Delayed Posttraumatic Stress Disorder: Systematic Review, Meta-Analysis, and Meta–Regression Analysis of Prospective Studies

Geert E. Smid, MD; Trudy T. M. Mooren, PhD;
Roos C. van der Mast, MD, PhD;
Berthold P. R. Gersons, MD, PhD; and Rolf J. Kleber, PhD

Objective: Prevalence estimates of delayed posttraumatic stress disorder (PTSD) have varied widely in the literature. This study is the first to establish the prevalence of delayed PTSD in prospective studies and to evaluate associated factors through meta-analytic techniques.

Data sources: Studies were located by an electronic search using the databases EMBASE, MEDLINE, and PsycINFO. Search terms were *posttraumatic stress disorder* [include all subheadings] AND (*delayed* OR *prospective* OR *longitudinal* OR *follow-up*). Results were limited to journal articles published between 1980 and April 4, 2008.

Study selection: We included longitudinal, prospective studies of humans exposed to a potentially traumatic event that assessed participants at 1 to 6 months after the event, that included a follow-up of at least 12 months after the event, and that specified rates of new onset and remission between assessments in study completers.

Data extraction: Data were extracted concerning the study design, demographic features, and event-related characteristics and the number of PTSD cases at first assessment, the number of PTSD cases among study dropouts, and the number of new event-related PTSD cases at each subsequent assessment among study completers. Data from 24 studies were included. Four of these provided additional data on initial subthreshold PTSD and subsequent risk of delayed PTSD.

Data synthesis: The proportion of PTSD cases with delayed PTSD was 24.8% (95% CI = 22.6% to 27.2%) after adjusting for differences in study methodology, demographic features, and event-related characteristics. Military combat exposure, Western cultural background, and lower cumulative PTSD incidence were associated with delayed PTSD. Participants with initial subthreshold PTSD were at increased risk of developing delayed PTSD.

Conclusions: Delayed PTSD was found among about a quarter of PTSD cases and represents exacerbations of prior symptoms.

J Clin Psychiatry 2009;70(11):1572–1582

Submitted: *June 22, 2008; accepted March 13, 2009.*
Online ahead of print: *July 14, 2009 (doi:10.4088/JCP.08r04484).*
Corresponding author: *Geert E. Smid, MD, Centrum '45, Nienoord 5, 1112 XE Diemen, & e Netherlands (g.smid@centrum45.nl).*

Epidemiologic studies of posttraumatic stress disorder (PTSD) have confirmed the potency of traumatic stress to induce long-standing suffering in susceptible individuals.[1] The diagnosis of PTSD applies when a person has been exposed to a traumatic event to which he or she responded with fear, helplessness, or horror and has 3 distinct types of symptoms consisting of reexperiencing of the event, avoidance of reminders of the event as well as emotional numbing, and hyperarousal for at least 1 month. The propensity of the disorder to occur with delayed onset has been formally recognized since its inclusion in the *Diagnostic and Statistical Manual of Mental Disorders,* Third Edition *(DSM-III)* in 1980. According to the *DSM-IV-TR*, delayed PTSD must be diagnosed if the "onset of symptoms is at least 6 months after the stressor."[2(p468)]

Prevalence estimates of delayed PTSD have varied widely in the literature. In a recent systematic review, Andrews et al[3] pointed out ambiguities in the definition of delayed PTSD. The "onset of the symptoms" could refer to any initial symptom that might eventually lead to the disorder, or only to additional symptoms that lead to full-blown PTSD. In contrast to the former, the latter definition allows for prodromal symptoms prior to developing full-blown delayed PTSD. In their review,[3] which included several prospective as well as cross-sectional and retrospective studies, delayed PTSD in the absence of any prior symptoms appeared to be extremely rare. Delayed PTSD resulting from exacerbations of prior symptoms was found to occur in 38.2% and 15.3% of military and civilian cases of PTSD, respectively.[3]

So far, no meta-analysis has been carried out to establish the prevalence of delayed PTSD. In the present study, we systematically identified prospective studies comprising assessments within specified time frames relative to the potentially traumatic event in order to establish the prevalence of delayed PTSD from the pooled data. In addition, we analyzed whether aspects of study methodology, demographic features, and event-related characteristics, including

type of event and cumulative incidence of event-related PTSD, were associated with delayed PTSD prevalence. Third, we evaluated the likelihood of endorsing prodromal PTSD symptoms before obtaining a delayed PTSD diagnosis. Finally, we examined the risk of developing event-related PTSD at different points in time from event exposure.

METHOD

Data Sources

Studies were located by an electronic search. In line with results of an earlier study[4] that showed the importance of searching multiple databases to find the maximum number of relevant studies, we searched the databases EMBASE, MEDLINE, and PsycINFO. Search terms were *posttraumatic stress disorder* [include all subheadings] AND (*delayed* OR *prospective* OR *longitudinal* OR *follow-up*). Results were limited to journal articles published between 1980 and April 4, 2008. This time frame was chosen because of the establishment of diagnostic criteria for (delayed) PTSD in the *DSM-III* in 1980.[5]

Study Selection

We selected prospective studies of humans exposed to a potentially traumatic event that assessed the presence or absence of PTSD repeatedly at 2 or more assessment times within specified time frames relative to the potentially traumatic event. Because of the methodological limitations of retrospectively obtained information on the presence or absence of symptoms of PTSD, including the potential for memory bias resulting in errors of omission and addition,[6] we chose to rely on prospective identification of PTSD onset. Thus, we defined delayed PTSD as PTSD identified at 1 or more follow-up assessments beyond 6 months after the event in cases in which no PTSD was present at the first assessment(s). We decided that the first outcome assessment should be at least 1 month after the event, so that the diagnostic criteria of PTSD would apply (duration of more than 1 month). In addition, we decided that the first outcome assessment should be no later than 6 months after the event in order to reduce the potential number of missed cases who had a time-limited episode of PTSD that had remitted prior to the first assessment. Furthermore, we considered that the interval between the last PTSD-negative and first PTSD-positive assessment should be centered beyond 6 months after the event so that individuals who did not meet criteria at the first assessment but did at the second would have a very high likelihood of having delayed onset as defined by the DSM. Therefore, we decided that the duration of follow-up should be at least 12 months after the event. In studies with 2 or more assessments beyond 6 months after the event, we decided that the last follow-up assessment should be at least 12 months after the event, so as to limit the potential number of included studies providing arguably delayed PTSD cases identified between 6 and 12 months after event. The time since the event was from the time of the specific trauma exposure or the end of the potential exposure, such as the end of deployment to a war zone in military samples.

We excluded studies not specifying rates of new onset and remission between assessments in study completers, ie, when either of the following was not analyzed or reported: (1) the number of PTSD cases at index assessment(s) in study completers and (2) the number of new PTSD cases and the number of initial PTSD cases no longer fulfilling the criteria for PTSD (recovery of PTSD) at follow-up in study completers.

