## ORIGINAL PAPER

# PTSD onset and course following the World Trade Center disaster: findings and implications for future research

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#### **Abstract**

Objective We sought to identify common risk factors associated with posttraumatic stress disorder (PTSD) onset and course, including delayed, persistent, and remitted PTSD following a major traumatic exposure.

Method Based on a prospective study of New York City adults following the World Trade Center disaster (WTCD), we conducted baseline interviews with 2,368 persons one year after this event and then at follow-up 1 year later to evaluate changes in current PTSD status based on DSM-IV criteria.

Results Baseline analysis suggested that current PTSD, defined as present if this occurred in the past 12 months, was associated with females, younger adults, those with lower self-esteem, lower social support, higher WTCD exposure, more lifetime traumatic events, and those with a history of pre-WTCD depression. At follow-up, current PTSD was associated with Latinos, non-native born persons, those with lower self-esteem, more negative life events, more lifetime traumatic events, and those with mixed handedness. Classifying respondents at follow-up

into resilient (no PTSD time 1 or 2), remitted (PTSD time 1, not 2), delayed (no PTSD time 1, but PTSD time 2), and persistent (PTSD both time 1 and 2) PTSD, revealed the following: compared to resilient cases, remitted ones were more likely to be female, have more negative life events, have greater lifetime traumatic events, and have pre-WTCD depression. Delayed cases were more likely to be Latino, be non-native born, have lower self-esteem, have more negative life events, have greater lifetime traumas, and have mixed handedness. Persistent cases had a similar profile as delayed, but were the only cases associated with greater WTCD exposures. They were also likely to have had a pre-WTCD depression diagnosis. Examination of WTCD-related PTSD at follow-up, more specifically, revealed a similar risk profile, except that handedness was no longer significant and WTCD exposure was now significant for both remitted and persistent cases.

Conclusion PTSD onset and course is complex and appears to be related to trauma exposure, individual predispositions, and external factors not directly related to the original traumatic event. This diagnostic classification may benefit from additional conceptualization and research as this relates to changes in PTSD status over time.

**Keywords** Posttraumatic stress disorder · Delayed-onset · Predisposition · Resiliency · Stressful events

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## Introduction

The 1980 inclusion of posttraumatic stress disorder (PTSD) in the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (DSM-III) [1], has resulted in numerous investigations [2–11]. Currently studies suggest that about 90% of adults have experienced at least one



lifetime traumatic event. Yet, only a minority of those exposed develop PTSD [12, 13]. Thus, factors beyond trauma exposure are required for PTSD onset [7, 14–17]. Research, for example, has consistently suggested that demographic characteristics such as age, gender, and race were associated with different rates of PTSD; with younger persons, women, and Latinos more likely to develop this disorder [4, 18–20]. Interpersonal and psychological characteristics, such as social support and self-esteem, have also been implicated in the onset and course of PTSD [4, 12, 15, 21]. Genetic and neurological factors have also been implicated [2, 17].

Delayed-onset PTSD was also introduced in DSM-III [1], and a number of studies have suggested that significant numbers of persons have experienced this type of onset [8, 22-24]. Recently it was reported that among a sample of 1,040 Somalia Peacekeepers, almost 7% were classified as delayed-PTSD cases, because they did not meet PTSD criteria 3 months after returning to the US, but did 12 months afterwards [21]. Another study recently reported a 3% prevalence of delayed PTSD 2 years after the World Trade Center disaster (WTCD) and 12 months after a baseline assessment [22]. Evidence has suggested that delayed PTSD is more prevalent among military veterans than among civilians exposed to non-combat stressors [24]. Few researchers, however, have prospectively assessed PTSD onset and course in a community sample or have fully explored specific clinical manifestations, including assessment of remitted, delayed, and persistent PTSD. Recent research focusing on these issues has suggested that post-trauma events, such as psychosocial resource losses are predictive of PTSD onset [22, 24]. It has also been noted that exposure to psychological trauma may intensify other negative social events, which can increase stress disorders or maintain existing ones [25]. There are also known preexisting, presumably biological factors, associated with PTSD, such as lower intelligence, non-right handedness/mixed handedness, attention deficit disorders (ADD), and having other pre-exposure neurological symptoms [2, 17, 26].

Previous community-based trauma studies, in general, have been limited in studying the onset and course of PTSD. First, many have been small and not representative of affected communities [18, 27]. Second, many studies often have not utilized standardized mental health measures [18]. Third, previous studies have tended to follow survivors of community disasters for only short periods of time (e.g., <12 months) or have been cross-sectional in design [16, 27]. Fourth, studies have investigated PTSD onset in both samples of veterans and civilians, but there has been little conceptual consistency [28–30]. Currently, DSM-IV describes delayed onset as a specifier for PTSD, whereby if symptoms meet criteria 6 months following the

traumatic exposure, but not before this time period, delayed PTSD is defined as present [31]. It has not gone unnoticed that the recognition of delayed PTSD has led to its acceptance as a compensated medical disorder by the U.S. Department of Veterans Affairs, which has led to increased benefit claims [28, 32]. However, the status of this diagnosis in VA disability compensation does not necessarily address the issue of the scientific and clinical merit of this diagnosis [28].

It has been noted that a limitation with the PTSD diagnosis relates to the criteria for delayed PTSD in the DSM [22, 28]. In particular, there has been no specification as to whether the onset of symptoms refers to any symptoms that might eventually lead to PTSD or only to the onset of "full-criteria" PTSD. In the latter interpretation, an individual with minimal initial symptoms that gradually worsened over time until the full PTSD criteria were met would not be classified as delayed PTSD. Given that it is common for trauma-exposed individuals to develop some initial symptoms [33], this definition of delayed-onset would mean that delayed PTSD would be extremely rare [28]. Alternatively, defining delayed PTSD as relating to a gradual symptom onset means that the occurrence of one additional symptom in a symptomatic individual could be sufficient for a "delayed" diagnosis, which is the current diagnostic standard [28]. To date, few studies investigating delayed PTSD explicitly interpret the DSM definition as ruling out any prior symptoms [29, 30].

Another issue concerns the time between trauma exposure and PTSD symptom onset. A number of investigators have not adhered to a delay of at least 6 months, but have used shorter time periods [28]. Other studies have specified onset delays of 12 months or more [22]. Retrospective studies of PTSD onset typically assess lifetime episodes with standardized clinical interviews to measure PTSD based on long-term recall, but are likely misleading [28]. Other researchers, however, have conducted prospective studies using interviews at predetermined assessment points [22]. This research design allows for assessment of both PTSD onset and course. In the current study, we used a prospective cohort design that collected data at one year and two years after the WTCD. We extended previous work by not only examining predictors of PTSD, but also the onset of delayed and remitted PTSD 2 years after this disaster, using a multinomial logit model among a large, community-based, population sample. Our research objective was to identify predictive factors that could explain both PTSD onset and remission and that occurred before, during, or after the index traumatic event, presumably responsible for the course of PTSD symptoms. This knowledge should aid in understanding PTSD onset among those exposed to traumatic events and in understanding its etiology, generally.



