of probable PTSD and individual patient demographic and diagnostic characteristics, controlling for the number of days between 9/11 and the time of questionnaire completion as a covariate in separate logistic regressions. Patients who developed probable PTSD were older (45.8 ± 9.3 vs. 40.9 ± 11.8 years) (OR = 1.04; 95% CI = 1.0 to 1.08; $p < .05$). However, gender (OR = 0.95; 95% CI = 0.40 to 2.26; p = .91), socioeconomic status (as assessed by household income, OR = 1.01; 95% CI = 0.85 to 1.21; p = .91), education level (OR = 0.99; 95% CI = 0.75 to 1.32; p = .96), quality of life (OR = 0.98; 95% CI = 0.95 to 1.01; p = .18), role function (OR = 1.09; 95% CI = 0.97 to 1.22; p = .15), and marital status (OR = 2.05; 95% CI = 0.85 to 4.95; p = .11) did not differentially predict the development of probable PTSD. Neither age at onset of bipolar disorder (OR = 0.97; 95% CI = 0.92 to 1.03; p = .36) nor diagnostic subtype (bipolar I vs. II; OR = 0.77; 95% CI = 0.24 to 2.50; p = .66) was differentially associated with the presence of new-onset probable PTSD.

Rates of probable PTSD were significantly associated with mood state at the time of the trauma (Figure 1). Patients in a hypomanic, manic, or mixed state were significantly more likely ($\chi^2 = 4.25$, df = 1, $p < .05$) than those in other mood states to develop probable PTSD; 62% (N = 8) developed probable PTSD subsequent to September 11, compared with 11% to 23% of those in other mood states.

Although the sample size was small (N = 6), 50% of those with current substance abuse developed probable

Figure 1. Development of Probable PTSD After September 11 by Bipolar Clinical Status



*p < .05.

PTSD (OR = 4.81; 95% CI = 0.88 to 26.30; p < .07). Neither a lifetime history of PTSD nor any anxiety disorder predating September 11 was associated with the development of subsequent probable PTSD in the sample as a whole or specifically in those in a hypomanic, manic, or mixed state.

Relationship of Development of PTSD With Television Viewing

Although interpretation of our measures of media exposure to trauma are limited by their retrospective assessment, we nonetheless found as expected that the development of probable PTSD was associated with the amount of television viewing of September 11–related material in the 10 days subsequent to the attacks (OR = 1.48; 95% CI = 0.99 to 2.20; p < .06) and with the level of reported distress during that first week (OR = 1.46; 95% CI = 0.97 to 2.21; p = .07), each at the level of a trend. However, the amount of television viewed on the actual day of the attack (OR = 1.02; 95% CI = 0.75 to 1.39; p = .89), changes in viewing habits (OR = 1.27; 95% CI = 0.78 to 2.1; p = .34), and identification with victims of the attack (OR = 1.31; 95% CI = 0.92 to 1.87; p = .13) were not predictive of the development of probable PTSD.

To evaluate whether the level of peritraumatic media exposure and/or the level of distress in response to September 11 might explain the association between bipolar mood state and probable PTSD, we performed additional analyses. First, we tested whether mood state proximal to the terrorist attacks univariately predicted television exposure or peritraumatic distress. Mood state was associated only with the amount of television viewing during the 10 days following September 11 (Kruskal-Wallis $\chi^2 = 13.3$, df = 3, p = .004): pairwise analyses revealed that depressed patients watched more television than those roughening (p = .005) or in recovery (p = .001). There were no other significant associations between mood state and peritraumatic variables. Mania/hypomania specifically was not associated with greater television exposure or peritraumatic distress. We then controlled for these peritraumatic exposure and distress variables as potential confounders in a logistic regression model of mood state as a predictor of new PTSD. Mania/hypomania remained a significant predictor of probable PTSD in response to the September 11 attacks, suggestive of a substantial increase in risk compared with those in recovery (OR = 17; 95% CI = 2.6 to 115.6; p = .0034).

We also examined the potential confounding of the age and substance use associations with probable PTSD, by peritrauma exposure and distress. After statistical adjustment for peritrauma exposure and distress, the prediction offered by patient age (OR = 1.04; 95% CI = 0.999 to 1.08; p = .056) and by substance abuse (OR = 2.37; 95% CI = 0.94 to 6.00; p = .069) remained at the level of a trend.

DISCUSSION

We found that 20% of a sample of outpatients with bipolar disorder met criteria for probable new-onset PTSD in response to indirect exposure to the September 11, 2001, attacks. Probable PTSD was present at assessments occurring, on average, more than a year after the event. This represents a notably higher rate than has been reported in the general population, including those, for example, from the New York area followed up at 6 months after September 11 (0.6%[4]).