Eligible studies were characterized by the specification of rates of new onsets and remissions of PTSD between assessments. To review sample selection criteria and to obtain additional data concerning other study or sample characteristics regarding these eligible studies, if necessary, cross references were used, or authors were contacted via e-mail; of 15 authors contacted, 10 responded. In one study,[7] the sample mean age was imputed using matched mean imputation.

Data Extraction

We extracted the following data concerning the study design and sample characteristics: assessment times, number of study completers, number of study dropouts after the first assessment (between 1 and 6 months after the event), percentage female, age (mean [SD] range), diagnostic instrument, diagnostic assessor (interviewer), and criterion for diagnosis (*DSM* edition and cutoff score). For each study, we summarized population and sampling framework descriptors and exclusion criteria. These data are summarized in Appendix 1.

Subsequently, we extracted the following numerical outcome data in study completers: PTSD cases at first assessment, number of PTSD cases among study dropouts, and number of new PTSD cases at each subsequent assessment among study completers.

When studies included more than 1 assessment between 1 and 6 months after the event, all cases identified at those occasions were considered to be undelayed PTSD cases.

In order to obtain insight into the subclinical symptom course before delayed PTSD diagnosis, we extracted additional data. We identified studies reporting the number of participants meeting 2 of 3 symptom criteria (reexperiencing, avoidance and numbing, or hyperarousal), without meeting full PTSD criteria, otherwise called borderline,[7] partial,[8] or subsyndromal[9,10] PTSD cases (hereafter called subthreshold PTSD cases). From those studies, we extracted the number of subthreshold PTSD cases at first assessment, and the number of delayed PTSD cases at follow-up among these cases.

Data Synthesis

Following the approach used in a previous review,[3] our primary outcome was the number of delayed PTSD cases

divided by the total number of PTSD cases detected across all assessments. The statistical procedures used to conduct this meta-analysis applied inverse-variance weighted effect sizes[11] to account for differences in sample sizes, thus taking into consideration that studies with larger sample sizes will yield more accurate estimates of the population parameters than studies with smaller sample sizes. For analysis purposes, the outcome proportions were transformed to logits.[11] When the outcome proportions equaled 0% or 100%, 0.5 was added to both cells (containing frequencies of events and nonevents) before applying the logit transformation.

Before combining studies in the meta-analysis, we evaluated the presence and possible causes of heterogeneity in the main outcomes. Because evidence was found for the presence of heterogeneity in the study outcomes ($Q_{23} = 139.5$, $P < .001$), subsequent pooled analyses used random-effects methods.

As the prevalence of delayed PTSD was determined in study completers, an important question was whether study completers were representative of the whole initial sample with regard to PTSD prevalence. Therefore, we calculated odds ratios of PTSD prevalence in study completers versus in study dropouts at the first assessment from studies providing the relevant data and assessed the pooled effect.

Publication bias—ie, preferential publication of striking findings—was evaluated by Egger's test of the intercept.[12] The underlying notion is that small studies, which would generally have had larger standard errors, are more likely not to have been published unless reporting striking findings. The Egger test relies on a regression approach to evaluate the relationship between standardized effect (effect size divided by the standard error) and precision (defined as the inverse of the standard error). With publication bias, smaller studies will skew this relationship, causing the regression intercept to deviate significantly from the origin.

Sensitivity analyses included assessment of the influence of each study on the overall estimates of delayed PTSD prevalence by recalculating the pooled outcome proportions with 1 study removed and all others included.

Meta-Regression Analyses

Meta-regression analyses allow the application of regression techniques to identify the causes of heterogeneity and assess the amount of variance between the effect sizes explained by a number of variables of interest.[11,13] We used meta-regression to evaluate the influence of 3 groups of variables (ie, those related to the study methodology, demographic features, and event-related characteristics) on delayed PTSD prevalence. The contribution of every group of variables to the explained variance was evaluated by calculating the significance of the change in Cochran's heterogeneity statistic (Q) for the model. From the final regression model, we calculated the pooled outcome adjusted for differences in study methodology, demographic features, and event-related characteristics.

We summarized the variables for use in the meta-regression as follows. Study methodology: assessment (full interview = 1, questionnaire or screening = 0), diagnostic criterion (*DSM-IV* = 1, other = 0), number of assessments, time of the last PTSD-negative assessment within 6 months after the event, and duration of the follow-up from the event exposure in months. Demographic features: Western or non-Western cultural background as defined by Organization for Economic Cooperation and Development (OECD) membership of the main country of origin (Western = 1, non-Western = 0), sex (female = 1, male = 0), and age (sample mean). Event-related characteristics: military combat exposure (yes = 1, no = 0), civilian war exposure (yes = 1, no = 0), and cumulative PTSD incidence (cumulative rates of PTSD in study completers across all assessment points).

We also applied meta-regression while exploring risk of delayed PTSD over time to adjust for differences in assessment times in studies with more than 1 follow-up assessment beyond 6 months after the event.

Analyses were performed using SPSS software (SPSS Inc, Chicago, Illinois) with macros provided by Lipsey and Wilson.[11]

RESULTS

Study Selection

We identified 572 potentially relevant studies from the results of the electronic searches. Of these, 397 did not include assessments between 1 and 6 months after the event and/or a follow-up at least 12 months after the event. In addition, 142 did not specify new onsets and remissions between assessments in study completers. Of the remaining 33 eligible studies, 4 studies[14–17] appeared to report about cohorts that were already included and were therefore excluded due to overlap. Two studies reporting on the same cohort[18,19] were excluded because the diagnostic instrument used did not assess PTSD according to *DSM* criteria. Two other studies reporting on the same cohort[20,21] were excluded because cases with full and subsyndromal PTSD were not differentiated. Finally, 1 study[22] was excluded because the sample was only partially followed up.

The remaining 24 studies[7–10,23–42] provided data from an aggregate sample of 6,182 persons in 11 countries. The first assessment took place a mean of 4 months after the event (SD = 1, range: 1–6). The mean duration of follow-up was 25 months (SD = 11, range: 12–60). Summary characteristics of the included studies are presented in Table 1. A timeline of all individual studies showing assessment times in relation to the potentially traumatic event under study is represented in Figure 1. The characteristics of each separate study are listed in the Appendix 1.