#### Data and methods

The data for the present research come from a prospective cohort study of English or Spanish speaking adults living in NYC on the day of the WTCD. For baseline, we conducted a diagnostic interview by telephone one year after the attacks. Subjects for this interview were recruited using random-digit dialing. This population sample was stratified by the 5 NYC boroughs and gender was sampled proportionately. Interviews were conducted in both English and Spanish. Baseline interviews occurred between October and December, 2002. For follow-up, we attempted to re-interview all baseline participants one year later (i.e., 2 years after the WTCD). All follow-up interviews occurred between October 2003, and February 2004. Trained interviewers using a computer-assisted telephone interviewing system conducted the surveys and were supervised by the survey contractor and the investigative staff. The Institutional Review Board (IRB) of the New York Academy of Medicine reviewed and approved the study's original protocols. Geisinger Clinic's IRB approved the current study analyses.

In total, 2,368 individuals completed the baseline interview and 1,681 completed the follow-up. Using standard survey response rate calculations [34], the baseline cooperation rate was approximately 63% and the re-interview rate for follow-up was 71% [35]. A sampling weight was developed for each wave to correct for potential selection bias related to the number of telephone numbers and persons per household and for the over-sampling of treatment-seeking respondents, which was part of the original study design. In addition, demographic weights also were used to adjust for slight differences in response rates by different demographic groups [36]. With these adjustments, our final study sample is representative of NYC adults who were living in NYC on the day of the WTCD [35]. Descriptions of this research have been presented elsewhere [14, 22, 35].

## PTSD assessment

Our PTSD scale was based on the *Diagnostic and statistical manual of mental disorders*, *Fourth Edition* (DSM-IV) [31]. This PTSD measure was developed for telephone administration and used in previous trauma studies [16, 37–39]. To meet PTSD criteria, a respondent had to be exposed to a traumatic event (Criteria A1) and had to have experienced intense feelings of fear, helplessness, or horror (Criteria A2), as well as had to have met Criteria B (re-experiencing), C (avoidance), and Criteria D (increased arousal). In addition, the symptoms for Criteria B, C, and D had to last 1 month or longer (Criteria E) and had to have a

negative impact on functional status or had to have caused significant distress (Criteria F). The focus of this study is on the occurrence of current PTSD, defined as onset within the past 12 months. Overall, our PTSD classification involved assessing three sets of events: the WTCD event, the most significant event other than the WTCD, and any other traumatic event. Our baseline and follow-up PTSD assessments covered the year prior to the date of interview. Results supporting the validity of our PTSD instrument have been reported elsewhere [16, 22, 39, 40].

For our examination of PTSD onset and remission at follow-up, we adapted the categories developed by Gray et al. [21], which included resilient, remitted, delayed, and persistent PTSD. Given that our respondents were living in NYC at the time of the attacks and could be considered at risk for PTSD, resilient cases were those who did not meet PTSD criteria at either baseline or follow-up. Remitted PTSD cases met criteria at baseline, but not at follow-up. Those individuals categorized as delayed PTSD cases, did not meet criteria at baseline, but met criteria at follow-up. Persistent PTSD cases met criteria at both baseline and at follow-up assessments. Finally, for our PTSD onset and remission classifications we grouped these data in two different ways: First, we classified this outcome for any source of PTSD (i.e., WTCD-related, worst event other than WTCD, and any other event) and, second, we classified this in terms of WTCDrelated PTSD. The results are presented for both types of PTSD classifications. As reported elsewhere, however, in our study it is important to note that given the impact of the WTCD event in NYC, current PTSD and WTCD-related PTSD tended to overlap [39, 40].

## **Predictor variables**

In our analyses, we focused on factors that would aid in assessing changes in PTSD status over time. All of the demographic variables were based on baseline data, unless the data were missing, in which case, the follow-up data were substituted. In addition, unless otherwise noted, all variables were measured the same way for baseline and follow-up. Finally, all of the stress, risk, and resource variables come from baseline for baseline PTSD and follow-up for follow-up PTSD, except where noted.

## Demographic characteristics

Based on previous research [22], our analyses included four demographic variables: age, female gender, married status, and race/ethnicity. Age was coded into four categories, 18-29, 30-44, 45-64, and 65+ for cross-tabulations and coded in decades for multivariate analyses (baseline mean = 43.3; SD = 20.4). Female gender and married



status (coded for married) were represented as binary variables. Consistent with most research [13], race/ethnicity was self-identified. For multivariate analyses, we used Latino only as a binary variable, since previous studies suggested this was the most significant predictor of psychological distress [14].

## Stress exposure variables

Our statistical models included three stressors that could have placed an individual at risk for mental health symptoms and PTSD. The first stressor variable, WTCD event exposure, was based on the baseline survey. This scale consisted of 14 possible events that the respondent could have experienced during the attacks [39]. We summed these events and coded them for cross-tabulations into low exposure (0–1 event), moderate exposure (2–3 events), high exposure (4-5 events), and very high exposure (6+ events). For multivariate analyses we coded this measure as a continuous variable (baseline mean = 2.0; SD = 1.6). Second, a negative life event scale was the sum of eight experiences that the respondent could have had in the 12 months before the WTCD for baseline (e.g., divorce, death of spouse) and since the WTCD for follow-up [25]. For multivariate analyses we used this as a continuous variable (baseline mean = 0.7; SD = 1.1). The third stressor measure focused on 10 traumatic events (e.g., forced sexual contact, being attacked with a weapon) that could have occurred anytime prior to the baseline survey or in the year preceding the follow-up survey [25]. For each wave, the traumatic events were used as continuous variables in multivariate analyses (baseline mean = 1.7; SD = 2.3).

#### Psychosocial resource variables

Our study included two variables related to psychosocial resources: social support and self-esteem. Social support was the sum of four questions about emotional, informational, and instrumental support currently available to the respondent (e.g., someone available to help if confined to bed), each categorized as a 4-point Likert scale [41]. This scale was used as a continuous variable for multivariate analysis (baseline mean = 6.9; SD = 4.6). Self-esteem was measured by the Rosenberg self-esteem scale [42]. This measure was the sum of five items in the original scale (e.g., on the whole, I am satisfied with myself), each categorized as a 4-point Likert scale and used as a continuous variable for multivariate analysis (baseline mean = 17.9; SD = 3.2). Although this scale was shortened from its original version, the reliability and validity of this modified instrument appeared excellent [14, 22, 35, 39], as were the other stressor/risk and resource measures used, which were discussed elsewhere in detail [16, 22, 39, 40].