Potential limitations of our study should be acknowledged. Although the DSM-IV definition for trauma includes the possibility of witnessing, as well as directly experiencing, events involving actual or threatened death or harm, whether exposure through the media to the events of 9/11 as experienced by subjects in this study fulfills this criterion remains somewhat controversial. Although our data suggest that such indirect traumatic exposure may be sufficient to provoke a traumatic stress syndrome, structured clinical diagnostic evaluation, including an assessment of level of distress and disability, would be more definitive. While the diagnosis of new-onset PTSD in this study should be considered provisional given the self-report nature of the assessment instrument, our findings point to a marked increase in distress associated with indirect exposure to the events of 9/11 that may have been somewhat state dependent in bipolar individuals.

Another potential limitation is that assessments were accrued over a period of a year starting 6 months after September 11; however, while this is a relatively broad period of assessment, we controlled for this in the analysis and did not see evidence that time to assessment contributed significantly to the findings. Although we did not perform serial follow-ups to examine the persistence of PTSD over time, the presence of 9/11-related probable PTSD at least 6 months after September 11 suggests

it is unlikely that these symptoms represent transient phenomena.

This markedly elevated rate of probable PTSD among bipolar patients is consistent with the high rates of anxiety disorders previously reported in bipolar patients[13] and elevated rates of anxiety sensitivity and neuroticism among bipolar patients in general.[15,22] Although the particular vulnerability to the development of PTSD of bipolar patients in a manic, hypomanic, or mixed state when exposed to trauma remains to be fully elucidated, one possible mediator may be the presence of a high level of hyperarousal in manic states, as there is evidence that hyperarousal mediates the severity and course of PTSD symptoms over time.[23] Further, PTSD is associated with elevated fears of anxiety sensations (anxiety sensitivity).[24,25] Anxiety sensitivity predicts the emergence of panic in response to chronic stress or biological provocation procedures (for review, see reference 26), and we have recently reported that manic states (i.e., mania, hypomania, or mixed episodes) are associated with greater elevations in anxiety sensitivity than depression or euthymia in patients with mood disorders.[22] Accordingly, patients in manic states may be at greater risk for amplification of their anxiety experience, or panic, in response to traumatic events. Tentative evidence linking PTSD severity to the degree of fears of death and loss of control at the time of the trauma[27] supports the notion that an intensification of the anxiety experience at the time of the trauma, in the presence of a manic state, could be etiologically important in the emergence or maintenance of PTSD.

The presence of high rates of probable PTSD (50%) in our small sample of patients with a substance abuse diagnosis is consistent with prior reports[28] and suggests an increased burden of risk in this comorbid group. The finding that higher rates of probable PTSD occurred among older individuals is puzzling and generally contrary to other studies that have reported increased rates of PTSD associated with younger age or no age effect.[2,8,29] In this study, we were unable to assess other potential factors that may have an age cohort effect, such as prior direct or indirect exposure to war, terrorism, or combat situations. Overall however, the lack of strong association with a number of pretrauma demographic and comorbidity variables previously found to be risk factors for the development of PTSD in general populations including gender,[30] a history of previous traumas or PTSD,[31] education level, and socioeconomic status[32,33] suggests that the presence of a manic state may be the most critical risk factor for adverse sequelae following indirect exposure to a traumatic event in bipolar individuals.