Data Synthesis

Forest plots for random-effects meta-analysis are presented in Figure 2. Overall, 25.8% (95% CI = 19.7% to 33.0%)

Table 1. Characteristics of Included Studies (N = 24 Studies)

Variable	N (%) of Studies
Study methodology	
Assessment	
Full interview	15 (63)
Questionnaire or screening	9 (38)
Diagnostic criterion	
DSM-IV	6 (25)
DSM-III-R	12 (50)
Cutoff score	6 (25)
No. of assessments	
2	17 (71)
3 or 4	7 (29)
Last assessment ≤ 6 mo	
1–2 mo	6 (25)
3–4 mo	10 (42)
5–6 mo	8 (33)
Duration of follow-up	
12 mo	6 (25)
13–24 mo	10 (42)
> 24 mo	8 (33)
Demographic features	
Cultural background	
Western	20 (83)
Non-Western	4 (17)
Sex	
< 50% female	14 (58)
≥ 50% female	10 (42)
Age, mean	
< 18 y	3 (13)
> 18 y	21 (88)
Event-related characteristics	
Type of event	
Civilian war exposure	2 (8)
Military combat exposure	2 (8)
Other	20 (83)
Cumulative PTSD incidence	
< 15%	5 (21)
15%–35%	11 (46)
> 35%	8 (33)

Figure 1. Assessment Times of Included Studies

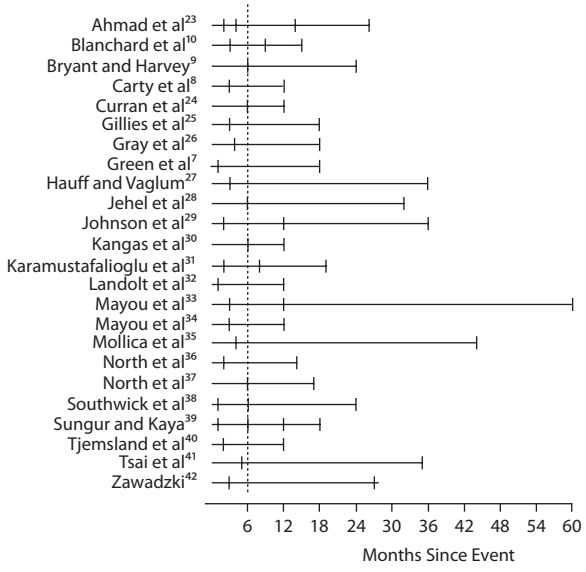
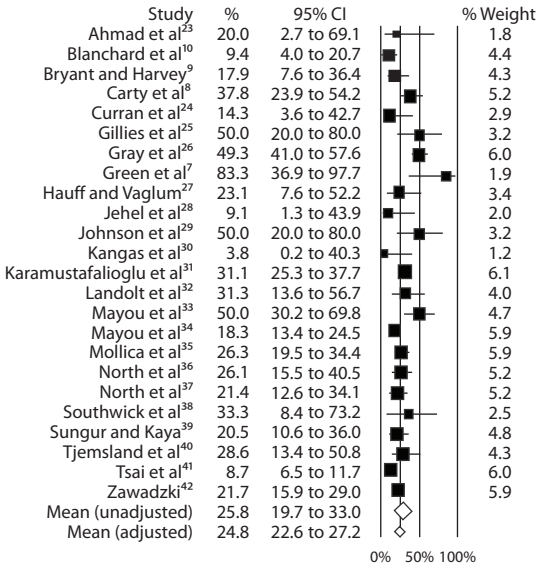


Figure 2. Proportion of PTSD Cases With Delayed PTSD



of study participants who fulfilled criteria for PTSD following exposure to the event under study were first identified at follow-up beyond 6 months after the event and were thus considered as having delayed PTSD.

We evaluated whether study completers were representative of the whole initial sample with regard to PTSD prevalence at the first assessment. Eighteen studies* provided the relevant data. The likelihood of participants with PTSD at first assessment to drop out was comparable with the likelihood in participants not so diagnosed (OR = 0.92, 95% CI = 0.74 to 1.15, $P = .474$). Therefore, we assumed dropout to be unrelated to PTSD diagnosis.

Evaluation of the possible publication bias suggested its absence regarding delayed PTSD prevalence (Egger test, $t = 0.25$, $P = .404$).

We completed sensitivity analyses by recalculating the pooled outcomes for the sample on multiple occasions with 1 of the studies removed at each iteration. The sensitivity analyses were especially important because some studies[26,41] included samples that were substantially larger than most of the other studies and thus may have exerted large effects on the overall effect estimate. These analyses yielded delayed PTSD prevalence estimates ranging from 24.4% (95% CI = 19.0% to 30.7%) to 27.5% (95% CI = 22.1% to 33.6%).

Meta-Regression Analyses

The characteristics of the study, demographic features, and event-related characteristics were entered subsequently

*References 7–10, 23–25, 27–30, 33–38, 42.

Table 2. Meta-Regression Analysis Relating Study Methodology, Demographic Characteristics, and Type of Event to Proportion of PTSD Cases With Delayed PTSD (N = 24 Studies)

Variable	B	SE	β
Step 1: Study methodology[a]			
Interview assessment	0.43	0.33	.27
DSM-IV	0.36	0.28	.25
No. of assessments	−0.22	0.24	−.15
Last assessment ≤ 6 months	0.15	0.09	.38
Duration of follow-up	0.03	0.01	.43*
Step 2: Demographic features[b]			
Western culture	1.55	0.52	.92**
Female sex	1.15	0.72	.35
Age	−0.02	0.01	−.33
Step 3: Event-related characteristics[c]			
Civilian war exposure	0.57	0.81	.24
Military combat exposure	1.40	0.54	.66**
Cumulative PTSD incidence	−2.18	0.70	−.42**

[a]For the model including the first step only, $R^2 = 0.07$, $Q = 1.8$, $df = 5$.
[b]For the second step: $R^2 = 0.30$, $Q = 12.4$, $df = 3$, $P < .01$. For the model including the two steps, $R^2 = 0.37$, $Q = 14.2$, $df = 8$.
[c]For the final step: $R^2 = 0.48$, $Q = 104.7$, $df = 3$, $P < .001$. For the final model including all three steps, $R^2 = 0.85$, $Q = 118.9$, $df = 11$, $P < .001$.
*$P < .05$.
**$P < .01$.
Abbreviations: B = regression coefficient, β = standardized regression coefficient (for the final step), Q = Cochran heterogeneity statistic for step/model, R^2 = explained variance.

into the regression model. Each subsequent step contributed significantly to the explained variance. In the final model, the following variables were included: assessment method (full interview vs screening), criteria (*DSM-IV* vs other), number of assessments, time of last PTSD-negative assessment within 6 months after the event, duration of follow-up, cultural background (Western vs non-Western), sex, age, type of event (civilian war exposure vs military combat exposure vs exposure to other potentially traumatic events), and cumulative PTSD incidence. The results are summarized in Table 2.

The results of the regression analyses showed that delayed PTSD prevalence was influenced by duration of follow-up, cultural background, type of event exposure, and cumulative PTSD incidence. Specifically, the meta-regression showed that the proportion of PTSD cases with delayed PTSD was larger (1) when the duration of follow-up was longer, (2) when the cultural background of the sample was predominantly Western as opposed to non-Western, (3) when the potentially traumatic exposure was to military combat as opposed to nonmilitary events, and (4) when cumulative PTSD incidence was lower.