## History of lifetime depression

In order to account for psychological vulnerabilities that could have existed prior to the WTCD, we assessed respondents for lifetime depression at baseline. Using a version of the *Structured clinical interview for DSM-III-R's* (SCID's) major depressive disorder scale [43], and consistent with the DSM-IV, respondents met criteria for lifetime depression if they ever had five or more depression symptoms for at least two-weeks. For this measure we used lifetime depression that occurred before the WTCD event based on age of onset. This measure has been used in other telephone-based population surveys of trauma survivors [16, 44]. Data related to the validity of this scale were previously reported and suggested that this scale can successfully diagnose depression in the general population [39, 40].

#### Perievent panic attack

Our study assessed whether the respondents met criteria for experiencing a perievent panic (PEP) attack during the WTCD event. This measure was based on the Diagnostic Interview Schedule (DIS) [45]. For our PEP measure, questions were phrased to assess panic symptoms that occurred during or shortly after the WTCD [16]. The presence of four or more symptoms classified the person as having a PEP attack, if these symptoms reached their peak within 10 min of onset [45]. This variable was coded as a binary measure. This PEP measure has been used and validated in previous WTCD studies [14, 16, 39, 46, 47]. Using this measure, it was previously reported that approximately 11% of New York City (NYC) adults experienced a PEP attack during the WTCD event [39].

## Neurological risk factors

Based on previous research [17], we also included two other predictors in our multivariable models, including mixed handedness and history of attention deficient disorder (ADD). For handedness, we asked respondents to report whether they considered themselves generally right handed, left handed, or both right and left handed (i.e., mixed handedness) [17]. Approximately 6% of respondents reported that they were mixed handed. For ADD classification, we asked respondents to report if a doctor ever told them they had a list of common medical conditions, including ADD. If they reported yes for this condition, they were classified as having ADD. This method of collecting data for medical conditions is routinely used in national



health surveys and has been reported to be generally accurate [48]. Approximately 2% of respondents reported having a history of ADD.

## **Descriptive variables**

We also used three other descriptive variables in our study related to current depression and psychiatric symptoms. The psychiatric symptom measures were based on the Brief Symptom Inventory-18 (BSI-18), a psychiatric scale derived from the Hopkins Symptom Checklist (HSCL) [49]. The BSI-18 has been standardized based on a community sample and has clinical cut-off scores to define cases [49]. We used a T-score of 65 or higher for case definition, representing a symptom score above the 90th percentile. The reliability and validity for the BSI-18 is reported to be high [49]. In the current study, we report the results only for anxiety and somatization. For current depression, we used the SCID depression measure discussed above to define depression as present at baseline and at follow-up, based on meeting the depression criteria in the past 12 months, respectively.

#### Statistical analysis

As reported elsewhere [35], analyses comparing the weighted baseline sample and U.S. Census data for NYC indicated that the sample was representative of NYC. Since the bivariate association between PTSD and our predictor variables have been reported elsewhere in detail [22, 39], we do not review these findings in the current study. Instead, we focus on multivariate models where we regressed baseline and follow-up PTSD, respectively, on study predictor variables. We note that a study focus was on the occurrence of any PTSD in the past 12 months, which included both WTCD-related and non-WTCD-related PTSD. However, given the impact of the WTCD event in our study, as we note below, most of the PTSD that occurred during the study's timeframe was related to the WTCD event. Finally, we examined factors related to remitted, delayed, and persistent PTSD, compared to resilient cases, using multi-nomial logit regression (MNLR). This statistical method is the equivalent of simultaneously estimating binary logits for all PTSD comparisons among the alternatives compared to a base model [50]. For comparison purposes, as noted, we conducted this analysis both for any PTSD and for WTCDrelated PTSD more specifically. Our analytical focus was guided by a general psychobiological stress model that we have used previously [2, 10, 22, 35], as well as general risk factor models from disaster epidemiology [16, 25]. For our analyses, we used the survey estimation (svy) command set in Stata, version 9.2 [51]. This estimation procedure adjusts the data for our sampling design, which included oversampling, stratification by borough and gender and, as noted earlier, case weights [35].

#### Results

Our summary demographic data for baseline and follow-up are presented in Table 1 (top panel). None of these minor demographic differences shown in Table 1 were found to be statistically significant. Also shown are WTCD exposure levels and the prevalence of current PTSD, depression, anxiety and current somatization at baseline and follow-up. As can be seen, the prevalence of adverse mental health outcomes did not increase during the follow-up period (Table 1, bottom panel). Noteworthy is that these data indicated that lifetime depression before the WTCD was 18.3% and that the majority of PTSD cases that occurred at baseline and follow-up appeared to be WTCD-related (for example, at baseline WTCD PTSD = 3.2% vs. any PTSD = 4.6%; at follow-up WTCD PTSD = 3.9 vs. any PTSD = 5.4%).

The multivariate logistic regression results predicting baseline and follow-up PTSD, respectively, are shown in Table 2. As can be seen, there are significant associations between PTSD at baseline and being female, being younger, having lower self-esteem, having lower social support, having greater WTCD event exposures, having greater lifetime traumatic events, and having a history of depression before the WTCD (all *P*-values < 0.05). However, the results were different for PTSD at follow-up. For example, at follow-up, being Latino, being nonnative born, having lower self-esteem, having more negative life events, having higher traumatic life events, and having mixed handedness, were now significant predictors of PTSD (all *P*-values < 0.05).