REFERENCES

1. Schuster MA, Stein BD, Jaycox L, et al. A national survey of stress reactions after the September 11, 2001, terrorist attacks. N Engl J Med 2001; 345:1507–1512
2. Schlenger WE, Caddell JM, Ebert L, et al. Psychological reactions to terrorist attacks: findings from the National Study of Americans' Reactions to September 11. JAMA 2002;288:581–588
3. Galea S, Ahern J, Resnick H, et al. Psychological sequelae of the September 11 terrorist attacks in New York City. N Engl J Med 2002;346: 982–987
4. Galea S, Vlahov D, Resnick H, et al. Trends of probable post-traumatic stress disorder in New York City after the September 11 terrorist attacks. Am J Epidemiol 2003;158:514–524
5. Otto MW, Perlman CA, Wernicke R, et al. Posttraumatic stress disorder in patients with bipolar disorder: a review of prevalence, correlates, and treatment strategies. Bipolar Disord 2004;6:470–479
6. Darves-Bornoz JM, Lemperiere T, Degiovanni A, et al. Sexual victimization in women with schizophrenia and bipolar disorder. Soc Psychiatry Psychiatr Epidemiol 1995;30:78–84
7. Mueser KT, Goodman LB, Trumbetta SL, et al. Trauma and posttraumatic stress disorder in severe mental illness. J Consult Clin Psychol 1998;66:493–499
8. Neria Y, Bromet EJ, Sievers S, et al. Trauma exposure and posttraumatic stress disorder in psychosis: findings from a first-admission cohort. J Consult Clin Psychol 2002;70:246–251
9. Schnurr PP, Friedman MJ, Rosenberg SD. Premilitary MMPI scores as predictors of combat-related PTSD symptoms. Am J Psychiatry 1993; 150:479–483
10. Kessler RC, Sonnega A, Bromet E, et al. Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry 1995;52: 1048–1060
11. McFarlane AC. The aetiology of post-traumatic morbidity: predisposing, precipitating and perpetuating factors. Br J Psychiatry 1989;154:221–228
12. Udwin O, Boyle S, Yule W, et al. Risk factors for long-term psychological effects of a disaster experienced in adolescence: predictors of post traumatic stress disorder. J Child Psychol Psychiatry 2000;41:969–979
13. Simon NM, Otto MW, Wisniewski SR, et al. Anxiety disorder comorbidity in bipolar disorder patients: data from the first 500 participants in the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD). Am J Psychiatry 2004;161:2222–2229
14. Bagby RM, Bindseil KD, Schuller DR, et al. Relationship between the five-factor model of personality and unipolar, bipolar and schizophrenic patients. Psychiatry Res 1997;70:83–94
15. Jain U, Blais MA, Otto MW, et al. Five-factor personality traits in patients with seasonal depression: treatment effects and comparisons with bipolar patients. J Affect Disord 1999;55:51–54
16. Pfefferbaum B, Seale TW, McDonald NB, et al. Posttraumatic stress two years after the Oklahoma City bombing in youths geographically distant from the explosion. Psychiatry 2000;63:358–370
17. Sachs GS, Thase ME, Otto MW, et al. Rationale, design, and methods of the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD). Biol Psychiatry 2003;53:1028–1042
18. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association; 1994
19. Sachs GS. Use of clonazepam for bipolar affective disorder. J Clin Psychiatry 1990;51(suppl 5):31–34
20. Sheehan DV, Lecrubier Y, Sheehan KH, et al. The Mini-International Neuropsychiatric Interview (MINI): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. J Clin Psychiatry 1998;59(suppl 20):22–23
21. Foa E, Cashman L, Jaycox L, et al. The validation of a self-report measure of posttraumatic stress disorder: the Posttraumatic Diagnostic Scale. Psychol Assess 1997;9:445–451
22. Simon NM, Smoller JW, Fava M, et al. Comparing anxiety disorders and anxiety-related traits in bipolar disorder and unipolar depression. J Psychiatr Res 2003;37:187–192
23. Schell TL, Marshall GN, and Jaycox LH. All symptoms are not created equal: the prominent role of hyperarousal in the natural course of posttraumatic psychological distress. J Abnorm Psychol 2004;113:189–197
24. Lang AJ, Kennedy CM, Stein MB. Anxiety sensitivity and PTSD among female victims of intimate partner violence. Depress Anxiety 2002;16: 77–83
25. Taylor S, Koch WJ, McNally RJ. How does anxiety sensitivity vary across the anxiety disorders? J Anxiety Disord 1992;6:249–259
26. McNally RJ. Anxiety sensitivity and panic disorder. Biol Psychiatry 2002;52:938–946

27. Gershuny BS, Cloitre M, Otto MW. Peritraumatic dissociation and PTSD severity: do event-related fears about death and control mediate their relation? Behav Res Ther 2003;41:157–166
28. De Bellis MD. Developmental traumatology: a contributory mechanism for alcohol and substance use disorders. Psychoneuroendocrinology 2002;27:155–170
29. Gamper G, Willeit M, Sterz F, et al. Life after death: posttraumatic stress disorder in survivors of cardiac arrest: prevalence, associated factors, and the influence of sedation and analgesia. Crit Care Med 2004;32:378–383
30. Holbrook TL, Hoyt DB, Stein MB, et al. Gender differences in long-term posttraumatic stress disorder outcomes after major trauma: women are at higher risk of adverse outcomes than men. J Trauma 2002;53:882–888
31. Hanson RF, Kilpatrick DG, Freedy JR, et al. Los Angeles County after the 1992 civil disturbances: degree of exposure and impact on mental health. J Consult Clin Psychol 1995;63:987–996
32. Ullman SE, Filipas HH. Predictors of PTSD symptom severity and social reactions in sexual assault victims. J Trauma Stress 2001;14:369–389
33. Magruder KM, Frueh BC, Knapp RG, et al. PTSD symptoms, demographic characteristics, and functional status among veterans treated in VA primary care clinics. J Trauma Stress 2004;17:293–301