When study methodology, demographic features, and event-related characteristics were adjusted for, 24.8% (95% CI = 22.6% to 27.2%) of PTSD cases were found to endorse delayed PTSD. The final model explained 85% of the variance between studies, and the residual heterogeneity was not significant (residual Q = 20.6, $df = 12$, $P = .057$). The adjusted mean delayed PTSD prevalence is also represented in Figure 2.

Risk of Delayed PTSD in Participants Meeting Subthreshold PTSD Criteria Initially

In order to test the hypothesis that delayed PTSD is most likely to occur in persons already reporting elevated symptoms, we compared the risk of delayed PTSD between participants meeting 2 of 3 PTSD symptom criteria initially (subthreshold PTSD) and those meeting less than 2 criteria. Relevant data were reported by 4 studies, all concerning samples of accidental injury victims.[7–10] Of participants with initial subthreshold PTSD, 26.2% (95% CI = 8.0% to 59.3%) went on to develop delayed PTSD. By contrast, of those meeting less than 2 criteria, only 4.1% (95% CI = 2.5% to 6.7%) developed delayed PTSD. Thus, in line with expectations, participants with initial subthreshold PTSD were at increased risk of developing delayed PTSD (OR = 10.7, 95% CI = 2.0 to 58.0, $P = .006$).

Risk of Delayed PTSD Onset Over Time

Six studies[10,23,29,31,33,39] with 2 follow-up assessments beyond 6 months after the event provided data to establish the risk of delayed PTSD onset at 2 subsequent points in time from event exposure. The number of new event-related PTSD cases was divided by the total number of study completers that had not (yet) been diagnosed with PTSD at the previous points. To clarify the relation of these percentages to the overall outcome, it should be kept in mind that these proportions have different numerators and denominators; the overall outcome represents the number of newly identified PTSD cases at 1 or more assessments beyond 6 months after the event divided by the total number of PTSD cases detected across all assessments. We adjusted for between-study differences in assessment times using meta-regression. At a mean of 9 months after the event (SD = 2, range: 8–14), the risk of delayed onset PTSD in study participants not so far diagnosed with PTSD was 6.0% (95% CI = 3.1% to 11.2%). At 25 months after the event (SD = 14, range: 15–60), the risk of delayed-onset PTSD in the remainder of study participants, who up to that point had not met PTSD criteria across at least 2 previous assessments, was 5.8% (95% CI = 3.3% to 10.0%). As none of the included studies comprised more than 2 follow-up assessments beyond 6 months after the event, we were unable to carry out additional analyses concerning the distribution of risk of delayed PTSD onset over time.

DISCUSSION

Prevalence of Delayed PTSD

The purpose of this study was to determine the prevalence of delayed PTSD across prospective studies of trauma-exposed populations. The results of this meta-analysis show that approximately a quarter of persons exposed to a potentially traumatic event who develop symptoms qualifying for a diagnosis of PTSD at 1 or more assessments may be considered as having delayed PTSD. We evaluated the possible

confounding influence of study dropout, single (large) studies, and publication bias in separate analyses demonstrating that these potential confounders were unlikely to play a substantial role. A secondary purpose was to evaluate factors associated with delayed onset of PTSD. Of the study characteristics, duration of follow-up was associated with higher prevalence of delayed PTSD. Samples whose cultural background was predominantly Western evinced more delayed PTSD than non-Western samples. Military combat exposure conferred a higher risk of delayed PTSD compared with exposure to other types of potentially traumatic events. In samples with low cumulative PTSD incidence, more delayed PTSD was found.

On subsets of samples providing relevant data, we performed follow-up analyses. In 4 studies of accident victims, delayed PTSD appeared to occur most often in individuals already reporting elevated symptoms. These results are consistent with a conceptualization of delayed PTSD as representing exacerbations of prior symptoms. Finally, from 6 studies with 2 follow-up assessments beyond 6 months after the event, we explored the risk of developing delayed PTSD over time. Six percent of participants without initial PTSD had worsened to meet criteria for PTSD at around 9 months, and of the remainder, an additional 6% had declined to the same degree between 9 and a mean of 25 months.

Study Strengths and Limitations

Our study is unique as it has determined delayed PTSD prevalence following a range of potentially traumatic events using data from prospective, longitudinal studies only. The study strengths are the systematic literature selection, the meta-analysis, and the exploration of heterogeneity in outcomes by meta-regression analyses.

The limitations of this study should be acknowledged. By choosing to rely on prospective identification of PTSD onset, some uncertainty about the exact time of onset remains, unless assessments would have been repeated very frequently. Most authors use time of PTSD identification in prospective studies as a substitute for time of PTSD onset. However, there is the possibility that some individuals who did not meet criteria at the first measurement point but did at the second might not have had a delayed onset beyond 6 months, as defined by the *DSM*, especially if the first point was early in the interval 1 to 6 months after the event. In our collection of studies, the average time of first assessment was 4 months after the event, and the average duration of follow-up was 25 months, so the average time of onset was centered well beyond 6 months after the event. In order to examine the influence of the time of the PTSD negative assessment within 6 months after the event on delayed PTSD prevalence, we included this variable in our meta-regression. This variable did not show a significant relation with delayed PTSD prevalence. By contrast, duration of follow-up was significantly related to delayed PTSD prevalence, suggesting that more delayed PTSD cases might have been detected if the mean duration of follow-up had been longer. Furthermore, we analyzed 6 studies[10,23,29,31,33,39] with more than 1 follow-up assessment beyond 6 months after the event. We found that risk of delayed onset did not decrease between 9 and 25 months after the event. Extrapolating from these results, there is no obvious reason to assume that risk of delayed onset would increase steeply proximal to the 9-month mark. This extrapolation lends further support to the conclusion that this potential limitation is unlikely to have biased our results.

A related issue is the consideration that delayed PTSD might merely reflect symptom fluctuation rather than representing true progression of symptoms. We dealt with this issue in 2 ways. If fluctuation were the case, one would have expected a decrease in the likelihood of identification of apparently delayed PTSD at later assessments in studies with more than 2 assessments. Therefore, we included the number of assessment times in our meta-regression. We did not find a significant relation with delayed PTSD prevalence. Secondly, we reasoned that absence of symptoms qualifying for a PTSD diagnosis at several assessments before delayed PTSD onset would be suggestive of the existence of a crescendo pattern of PTSD symptoms in delayed PTSD. Therefore, we took a closer look at studies comprising more than 2 assessments. In all those studies,[10,23,29,31,33,39] fresh PTSD cases were detected at the third or even fourth assessment. Moreover, in these studies, the average number of delayed cases detected at the first or second follow-up assessment beyond 6 months after the event was about the same.

Some reported symptoms of PTSD may have been related to events other than the event under study, for example, to intervening new traumatic events. On the one hand, this seems unlikely because the authors of the majority of studies* explicitly stated that questions about symptoms were specifically directed to symptoms related to the event under study. In addition, 2 studies reported that delayed PTSD cases following road traffic accidents were not related to new accidents.[33,34] On the other hand, it may be difficult for study participants to disentangle old and new symptoms. However, as elaborated more in detail below, intervening events may act as precipitants of delayed PTSD onset, rather than merely being confounders.