As discussed, to address limitation in past studies, we conducted multinomial logit regression (MNLR) analysis using the predictor variables described, with remitted, delayed, and persistent PTSD as the included categories and resilient cases as the reference category (Table 3). Since PEP and marital status were not significant in the logistic regressions, we eliminated these variables in the MNLR. This MNLR model predicting prospective changes in PTSD status suggested that, compared to resilient cases, remitted cases were more likely to be female (risk ratio [RR] = 3.85, P < 0.001), to have more negative life events (RR = 1.37, P = 0.037), greater lifetime traumatic events (RR = 1.41, P < 0.001), and to have had pre-WTCD depression (RR = 3.98, P < 0.001). Delayed-PTSD cases were more likely to be Latino (RR = 2.45,



Table 1 Study population characteristics at baseline versus follow-up<sup>a</sup>

Study variables	1 year post disaste	r (N = 2,368)	2 years post disaster ( $N = 1,681$ )			
	Percent (N)	95% CI	Percent (N)	95% CI		
Age						
18–29	27.2 (483)	24.8-29.7	22.7 (284)	20.1-25.6		
30–44	34.2 (866)	31.8-36.7	32.9 (596)	30.1-35.8		
45–64	28.8 (726)	26.5-31.1	32.5 (586)	29.8-35.4		
65+	9.8 (248)	8.5-11.4	11.9 (215)	10.1-13.9		
Gender						
Male	46.2 (1016)	43.6-48.8	46.2 (693)	43.2-49.3		
Female	53.8 (1352)	51.2-56.4	53.8 (988)	50.7-56.9		
Race						
White	39.3 (1015)	36.9-41.7	43.0 (782)	40.1-45.9		
African-American	26.3 (606)	24.2-28.6	26.0 (422)	23.4-28.7		
Hispanic/Latino	25.7 (559)	23.5-28.1	24.1 (367)	21.5-26.9		
Asian	5.2 (99)	4.1-6.6	4.6 (62)	3.4-6.1		
Other	3.5 (89)	2.7-4.5	2.4 (48)	1.7-3.4		
Borough of residence						
Manhattan	21.1 (555)	19.8-22.5	21.0 (411)	19.5-22.6		
Bronx	15.6 (373)	14.4–16.8	15.5 (252)	14.1-17.0		
Brooklyn	30.1 (707)	28.5-31.7	30.3 (490)	28.4-32.2		
Queens	27.9 (594)	26.4-29.4	27.7 (423)	26.0-29.5		
Staten Island	5.4 (139)	4.7-6.2	5.5 (105)	4.65-6.3		
Exposure to WTCD						
Low exposure	26.5 (510)	24.2-28.9	26.7 (362)	24.0-29.61		
Moderate exposure	44.0 (1003)	41.4–46.6	43.9 (719)	40.9-47.0		
High exposure	22.0 (594)	20.0-24.2	21.8 (416)	19.4-24.4		
Very high exposure	7.5 (261)	6.4-8.8	7.6 (184)	6.3-9.1		
Depression past year	11.8 (416)	10.4–13.3	11.6 (277)	9.9-13.4		
Lifetime depression before WTCD	18.3 (588)	16.6–20.2	18.3 (420)	16.2-20.5		
PTSD past year—any	4.6 (174)	3.7–5.6	5.4 (134)	4.3-6.7		
PTSD past year—WTCD-related	3.2 (127)	2.5-4.1	3.9 (106)	3.0-4.9		
BSI anxiety past month	10.2 (362)	8.9-11.6	9.1 (217)	7.6–10.9		
BSI somatization past month	10.8 (359)	9.412.3	11.2 (249)	9.6-13.2		
SF-12 poor physical health past month	7.0 (201)	5.9-8.4	7.5 (163)	6.2-9.2		

WTCD World Trade Center disaster, CI confidence interval, BSI Brief Symptom Inventory

P=0.005), non-native born (RR = 1.86, P=0.041), to have lower self-esteem (RR = 0.77, P<0.001), more negative life events (RR = 1.97, P<0.001), greater lifetime traumas (RR = 1.22, P=0.003), and to have mixed handedness (RR = 2.46, P=0.028). By comparison, persistent cases were more likely to be female (RR = 2.80, P=0.031), Latino (RR = 2.54, P=0.033), non-native born (RR = 2.73, P=0.026), to have lower self-esteem (RR = 0.75, P<0.001), greater negative life events (RR = 2.20, P<0.001), greater WTCD exposure (RR = 1.70, P=0.001), greater lifetime trauma exposure (RR = 1.40, P<0.001), mixed handedness (RR = 4.63,

P = 0.012), and to have a history of pre-WTCD depression (RR = 4.08, P = 0.003).

A focus of this study was on changes in PTSD status over time. Thus, we also ran the MNLR model based on WTCD-related PTSD. Since the onset of PTSD in the current study was substantially related to the WTCD event, these results were very similar to the results for any (i.e., all cause) PTSD as reported for Table 3. As can be seen in Table 4, the major difference was that handedness was no longer significant and that WTCD exposure now was significant for both remitted and persistent PTSD. Another difference was that younger age was now protective for



<sup>&</sup>lt;sup>a</sup> Percents are based on weighted data; Ns are unweighted

**Table 2** Logistic regressions predicting any PTSD in past 12 months at baseline/follow-up (N = 1,681)

Predictor variables	Year 1 po	ost disaster		Year 2	Year 2 post disaster			
	OR	95% CI	P-value	OR	95% CI	P-value		
Female	3.64	1.94-6.82	< 0.001	1.18	0.68-2.05	0.562		
Age	0.78	0.63-0.97	0.026	1.14	0.95-1.37	0.154		
Latino	1.15	0.58-2.31	0.683	2.33	1.28-4.24	0.006		
Married	0.58	0.32 - 1.05	0.074	0.87	0.50-1.50	0.611		
Non-native born	0.94	0.52-1.72	0.856	1.95	1.12-3.41	0.019		
Panic attack	1.36	0.65 - 2.84	0.419	1.78	0.87-3.63	0.112		
Self-esteem	0.88	0.81-0.95	0.001	0.77	0.71-0.84	< 0.001		
Social support	0.90	0.83-0.97	0.007	1.05	0.96-1.15	0.314		
Negative life events	1.20	0.95-1.52	0.130	1.92	1.47-2.52	< 0.001		
WTCD exposure	1.34	1.10-1.63	0.004	1.19	0.95-1.48	0.124		
Lifetime trauma exp.	1.33	1.13-1.55	< 0.001	1.19	1.06-1.34	0.004		
Mixed handedness	1.80	0.73-4.47	0.203	2.61	1.24-5.48	0.011		
Attention deficit	1.60	0.63-4.06	0.323	2.45	0.82-7.33	0.110		
Lifetime depression pre-WTCD	3.30	1.75-6.23	< 0.001	1.21	0.64-2.31	0.559		
PTSD outcomes—Year 2 <sup>a</sup>				%	(N)	95% CI		
Resilient cases	_	_	_	91.7	(1,468)	90.2–93.1		
Remitted cases	_	_	_	2.9	(79)	2.2-3.9		
Delayed cases	_	_	_	4.4	(98)	3.4-5.7		
Persistent cases	_	_	_	0.9	(36)	0.6-1.3		

WTCD World Trade Center disaster, CI confidence interval, OR odds ratio

Table 3 Multi-nomial logit regression predicting any current PTSD at follow-up (N = 1,681)