Finally, by choosing to include studies that allowed us to extract crude numbers of cases, we excluded studies that failed to provide information on new onsets and remissions between assessments. The exclusion of these studies may have biased our results.

Explaining Delayed PTSD

Explaining delayed PTSD may be facilitated by categorizing associated factors in relation to the time of the traumatic

*References 7, 8, 23, 26–32, 36, 37, 39–42.

event exposure into preexistent, event-related, and postevent factors.

Preexistent factors: Western cultural background. Unexpectedly, a Western cultural background appeared to be strongly related to delayed PTSD prevalence. The Taiwanese earthquake survivor study[41] exerted the strongest effects in this respect, with only 8.7% of PTSD cases being delayed. Our finding supports the hypothesis by Hough et al[43] that ethnocultural factors may potentially affect all stages in the development of PTSD, influencing its time of onset.[43] Speculatively, non-Western traumatic event survivors may be less likely to harbor expectations regarding reparative efforts by the authorities compared with Western survivors, which might render them less prone to deception and frustration at later stages following traumatic event exposure. Related to this is the possibility that disasters and war may be more unanticipated in the Western world.

Event-related factors.

Military combat exposure. Military combat exposure was found to confer a high risk of delayed PTSD. This finding is in line with the results of 2 large-scale cross-sectional studies,[44,45] and the results of the systematic review[3] cited previously. Thus, considerable evidence exists that delayed onset PTSD may follow military combat exposure more often than exposure to other types of potentially traumatic events.

Our finding that military combat exposure is associated with delayed PTSD appears to be consistent with the view that postponement of symptom onset in delayed PTSD may be adaptive. This view has actually shaped the concept of delayed PTSD from its origin. Delayed PTSD was included in the initial definition of PTSD in the *DSM-III* in 1980 to accommodate the syndrome encountered in Vietnam veterans.[46] The onset of symptoms was assumed to be postponed because a stress reaction in the midst of combat is not adaptive.[46] Consistent with this view is the finding that severe injury as a consequence of traumatic event exposure appears to be associated with delayed PTSD in both civilian accidental injury victims[8,47] and soldiers.[48]

In addition to combat exposure per se, belonging to a military group may be relevant with regard to explaining delayed PTSD. Initial minimization of symptoms by military personnel may reflect reluctance to endorse genuine distress from fear of stigma in the military context.[26] Alternatively, group membership may initially promote a feeling of safety and thereby foster resilience toward symptoms of PTSD. This effect may subsequently decrease as group support diminishes over time.

Event-related morbidity. Using cumulative PTSD incidence as a measure of event-related morbidity, we found that lower cumulative PTSD incidence was associated with higher rates of delayed PTSD. This remarkable finding suggests that factors postponing PTSD symptom onset may also promote resilience. This possibility is consistent with the view outlined previously, highlighting the adaptive value of delayed symptom onset.

Post-event factors. Minor waxing and waning of symptoms appears to be an untenable explanation for delayed PTSD, as indicated by a study[26] demonstrating large increases in reported symptoms in delayed PTSD. In addition, decreases in measures of social, physical, and emotional functioning were reported to co-occur with delayed PTSD diagnosis in another study.[41]

Prodromal symptoms. We found that, in a subsample of studies representative of accidental injury victims, initial subthreshold PTSD cases (characterized by meeting 2 of 3 PTSD symptom criteria) were more likely to go on to develop delayed PTSD than participants meeting less than 2 PTSD criteria initially. This finding is consistent with previous reports[3,8–10,49,50] emphasizing the likelihood of delayed PTSD cases to endorse prodromal symptoms. The conclusion that delayed PTSD most often represents progressive addition of more symptoms over time appears justified. Possibly, prodromal symptoms such as intrusive memories, increased startle reactions, sleep disturbance, or impaired concentration act to increase allostatic load[51] and the risk of PTSD.

Secondary gain. Increased symptom reporting over time may be motivated by secondary gains when compensation claims, disability pensions, or other forms of reward are at stake. In military populations it has been suggested that secondary financial gain may play a key role. The US military benefits system, for example, operates in terms of the maintenance of PTSD years after combat. One could argue that this practice might impact on the time of onset. In the same vein, our finding that a Western cultural background is associated with increased delayed PTSD prevalence might, although speculatively, be related to the fact that financial rewards may be more likely to be available in Western countries. In the scientific literature, the emphasis on secondary gain has diminished with increasing attention to the effects of traumatic events.[52] We found, in line with previous findings,[3] that the delayed progression of symptoms cannot be considered rare but is a consistent finding across studies in many different contexts, which suggests that exaggerated reporting due to a desire for compensation is unlikely to be a major factor. Consistent with this view is our finding of a relatively low delayed PTSD prevalence (14%) in one litigant sample.[24]

Intervening events. Intervening stressful life events have been shown to increase the risk of delayed PTSD.[41] Clinically, several individuals with delayed PTSD appear to have the onset of their symptoms precipitated by a relatively minor new life event that may have reminded the sufferer of something about the original trauma. Intervening stressful life events thus appear to be capable of precipitating delayed PTSD onset.

Clinical and Research Implications

Our study highlights the importance of long-term follow-up of groups at increased risk of developing delayed

PTSD following traumatic exposure, particularly military combatants and individuals reporting elevated PTSD symptoms at earlier stages. Our results emphasize the need for long-term availability of specialized mental health care facilities as well as the repeated screening of those reporting elevated symptoms.

Knowledge of delayed PTSD may contribute to the clinician's diagnostic accuracy, as the remoteness of the traumatic event in delayed PTSD poses a challenge to the clinician's diagnostic capabilities. Educating the patient and his or her relatives may be important, given the fact that symptom progression may be upsetting and may seem paradoxical. Subthreshold symptoms of PTSD merit clinical attention in individuals seeking professional help because they appear to confer an increased risk of delayed PTSD.

Delayed PTSD highlights the importance of reporting new onsets and remissions between assessments in PTSD research. When only PTSD point prevalence series are analyzed from repeated measurements, the general tendency of symptom decrease masks symptom increase in a subset of exposed victims. The total number of persons suffering clinically relevant consequences at some point following a traumatic event will then be underestimated. In addition, future researchers investigating PTSD course should attempt to include data on help-seeking behavior and/or mental health service utilization, as these might have interesting relations with delayed PTSD onset.

Our results support the suggestion by Andrews et al,[3] that future *DSM* editions require a definition of delayed PTSD that explicitly allows for prodromal symptoms. In other words, although the onset of full syndromal PTSD is delayed in some individuals, we expect subsyndromal PTSD symptoms in those individuals prior to their meeting full diagnostic criteria. "Delayed onset of symptoms" should perhaps be reconsidered as "delayed onset of the disorder" in *DSM-V*. Such a definition appears to be theoretically more plausible, to reflect empirical findings more accurately, and to be clinically more useful. In addition, such a definition facilitates scientific study of the phenomenon. Future studies are needed that consider possible explanations of delayed PTSD in more detail in a prospective manner.