Variables	Remitted cases $(n = 79)$			Delayed cases $(n = 98)$			Persistent cases $(n = 36)$			Overall <i>P</i> -value <sup>b</sup>
	RR <sup>a</sup>	95% CI	P-value	RR <sup>a</sup>	95% CI	P-value	RRa	95% CI	P-value	
Female	3.85	1.99-7.49	< 0.001	1.28	0.71-2.33	0.408	2.80	1.10-7.12	0.031	0.002
Age	0.79	0.61-1.01	0.063	1.10	0.92 - 1.32	0.300	1.10	0.82 - 1.48	0.536	0.061
Latino	1.33	0.57 - 3.12	0.512	2.45	1.31-4.58	0.005	2.54	1.08-5.99	0.033	0.024
Non-native born	0.90	0.42 - 1.93	0.788	1.86	1.03-3.38	0.041	2.73	1.13-6.59	0.026	0.026
Self-esteem	0.94	0.86 - 1.03	0.193	0.77	0.70 – 0.84	< 0.001	0.75	0.67 - 0.85	< 0.001	< 0.001
Social support	0.96	0.86 - 1.07	0.437	1.04	0.95 - 1.14	0.428	0.98	0.87 - 1.11	0.731	0.061
Negative life events	1.37	1.02 - 1.87	0.037	1.97	1.47-2.65	< 0.001	2.20	1.58-3.06	< 0.001	< 0.001
WTCD exposure	1.20	0.95 - 1.52	0.135	1.19	0.94-1.51	0.146	1.70	1.25-2.31	0.001	0.002
Lifetime trauma exp.	1.41	1.21-1.63	< 0.001	1.22	1.07-1.40	0.003	1.40	1.17-1.67	< 0.001	< 0.001
Mixed handedness	1.58	0.52 - 4.77	0.416	2.46	1.10-5.47	0.028	4.63	1.40-15.33	0.012	0.029
Attention deficit	1.35	0.37-4.98	0.650	2.52	0.78 - 8.15	0.123	2.88	0.81-10.26	0.102	0.146
Lifetime dep. pre-WTCD	3.98	2.06-7.68	< 0.001	1.23	0.61-3.46	0.567	4.08	1.62-10.31	0.003	< 0.001

WTCD World Trade Center disaster, CI confidence interval

remitted PTSD and older age was a risk factor for delayed PTSD (Table 4).

Overall assessment of the predictor variables in MNLR is given by the likelihood ratio tests shown in Tables 3

and 4. These tests are equivalent to assessing the difference in the model without each predictor variable, respectively, against the full model with all the variables added; hence this assesses the net effect of adding each variable,



<sup>&</sup>lt;sup>a</sup> Percents are based on weighted data; Ns are unweighted

<sup>&</sup>lt;sup>a</sup> Relative risk ratio (RR) compared to resilient cases (N = 1,468)

<sup>&</sup>lt;sup>b</sup> Based on the likelihood ratio test results

**Table 4** Multi-nomial logit regression predicting WTCD-related PTSD at follow-up (N = 1,681)

Variables	Remitted cases $(n = 63)$			Delayed cases $(n = 81)$			Persistent cases $(n = 25)$			Overall P-value <sup>b</sup>
	RR <sup>a</sup>	95% CI	P-value	RR <sup>a</sup>	95% CI	P-value	RR <sup>a</sup>	95% CI	P-value	
Female	2.99	1.48-6.03	0.002	1.27	0.66-2.43	0.469	2.98	1.02-8.66	0.045	0.022
Age	0.78	0.61-0.99	0.041	1.32	1.10-1.58	0.003	1.27	0.95 - 1.69	0.109	0.039
Latino	1.58	0.62-4.03	0.336	2.56	1.27-5.18	0.008	2.82	1.13-7.03	0.026	0.004
Non-native born	1.38	0.64-2.99	0.417	2.28	1.13-4.57	0.021	1.67	0.58-4.81	0.340	0.048
Self-esteem	0.98	0.88 - 1.09	0.703	0.79	0.71 - 0.88	< 0.001	0.74	0.66-0.84	< 0.001	< 0.001
Social support	0.96	0.85 - 1.08	0.481	1.00	0.91-1.12	0.871	0.93	0.80-1.08	0.357	0.196
Negative life events	1.41	1.01-1.97	0.046	1.67	1.22-2.27	< 0.001	1.95	1.30-2.91	0.001	< 0.001
WTCD exposure	1.44	1.15-1.81	0.002	1.21	0.92 - 1.58	0.176	1.90	1.40-2.58	< 0.001	< 0.001
Lifetime trauma exp.	1.28	1.12-1.47	< 0.001	1.24	1.08-1.43	0.003	1.33	1.12-1.58	0.001	< 0.001
Mixed handedness	2.07	0.72 - 5.99	0.179	1.54	0.66-3.61	0.321	3.99	0.79-20.25	0.094	0.142
Attention deficit	1.31	0.35-4.94	0.691	1.98	0.69-5.67	0.204	1.83	0.37-9.02	0.460	0.303
Lifetime dep. Pre-WTCD	4.95	2.46-9.96	< 0.001	1.34	0.63-2.88	0.445	4.70	1.82-12.14	0.001	< 0.001

WTCD World Trade Center disaster, CI confidence interval

respectively. As can be seen for any PTSD (Table 3), female gender was significant (P = 0.002), as was being Latino (P = 0.024), non-native born (P = 0.026), having lower self-esteem (P < 0.001), greater negative life events (P < 0.001), higher WTCD exposure (P = 0.002), greater lifetime traumatic events (P < 0.001), mixed handedness (P = 0.029),and having pre-WTCD depression (P < 0.001), suggesting these predictors made a significant contribution in the model. For WTCD-related PTSD (Table 4), the main difference is that handedness was no longer significant and age was now significant in the model. We also assessed overall model fit for our MNLR using the "fitstat" procedure in Stata. This resulted in an overall likelihood ratio of 319.8 (P < 0.0001) and a McFadden's adjusted  $R^2 = 0.20$ , suggesting adequate fit for the all-cause MNLR PTSD model [50]. These overall model results were slightly less for the WTCD-related PTSD model. For example, for WTCD-related PTSD model the likelihood ratio = 243.6 (P < 0.0001) and the McFadden's adjusted  $R^2 = 0.17$ .

## Discussion

Consistent with previous research [7, 16, 22, 52], at baseline logistic regression analysis showed that females, younger persons, those with lower self-esteem, lower social support, greater WTCD exposures, greater lifetime traumatic exposures, and those with a history of pre-WTCD depression were more likely to have current PTSD. At follow-up, however, this regression showed that Latinos, nonnative born persons, those with lower self-esteem, more

negative life events, higher traumatic life events, and those with mixed handedness, were more likely to meet the criteria for current PTSD.