Our evidence clearly suggests that delayed PTSD may occur in a subset of individuals following a potentially traumatic event, particularly military combatants and individuals reporting elevated PTSD symptoms at earlier stages. Facilities aiming at preventing and treating long-term event-related psychopathology should target their efforts toward these groups.

Author affiliations: Foundation Centrum '45, Diemen (Drs Smid and Gersons); Foundation Centrum '45, Oegstgeest (Dr Mooren); Leiden University, Leiden (Dr van der Mast); Academic Medical Centre, Amsterdam (Dr Gersons); and Utrecht University, Utrecht, and Institute for Psychotrauma, Diemen (Dr Kleber), The Netherlands.
Financial disclosure: None reported.
Funding/support: None reported.

Acknowledgment: We are grateful to Joop Hox, PhD; Marianne Hubregtse, MA; and Gerty Lensvelt, PhD, all from Utrecht University, for their advice on meta-analytical techniques, and to Iris Engelhard, PhD, Utrecht University, and Peter van der Velden, PhD, Institute for Psychotrauma, Diemen, for their critical comments on earlier versions of the manuscript. None of the acknowledged individuals have financial or other relationships relevant to the subject of this article.

REFERENCES

1. Kessler RC, Sonnega A, Bromet E, et al. Posttraumatic stress disorder in the National Comorbidity Survey. *Arch Gen Psychiatry.* 1995;52(12):1048–1060.
2. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders,* Fourth Edition, Text Revision. American Psychiatric Association; 2000.
3. Andrews B, Brewin CR, Philpott R, et al. Delayed-onset posttraumatic stress disorder: a systematic review of the evidence. *Am J Psychiatry.* 2007;164(9):1319–1326.
4. McDonald S, Taylor L, Adams C. Searching the right database: a comparison of four databases for psychiatry journals. *Health Libr Rev.* 1999;16(3):151–156.
5. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders,* Third Edition. American Psychiatric Association; 1980.
6. Harvey AG, Bryant RA. Memory for acute stress disorder symptoms: a two-year prospective study. *J Nerv Ment Dis.* 2000;188(9):602–607.
7. Green MM, McFarlane AC, Hunter CE, et al. Undiagnosed posttraumatic stress disorder following motor vehicle accidents. *Med J Aust.* 1993;159(8):529–534.
8. Carty J, O'Donnell ML, Creamer M. Delayed-onset PTSD: a prospective study of injury survivors. *J Affect Disord.* 2006;90(2-3):257–261.
9. Bryant RA, Harvey AG. Delayed-onset posttraumatic stress disorder: a prospective evaluation. *Aust N Z J Psychiatry.* 2002;36(2):205–209.
10. Blanchard EB, Hickling EJ, Barton BA, et al. One-year prospective follow-up of motor vehicle accident victims. *Behav Res Ther.* 1996;34(10):775–786.
11. Lipsey MW, Wilson DB. *Practical Meta-Analysis*. Thousand Oaks, CA: Sage Publications; 2001. Bickman L, Rog DJ, eds. *Applied Social Research Methods Series;* vol. 49.
12. Egger M, Smith GD, Schneider M, et al. Bias in meta-analysis detected by a simple, graphical test. *BMJ.* 1997;315(7109):629–634.
13. Hedges LV. Fixed effect models. In: Cooper HM, Hedges LV, eds. *The Handbook of Research Synthesis*. New York, NY: Russell Sage Foundation; 1994:285–300.
14. Bryant RA, Harvey AG, Guthrie RM, et al. Acute psychophysiological arousal and posttraumatic stress disorder: a two-year prospective study. *J Trauma Stress.* 2003;16(5):439–443.
15. Harvey AG, Bryant RA. The relationship between acute stress disorder and posttraumatic stress disorder: a 2-year prospective evaluation. *J Consult Clin Psychol.* 1999;67(6):985–988.
16. Mayou R, Bryant B. Outcome 3 years after a road traffic accident. *Psychol Med.* 2002;32(4):671–675.
17. Mayou R, Bryant B, Duthie R. Psychiatric consequences of road traffic accidents. *BMJ.* 1993;307(6905):647–651.
18. McFarlane AC. Long-term psychiatric morbidity after a natural disaster: implications for disaster planners and emergency services. *Med J Aust.* 1986;145(11-12):561–563.
19. McFarlane AC. The longitudinal course of posttraumatic morbidity: the range of outcomes and their predictors. *J Nerv Ment Dis.* 1988;176(1):30–39.
20. Vaiva G, Boss V, Ducrocq F, et al. Relationship between posttrauma GABA plasma levels and PTSD at 1-year follow-up. *Am J Psychiatry.* 2006;163(8):1446–1448.
21. Vaiva G. Taux plasmatiques de GABA: au decours d'un psychotrauma et survenue de troubles psychotraumatiques. *Stress Et Trauma.* 2005;5:131–139.
22. Freedman SA, Brandes D, Peri T, et al. Predictors of chronic posttraumatic stress disorder: a prospective study. *Br J Psychiatry.* 1999;174:353–359.
23. Ahmad A, Mohamed HT, Ameen NM. A 26-month follow-up of posttraumatic stress symptoms in children after the mass-escape tragedy in