However, MNLR provided more information related to symptom manifestation over time for both any current PTSD and for WTCD-related PTSD. In particular, compared to PTSD resilient cases, remitters were more likely to be females, have greater negative life events, have greater lifetime traumatic exposures, and to have a history of pre-WTCD depression. By comparison, delayed-onset cases tended to be Latinos, be immigrants, have lower self-esteem, have greater negative life events, have more lifetime traumatic events, and to have mixed handedness. Persistent cases were more likely to be females, be Latinos, be immigrants, have lower self-esteem, have greater negative life events, have greater exposure to WTCD events, have more lifetime traumatic exposures, have mixed handedness, and to have pre-WTCD depression. For WTCD-related PTSD, the results were similar except that handedness was no longer significant and that WTCD exposure now was significant for both remitted and persistent PTSD cases. Another difference was that younger age was protective for remitted PTSD and older age was a risk factor for delayed PTSD. In summary, these results seem to suggest that changes in PTSD status over time and later onset PTSD were not simply due to minor fluctuations in symptom status, but also reflected the impact of other pre- and post-trauma psychosocial factors. For WTCD-related PTSD, the effect of exposure to this event had more impact and the effect of predisposing factors, such as handedness, had less impact.

It has been suggested that delayed PTSD, defined as meeting the criteria 6 months or more post trauma



<sup>&</sup>lt;sup>a</sup> Relative risk ratio (RR) compared to resilient cases (N = 1.512)

<sup>&</sup>lt;sup>b</sup> Based on the likelihood ratio test results

regardless of previous PTSD symptom status, accounts for a significant number of PTSD cases in both military and civilian populations [28]. If a conservative definition were used, namely that the onset of PTSD symptoms that manifest themselves are delayed by at least 6 months, but not before this time, delayed-onset PTSD would be extremely rare, since most persons present with at least some symptom immediately post exposure [28]. However, when the more inclusive definition is used, this is not the case at all. Consequently, the delayed-onset war veteran is not uncommon in the VA system [53]. In fact, this diagnosis is sufficiently common to raise questions scientifically about what causes some individuals with few initial symptoms to remain vulnerable to PTSD over long time periods [22]. A recent study of soldiers medically evacuated from combat in Iraq and Afghanistan suggests that severe injuries tend to retard the development of PTSD, and this may account for some delayed cases [54]. Previously, some predicted significant rates of delayed stress reactions among veterans after the Vietnam War, because of emotional numbing, troop management, and military training at the time [55, 56]. However, over the past two decades "delayed" PTSD has evolved into a fully compensated medical disorder in the VA heath care system, now one of the most common in this system [28, 53].

Simply from a measurement perspective, changes in diagnostic status related to delayed PTSD might be due to an underreporting of symptoms at the initial assessment or an over-reporting of symptoms at later assessments. The opposite could be the case for remitted PTSD. There has also been speculation that delayed-PTSD may result from classical conditioning of fear and anxiety responses to trauma-related cues, reinforcing avoidance and re-experiencing symptoms [21]. However, for war veterans at least, the VA compensation system might be an equally plausible explanation for this "delayed" diagnosis [53]. Finally, and in line with our findings, changes in diagnosis may reflect changes in the victim's psychosocial circumstances, whereby exposure to negative life events and events leading to lower self-esteem may result in actual increased PTSD symptom manifestation [57]. We also showed that underlying vulnerabilities related to preexisting conditions, such as having a history of pre-exposure depression, contributes to the onset and course of PTSD post-exposure.

The association of mixed handedness with PTSD deserves further comment. It has been hypothesized that those exposed to psychological trauma with a lesser degree of cerebral lateralization have a greater likelihood of developing PTSD, because the right brain hemisphere is thought significant in threat identification and in the regulation of emotional response. Persons with reduced cerebral lateralization for language, *as* indexed by mixed-handedness, are thought to be more sensitive to perceived threat and prone to

experience emotions more intensely, because their cerebral organization is thought to give greater primacy to right hemisphere contributions in cognitive processes [17]. Noteworthy was that this finding did not hold for WTCD-related PTSD, which appeared to be more susceptible, understandably, to exposure to WTCD events (Table 4).

Additional research on individuals who do not meet full-PTSD criteria, but nevertheless, have many PTSD symptoms may provide additional insights [22]. Referred to as partial PTSD [58] or subsyndromal PTSD [59], these classifications typically require individuals to have a certain number of symptoms from Criteria B, C, and D. These classifications are not without controversy [22], but it has been noted that individuals meeting criteria for partial-PTSD have some impairment in work and social interaction domains, though not nearly the level exhibited by persons meeting full-criteria [58]. Thus, examining how changes in psychosocial status relate to changes in the number and severity of symptoms for individuals not meeting full criteria may further illuminate the course and onset of PTSD [22]. Similarly, as with many other mental health disorders [61], examination of cases above or below the diagnostic threshold can be insightful and points to the limits of only focusing on the full-criteria for PTSD. While studies exploring different classification schemes for delayed PTSD could be insightful, an earlier study suggested that on average there was a 6-point mean change in PTSD symptom scores between baseline (year 1) and follow-up (year 2) timeframes that were used to define remitted and delayed cases [22]. The latter finding seems to suggest that we can reject the hypothesis that only slight changes in PTSD symptoms over time account for most delayed PTSD. Other explanations seem warranted, some of which we highlighted above.

Finally, the reason why some individuals might experience more negative life events and diminished selfesteem in the post-disaster period is not clear. Some argue that these periods can be characterized as adverse environments in some communities [22, 60]. In their study of the economic and social consequences of the Exxon Valdez oil spill in the Prince William Sound, Palinkas et al. [62, 63] noted that this environmental disaster was not particularly life threatening. Nevertheless, this event disrupted subsistence production, strained family and community relations, and increased social inequality. Individuals living in communities directly affected by a traumatic event (e.g., lower Manhattan after the WTCD) may cause some persons to experience more negative life events (e.g., job loss, family problems, health problems, etc.), which could increase their PTSD symptoms after the traumatic exposure [22]. In addition, it is entirely possible that trauma victims, such as war veterans, might remain subclinical for long periods only to meet full criteria following a triggering



event indirectly related to the original trauma and quite distally removed [28, 48].

Our research has several limitations. First, we omitted individuals without telephones, those who did not speak English or Spanish, and those too disabled to undertake the interview or who were institutionalized. Given that the sample matched the 2000 Census for NYC, elimination of these persons did not appear to introduce overall demographic bias. We are limited, though, in generalizing these findings beyond the major ethnic groups in NYC. Additionally, our mental health measures were based on selfreport. Although there has been significant progress in assessing mental health with standardized instruments administered by interviewers [3, 7], there continues to be discrepancies between interviewer and clinician-based assessments [14]. In addition, it has been recently noted that participation in trauma-focused investigations in a baseline interview may result in sensitization of health problems at follow-up, increasing symptom reporting [64]. Another limitation was that our timeframe included only a 1-year baseline and a 1-year follow-up. A longer timeframe would have been preferred in our study, but this option was not available. Conversely, assessment of PTSD more frequently than every 12 months would have also been desirable for measuring fluctuations in PTSD status. Given our limited timeframes and the unresolved issues surrounding delayed PTSD, we note that the term "later onset" PTSD may have been equally applicable in our study. Finally, although our sample matched the NYC adult population, as noted elsewhere, our response rate was less than optimal [35]. The strengths of the study, however, include our focus on a time-limited traumatic event, the use of a large random and representative community sample, the assessment of mental health status using DSM-IVbased measures, the implementation of a prospective study design, and the use of advanced multivariate methods. To our knowledge, this is the only study of PTSD onset and course to date that has combined all of these methods into a single study.

The current study suggests that PTSD onset and course following a defined, time-limited event, such as the WTCD, represent complex phenomena that include both psychosocial and environmental factors, both related and unrelated to the indexed traumatic event, as well as pre-existing vulnerabilities. Given our findings, we suggest that additional information be included with the onset of PTSD and its diagnoses, including past mental health and medical history, previous syndromal status, length of time from the index trauma to symptom onset, the presence of potential "triggering" events, and significant changes in psychosocial resource variables. Without these additional clinical data, it is difficult to conceptualize how significant progress might be made in this field. In short, our findings suggest

that the diagnosis of PTSD onset and course, and especially delayed-onset PTSD, should probably be used more cautiously at this time, pending additional conceptualization and further research.

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#### References

- American Psychiatric Association (1980) Diagnostic and statistical manual of mental disorders, 3rd edn. American Psychiatric Association, Washington, DC
- Boscarino JA (2008) Psychobiologic predictors of disease mortality after psychological trauma: implications for research and clinical surveillance. J Nerv Ment Dis 196:100–107
- Adams RE, Bromet EJ, Panina N, Golovakha E, Goldgaber D, Gluzman S (2002) Stress and well-being after the Chornobyl nuclear power plant accident. Psychol Med 32:143–156
- Brewin CR, Andrews B, Valentine JD (2000) Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. J Consult Clin Psychol 68:748–766
- Bromet EJ, Parkinson DK, Schulberg HC, Dunn LO, Gondek PC (1982) Mental health of residents near the Three Mile Island reactor: a comparative study of selected groups. J Prev Psychiatry 1:225–276
- McFarlane AC (1988) Aetiology of post-traumatic stress disorders following a natural disaster. Br J Psychiatry 152:116–121
- Kessler RC, Sonnega A, Bromet E, Hughes H (1995) Posttraumatic stress disorder in the national comorbidity survey. Arch Gen Psychiatry 52:1048–1060
- McFarlane A (2004) The contribution of epidemiology to the study of traumatic stress. Soc Psychiatry Psychiatr Epidemiol 39:874–882
- Yehuda R (2002) Post-traumatic stress disorder. N Engl J Med 346:108–114
- Boscarino JA (2004) Association between posttraumatic stress disorder and physical illness: results and implications from clinical and epidemiologic studies. Ann NY Acad Sci 1032:141– 153
- 11. Kessler RC (2000) Posttraumatic stress disorder: the burden to the individual and to society. J Clin Psychiatry 61(Suppl 5):4–12
- Breslau N, Peterson EL, Schultz LR, Lucia VC (2004) Estimating post-traumatic stress disorder in the community: lifetime perspective and the impact of typical traumatic events. Psychol Med 34:889–898
- Breslau J, Kendler KS, Su M, Gaxiola-Aguilar S, Kessler RC (2005) Lifetime risk and persistence of psychiatric disorders across ethnic groups in the United States. Psychol Med 35:317– 327
- Adams RE, Boscarino JA (2005) Differences in mental health outcomes among Whites, African Americans, and Hispanics following a community disaster. Psychiatry 68:250–265
- Boscarino JA (1995) Post-traumatic stress and associated disorders among Vietnam veterans: the significance of combat exposure and social support. J Traumatic Stress 8:317–336
- Galea S, Ahern J, Resnick H, Kilpatrick D, Bucuvalas M, Gold J, Vlahov D (2002) Psychological sequelae of the September 11 terrorist attacks in New York City. N Engl J Med 346:982–987



- Boscarino JA, Hoffman SN (2007) Consistent association between mixed lateral preference and PTSD: confirmation among a national study of 2,490 US Army Vietnam veterans. Psychosom Med 69:365–369
- Bromet EJ, Dew MA (1995) Review of psychiatric epidemiologic research on disasters. Epidemiol Rev 17:113–119
- Norris FH, Friedman MJ, Watson PJ, Byrne CM, Diaz E, Kaniasty K (2002) 60, 000 disaster victims speak: part I: an empirical review of the empirical literature, 1981–2001. Psychiatry 65:207–239
- Rubonis AV, Bickman L (1991) Psychological impairment in the wake of disaster: the disaster-psychopathology relationship. Psychol Bull 109:384–399
- Gray MJ, Bolton EE, Litz BT (2004) A longitudinal analysis of PTSD symptom course: delayed-onset PTSD in Somalia Peacekeepers. J Consult Clin Psychol 72:909–913
- Adams RE, Boscarino JA (2006) Predictors of PTSD and delayed-PTSD after disaster: the impact of exposure and psychological resources. J Nerv Ment Dis 194:485–493
- Buckley TC, Blanchard DB, Hickling EJ (1996) A prospective examination of delayed onset PTSD secondary to motor vehicle accidents. J Abnorm Psychol 105:617–625
- 24. Prigerson HG, Maciejewski PK, Rosenheck RA (2001) Combat trauma: trauma with highest risk of delayed onset and unresolved posttraumatic stress disorder symptoms, unemployment, and abuse among men. J Nerv Ment Dis 189:99–108
- Freedy JR, Kilpatrick DG, Resnick HS (1993) Natural disasters and mental health: theory, assessment, and intervention. J Soc Behav Pers 8:49–103
- Boscarino JA (2006) PTSD and mortality among US Army veterans: a 30-year follow-up. Ann Epidemiol 16:248–258
- North CS, Nixon SJ, Shariat S, Mallonee S, McMillen JC, Spitznagel EL, Smith EM (1999) Psychiatric disorders among survivors of the Oklahoma City bombing. J Am Med Assoc 282:755–762
- Andrews B, Brewin CR, Philpott R, Stewart L (2007) Delayed posttraumatic stress disorder: a systematic review of the evidence. Am J Psychiatry 164:1319–1326
- Bryant RA, Harvey AG (2002) Delayed-onset posttraumatic stress disorder: a prospective evaluation. Aust NZ J Psychiatry 36:205–209
- North CS, Pfefferbaum B, Tivis L, Kawasaki A, Reddy C, Spitznagel EL (2004) The course of posttraumatic stress disorder in a follow-up study of survivors of the Oklahoma City bombing. Ann Clin Psychiatry 16:209–215
- American Psychiatric Association (1994) Diagnostic and statistical manual of mental disorders. 4th edn. American Psychiatric Association, Washington, DC
- Atkinson RM, Henderson RG, Sparr LF, Deale S (1982)
   Assessment of Vietnam veterans for posttraumatic stress disorder in Veterans Administration disability claims. Am J Psychiatry 139:1118–1121
- 33. Brewin CR (2003) Posttraumatic stress disorder: malady or myth. Yale University Press, New Haven
- 34. American Association for Public Opinion Research (2000) Standard definitions: final dispositions of case codes and outcomes rates for surveys. American Association for Public Opinion Research, Ann Arbor
- Adams RE, Boscarino JA, Galea S (2006) Social and psychological resources and health outcomes after World Trade Center disaster. Soc Sci Med 62:176–188
- Kessler RC, Little RJ, Groves RM (1995) Advances in strategies for minimizing and adjusting for survey nonresponse. Epidemiol Rev 17:192–204
- Kilpatrick DG, Ruggiero KJ, Acierno R, Saunders BE, Resnick HS, Best CL (2003) Violence and risk of PTSD, major