Iraqi Kurdistan. *Nord J Psychiatry.* 1998;52(5):357–366.
24. Curran PS, Bell P, Murray A, et al. Psychological consequences of the Enniskillen bombing. *Br J Psychiatry.* 1990;156:479–482.
25. Gillies ML, Barton J, Di Gallo A. Follow-up of young road accident victims. *J Trauma Stress.* 2003;16(5):523–526.
26. Gray MJ, Bolton EE, Litz BT. A longitudinal analysis of PTSD symptom course: delayed-onset PTSD in Somalia peacekeepers. *J Consult Clin Psychol.* 2004;72(5):909–913.
27. Hauff E, Vaglum P. Chronic posttraumatic stress disorder in Vietnamese refugees: a prospective community study of prevalence, course, psychopathology, and stressors. *J Nerv Ment Dis.* 1994;182(2):85–90.
28. Jehel L, Paterniti S, Brunet A, et al. Prediction of the occurrence and intensity of posttraumatic stress disorder in victims 32 months after bomb attack. *Eur Psychiatry.* 2003;18(4):172–176.
29. Johnson SD, North CS, Smith EM. Psychiatric disorders among victims of a courthouse shooting spree: a three-year follow-up study. *Community Ment Health J.* 2002;38(3):181–194.
30. Kangas M, Henry JL, Bryant RA. The course of psychological disorders in the 1st year after cancer diagnosis. *J Consult Clin Psychol.* 2005;73(4):763–768.
31. Karamustafalioglu OK, Zohar J, Güveli M, et al. Natural course of posttraumatic stress disorder: a 20-month prospective study of Turkish earthquake survivors. *J Clin Psychiatry.* 2006;67(6):882–889.
32. Landolt MA, Vollrath M, Timm K, et al. Predicting posttraumatic stress symptoms in children after road traffic accidents. *J Am Acad Child Adolesc Psychiatry.* 2005;44(12):1276–1283.
33. Mayou R, Tyndel S, Bryant B. Long-term outcome of motor vehicle accident injury. *Psychosom Med.* 1997;59(6):578–584.
34. Mayou R, Bryant B, Ehlers A. Prediction of psychological outcomes one year after a motor vehicle accident. *Am J Psychiatry.* 2001;158(8):1231–1238.
35. Mollica RF, Sarajlic N, Chernoff M, et al. Longitudinal study of psychiatric symptoms, disability, mortality, and emigration among Bosnian refugees. *JAMA.* 2001;286(5):546–554.
36. North CS, Smith EM, Spitznagel EL. One-year follow-up of survivors of a mass shooting. *Am J Psychiatry.* 1997;154(12):1696–1702.
37. North CS, Pfefferbaum B, Tivis L, et al. The course of posttraumatic stress disorder in a follow-up study of survivors of the Oklahoma City bombing. *Ann Clin Psychiatry.* 2004;16(4):209–215.
38. Southwick SM, Morgan CA, Darnell A, et al. Trauma-related symptoms in veterans of Operation Desert Storm: a 2-year follow-up. *Am J Psychiatry.* 1995;152(8):1150–1155.
39. Sungur M, Kaya B. The onset and longitudinal course of a man-made posttraumatic morbidity: survivors of the Sivas disaster. *Int J Psychiatry Clin Pract.* 2001;5(3):195–202.
40. Tjemsland L, Soreide JA, Malt UF. Posttraumatic distress symptoms in operable breast cancer, pt 3: status one year after surgery. *Breast Cancer Res Treat.* 1998;47(2):141–151.
41. Tsai KY, Chou P, Chou FH-C, et al. Three-year follow-up study of the relationship between posttraumatic stress symptoms and quality of life among earthquake survivors in Yu-Chi, Taiwan. *J Psychiatr Res.* 2007;41(1-2):90–96.
42. Zawadzki B. Uwarunkowania zaburzenia stresowego pourazowego (PTSD) w grupie ofiar powodzi: rola charakterystyki zdarzenia i jego dlugotrwalych materialnych nastepstw oraz reaktywnosci emocjonalnej. *Przegląd Psychologiczny.* 2006;49:227–242.
43. Hough RL, Canino GJ, Abueg FR, et al. PTSD and related stress disorders among Hispanics. In: Marsella AJ, Friedman MJ, Gerrity ET, et al, eds. *Ethnocultural Aspects of Posttraumatic Stress Disorder: Issues, Research, and Clinical Applications*. Washington, DC: American Psychological Association; 1996:301–338.
44. Helzer JE, Robins LN, McEvoy L. Posttraumatic stress disorder in the general population: findings of the epidemiologic catchment area survey. *N Engl J Med.* 1987;317(26):1630–1634.
45. Prigerson HG, Maciejewski PK, Rosenheck RA. Combat trauma: trauma with highest risk of delayed onset and unresolved posttraumatic stress disorder symptoms, unemployment, and abuse among men. *J Nerv Ment Dis.* 2001;189(2):99–108.
46. Andreasen NC. Acute and delayed posttraumatic stress disorders: a history and some issues. *Am J Psychiatry.* 2004;161(8):1321–1323.
47. Ehlers A, Mayou RA, Bryant B. Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. *J Abnorm Psychol.* 1998;107(3):508–519.
48. Grieger TA, Cozza SJ, Ursano RJ, et al. Posttraumatic stress disorder and depression in battle-injured soldiers. *Am J Psychiatry.* 2006; 163(10):1777–1783.
49. Blanchard EB, Hickling EJ. Delayed-onset PTSD. In: Blanchard EB, Hickling EJ, eds. *After the Crash: Psychological Assessment and Treatment of Survivors of Motor Vehicle Accidents*. 2nd ed. Washington, DC: American Psychological Association; 2004:161–172.
50. Buckley TC, Blanchard EB, Hickling EJ. A prospective examination of delayed onset PTSD secondary to motor vehicle accidents. *J Abnorm Psychol.* 1996;105(4):617–625.
51. McEwen BS. Protective and damaging effects of stress mediators. *N Engl J Med.* 1998;338(3):171–179.
52. Jones E, Wessely S. A paradigm shift in the conceptualization of psychological trauma in the 20th century. *J Anxiety Disord.* 2007; 21(2):164–175.

Editor's Note: We encourage authors to submit papers for consideration as a part of our Early Career Psychiatrists section. Please contact Marlene Freeman, MD, at mfreeman@psychiatrist.com.

Appendix 1 on pages 1581–1582

Appendix 1. Details of Included Studies

Study (Country)	N	Assessment Times (months since event)	Population	Sampling Framework	Exclusion Criteria	Diagnosis (assessor), Criterion	Female Sex, %	Age, Mean (SD) [range], y
Ahmad et al, 1998[23] (Iraq)	15	2, 4, 14, 26	Kurdish children (6–16 y) exposed to Mass Escape Tragedy in Iraq	Families living in temporary camp on Iraqi-Turkish border; children were from same province	(Family) history of mental disorder	PTSS-C (clinician), *DSM-III-R*	33	11 (3) [6–16]
Blanchard et al, 1996[10] (United States)	132	3, 9, 15	Road traffic accident victims (> 17 y) seeking medical care within 48 h	Referrals from health care providers and participants responding to local media advertising	NR	CAPS (experienced assessors), *DSM-III-R*	67	35 (13) [17–71]
Bryant and Harvey 2002[9] (Australia)	103	6, 24	Adults (16–65 y) admitted > 24 h to hospital following a road traffic accident	Admissions at 1 hospital over a 10-mo period	Non–English speaking, prescribed narcotic analgesics (excluding codeine), posttraumatic amnesia > 24 h	CIDI-PTSD (clinicians), *DSM-III-R*	43	31 (12) [17–63]
Carty et al, 2006[8] (Australia)	301	3, 12	Adults (18–70 y) admitted to hospital > 24 h following accidental injury (75% road traffic accident)	Admissions at 1 hospital over an 18-mo period	Non–English speaking, moderate or severe traumatic brain injury, psychosis, self-harm, intravenous drug abuse	CAPS (clinician), *DSM-IV*	25	37 (9) [18–70]
Curran et al, 1990[24] (United Kingdom)	25	6, 12	Adult litigants following bombing, 1987	Survivors referred to medico-legal department	Children < 14 y	Interview (clinician), *DSM-III-R*	48	30 (14) [NR]
Gillies et al, 2003[25] (United Kingdom)	27	3, 18	Children and adolescents (5–18 y) attending accident and emergency departments following a road traffic accident	Admissions to 5 Glasgow hospitals over a 5-mo period	Accidents without second-party involvement	PTSD-RI (clinician), ≥ 10, moderate	39	12 (6) [5–18]
Gray et al, 2004[26] (United States)	1040	4, 18	Active duty military personnel following deployment to Somalia, 1992–1993	Units from several military installations that supplied troops for the mission in Somalia	None	PCL or MISS (self-report), ≥ 68 or ≥ 92	6	26 (6) [NR]
Green et al, 1993[7] (Australia)	18	1, 18	Adults admitted at orthopedic ward following a road traffic accident	Admissions to 1 hospital trauma team over a 3-mo period	Non–English speaking, brain damage	DIS (NR), *DSM-III-R*	79	NR
Hauff and Vaglum 1994[27] (Norway)	131	3, 36	Vietnamese refugees (> 15 y) arriving in Southeast Norway in 1982	All Vietnamese boat refugees rescued at random by Norwegian merchant vessels in the South China Sea	None	Interview, PSE (clinician), *DSM-III-R*	21	26 (9) [15–58]
Jehel et al, 2003[28] (France)	26	6, 32	Victims exposed to bombing attack, 1996	All victims referred to victim aid organization	Non–French speaking	QPTS (self-report), *DSM-III-R*	47	42 (15) [NR]
Johnson et al, 2002[29] (United States)	77	2, 12, 36	Individuals exposed to courthouse shooting incident, 1992	Employees of courthouse and offices of individuals directly involved	None	DIS-DS (experienced assessors), *DSM-III*	65	39 (11) [NR]
Kangas et al, 2005[30] (Australia)	49	6, 12	Hospital admissions diagnosed with first-onset head and neck cancer or lung cancer	Admissions to medical oncology unit in major metropolitan hospital	Non–English speaking, brain metastases at diagnosis, history of brain impairment or psychotic illness, significant noncancer medical problems	SCID (clinicians), *DSM-IV*	24	59 (13) [24–84]

(continued)

Appendix 1 (continued). Details of Included Studies

Study (Country)	N	Assessment Times (months since event)	Population	Sampling Framework	Exclusion Criteria	Diagnosis (assessor), Criterion	Female Sex, %	Age, Mean (SD) [range], y
Karamustafalioglu et al, 2006[31] (Turkey)	464	2, 8, 19	Adults (> 16 y) exposed to earthquake (7.4 on the Richter scale), 1999	All inhabitants of Avcilar quarter, Istanbul	None	PTSD self-test (self-report). *DSM-IV*	71	34 (13) [NR]
Landolt et al, 2005[32] (Switzerland)	68	1, 12	Children and adolescents (6.5–14.5 y) admitted > 24 h at children's hospitals following a road traffic accident	Admissions to 4 hospitals over a 24-mo period	Non–German speaking, severe head trauma, mental retardation	PTSD-RI (trained graduate students), ≥ 25, moderate	46	10 (3) [7–15]
Mayou et al, 1997[33] (United Kingdom)	111	3, 12, 60	Adults (18–70 y) attending accident department following a road traffic accident	Attendants of 1 hospital living in Oxfordshire with either multiple injuries or whiplash and no other injuries	Head injuries leading to unconsciousness > 15 min	Interview, PSE (assessor NR), PSS (self-report, at 60 mo), *DSM-III-R*	33	30 (12) [17–69]
Mayou et al, 2001[34] (United Kingdom)	689	3, 12	Adults (17–69 y) attending accident department following a road traffic accident	Admissions to 1 hospital accident department over a 12-mo period	Head injuries leading to unconsciousness > 15 min, too ill, overseas visitors, learning or psychiatric problems	PSS (self-report), *DSM-IV*	48	32 (13) [17–69]
Mollica et al, 2001[35] (Croatia)	375	4, 44	Adult Bosnian refugees living in a refugee camp in Croatia, 1996–1999	Registries at private social service agency	None	HTQ (clinicians and trained interviewers), *DSM-IV*	65	50 (16) [NR]
North et al, 1997[36] (United States)	124	2, 14	Adult survivors of mass shooting incident, 1991	Data from police and responders to media advertisements	None	DIS-DS (clinicians and trained interviewers), *DSM-III-R*	53	39 (14) [NR]
North et al, 2004[37] (United States)	137	6, 17	Adult (> 18 y) survivors of Oklahoma City bombing, 1995	Department of Health registry of survivors	Severe injury	DIS-DS (trained interviewers), *DSM-III-R*	51	44 (12) [19–89]
Southwick et al, 1995[38] (United States)	62	1, 6, 24	Military personnel following deployment to Iraq, 1991	All members of 2 reserve units attending first monthly training session following return	None	MISS (self-report), ≥ 89	21	30 (10) [NR]
Sungur and Kaya 2001[39] (Turkey)	79	1, 6, 12, 18	Survivors of fire-setting by religious fundamentalists at Sivas, 1993	Sample selected from 3 groups differing in exposure severity	NR	PTSD interview (NR), *DSM-III-R*	22	30.1 (NR) [NR]
Tjemsland et al, 1998[40] (Norway)	106	2, 12	Female adults (< 71 y) diagnosed with operable breast cancer 1 or 2 weeks before admission	Consecutive admissions to 1 hospital over a 2-y period	Non–Norwegian speaking	IES, GHQ-28 (self-report), ≥ 20 and ≥ 2 (6 items)	100	50 (NR) [33–70]
Tsai et al, 2007[41] (Taiwan)	1756	5, 35	Earthquake (7.3 on the Richter scale) survivors (> 15 y) living in Yu-Chi, 1999	Yu- Chi township residents	None	DRPST (trained interviewers), ≥4	54	55 (17) [16–98]
Zawadzki 2006[42] (Poland)	267	3, 27	Flood survivors (14–75 y) living in 2 most affected districts in Gdansk, 2001	Relocated residents registered by local committee	None	MISS (self-report), *DSM-IV*	59	37 (15) [14–75]

Abbreviations: CAPS = Clinician-Administered PTSD Scale, CIDI-PTSD = Composite International Diagnostic Interview-PTSD Module, DIS-DS = Diagnostic Interview Schedule-Disaster Supplement, DRPST = Disaster-Related Psychological Screening Test, DSM = *Diagnostic and Statistical Manual of Mental Disorders*, GHQ-28 = 28-item General Health Questionnaire, HTQ = Harvard Trauma Questionnaire, IES = Impact of Event Scale, ISS = Injury Severity Score, MISS = Mississippi Scale for Combat-Related PTSD, PCLC = PTSD Checklist-Civilian Version, NR = not reported, PSE=Present State Examination, PSS = PTSD Symptom Scale, PTSD = posttraumatic stress disorder, PTSD-RI=PTSD Reaction Index, PTSS-C = Posttraumatic Stress Symptoms for Children, QPTS = Questionnaire of Posttraumatic Stress, SCID = Structured Clinical Interview for DSM-III-R.