- depression, substance abuse/dependence, and comorbidity: results from the national survey of adolescents. J Consult Clin Psychol 71:692–700
- Resnick HS, Kilpatrick DG, Dansky BS, Saunders BE, Best CL (1993) Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. J Consult Clin Psychol 61:984–991
- Boscarino JA, Adams RE, Figley CR (2004) Mental health service use 1-year after the World Trade Center disaster: implications for mental health care. Gen Hosp Psychiatry 26:346–358
- Boscarino JA, Galea S, Adams RE, Ahern J, Resnick H, Vlahov D (2004) Mental health service and psychiatric medication use following the terrorist attacks in New York City. Psychiatr Serv 55:274–283
- 41. Sherbourne CD, Stewart AL (1991) The MOS social support survey. Soc Sci Med 32:705–714
- Rosenberg M (1979) Conceiving the self. Basic Books, New York
- Spitzer RL, Williams JB, Gibbon M (1987) Structured clinical interview for DSM-III-R—non-patient version. Biometrics Research Department, New York State Psychiatric Institute, New York
- 44. Acierno R, Kilpatrick DG, Resnick H, Saunders B, De Arellano M, Best C (2000) Assault, PTSD, Family substance use, and depression as risk factors for cigarette use in youth: findings from the National Survey of Adolescents. J Trauma Stress 13:381–396
- Robins LN, Cottler LB, Bucholz KK, Compton WM, North CS, Rourke KM (1999) Diagnostic interview schedule for DSM-IV. Washington University School of Medicine, Department of Psychiatry, St. Louis (Revised January 9, 2002)
- 46. Adams RE, Boscarino JA (2005) Stress and well-being in the aftermath of the World Trade Center attack: the continuing effects of a community-wide disaster. J Community Psychol 33:175–190
- Boscarino JA, Galea S, Ahern J, Resnick H, Vlahov D (2003)
   Psychiatric medication use among Manhattan residents following the World Trade Center disaster. J Trauma Stress 16:301–306
- Boscarino JA (1997) Diseases among men 20 years after exposure to severe stress: implications for clinical research and medical care. Psychosom Med 59:605–614
- Derogatis LR (2001) Brief symptom inventory 18 (BSI-18) manual. NCS Assessments, Minnetonka
- Long JS, Freese J (2006) Regression models for categorical dependent variables using Stata, 2nd edn. Stata Corporation, College Station
- 51. Stata Corporation (2007) Stata, version 9.2. Stata Corporation, College Station
- Bromet EJ, Sonnega A, Kessler RC (1998) Risk factors for DSM-III-R posttraumatic stress disorder: findings from the National Comorbidity Survey. Am J Epidemiol 147:353–361
- National Academy of Sciences (2007) PTSD compensation and military service. National Academy of Sciences Press, Washington. DC
- Grieger TA, Cozza SJ, Ursano RJ, Hoge C, Martinez PE, Engel CC, Wain HJ (2006) Posttraumatic stress disorder and depression in battle-injured soldiers. Am J Psychiatry 163:1777–1783
- Horowitz MJ, Solomon GF (1975) A prediction of delayed stress response syndromes in Vietnam veterans. J Soc Issues 31:67–80
- Shatan CF (1973) The grief of soldiers: Vietnam combat veterans' self-help movement. Am J Orthopsychiatry 43:640–653
- Solomon Z, Mikulincer M (2006) Trajectories of PTSD: a 20year longitudinal study. Am J Psychiatry 163:659–666
- Breslau N, Lucia VC, Davis GC (2004) Partial PTSD versus full PTSD: an empirical examination of associated impairment. Psychol Med 34:1205–1214



- 59. Galea S, Vlahov D, Resnick H, Ahern J, Susser E, Gold J, Bucuvalas M, Kilpatrick D (2003) Trends of probable post-traumatic stress disorder in New York City after the September 11 terrorist attacks. Am J Epidemiol 158:514–524
- Picou JS, Marshall BK, Gill DA (2004) Disaster, litigation, and the corrosive community. Soc Forces 82:1493–1522
- 61. Fehm L, Beesdo K, Jacobi F, Fiedler A (2008) Social anxiety disorder above and below the diagnostic threshold: prevalence, comorbidity and impairment in the general population. Soc Psychiatry Psychiatr Epidemiol 43:257–265
- Palinkas LA, Downs MA, Petterson JS, Russell J (1993) Social, cultural, and psychological impacts of the Exxon Valdez oil spill. Hum Organ 52:1–13
- Palinkas LA, Petterson JS, Russell J, Downs MA (1993) Community patterns of psychiatric disorder after the Exxon Valdez oil spill. Am J Psychiatry 150:1517–1523
- 64. Verschuur MJ, Spinhoven P, van Emmerik AA, Rosendaal FR (2008) Participation in a trauma-focused epidemiological investigation may result in sensitization for current health problems. Soc Psychiatry Psychiatr Epidemiol 43:132–139

