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Delayed Onset Posttraumatic Stress Disorder:

A Systematic Review of the Evidence

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Abstract

Objective: Since its introduction in DSM-III there has been controversy over the prevalence, and even the existence, of delayed-onset posttraumatic stress disorder (DOPTSD). In an attempt to resolve the discrepant findings we conducted a systematic review of the evidence concerning this condition.

Method: The literature was searched for case reports and group studies with adequate measurement of DOPTSD according to DSM criteria.

Results: Ten case studies and 19 group studies met criteria for inclusion in the review. Studies consistently showed that delayed onset PTSD in the absence of any prior symptoms was extremely rare, whereas delayed onsets that represented exacerbations or reactivations of prior symptoms accounted on average for 38.2% and 15.3% respectively of military and civilian cases of PTSD.

Conclusions: The discrepant findings in the literature concerning prevalence can be largely, but not completely, explained as being due to definitional issues. Very little is known at present about what distinguishes the delayed onset and immediate onset forms of the disorder. Continuing scientific study of DOPTSD requires future editions of the DSM to adopt a definition that explicitly accepts the likelihood of at least some prior symptoms.

Delayed-onset posttraumatic stress disorder: A systematic review of the evidence

The delayed-onset subtype of posttraumatic stress disorder (DOPTSD) was formally described in 1980 when PTSD was first recognized as a diagnostic entity in DSM-III. Its inclusion was based on existing literature indicating that many soldiers do not develop symptoms until they return home as stress reactions are not adaptive in combat (1). Since then, numerous studies have investigated the condition in combat veteran and civilian samples, with no consensus as to its prevalence. Reported rates have varied between 0% and 68% of PTSD cases, and some authors have even expressed skepticism about whether the condition exists at all (2, 3). In this article, anticipating possible revision of PTSD in DSM-V, we attempt to resolve these conflicting findings through a systematic review of the evidence from both case and group studies. We also examine evidence for triggers to onset from the case reports and review the evidence for initial differences between individuals with delayed onset, acute onset and no PTSD onset from the group studies. The wide variety of research designs and definitions of delayed onset precludes the use of formal meta-analytic techniques.

DSM-IV-TR describes delayed onset as a specifier of PTSD which “indicates that at least 6 months have passed between the traumatic event and the onset of the symptoms” (p.465). This criterion is consistent with all previous DSM versions. It is also specifically noted that PTSD symptom onset usually occurs within 3 months after the trauma, indicating that delayed onset is the exception to the rule. The inclusion of DOPTSD in DSM-III led to its acceptance as a potentially compensable disorder by the Veterans Administration (VA) in the USA. This in turn led to a rise in benefit claims for the disorder, presumably by veterans previously denied the opportunity (4). Within two years of the publication of DSM-III, Atkinson et al. (4) cautioned

against accepting all claims of delayed onset as genuine because of the secondary gains involved. In this context Sparr and Pankratz (5) reported five cases of factitious PTSD among men presenting at a VA medical center claiming to be Vietnam veterans.

Before summarizing the literature, sources of variation in definition, design and assessment are discussed that have a bearing on inclusion criteria for studies in this review and evaluation of the evidence. Starting with definition, one of the main stumbling blocks concerns ambiguity in the criterion for delayed onset in DSM. There is no clarification of whether "the onset of the symptoms…" refers to *any* symptoms that might eventually lead to the disorder or only to full-blown PTSD itself. In the former interpretation (which we term Definition 1) an individual with minimal initial symptoms that gradually worsened until full PTSD criteria were met after a substantial time period would not be classified as having DOPTSD. Given that it is very common for trauma-exposed individuals to develop at least some symptoms in the short term (6, 7), Definition 1 DOPTSD should be relatively uncommon if it exists at all. Alternatively, defining delayed onset as relating to the full disorder (which we term Definition 2) means that, in theory, the onset of just one additional symptom in an already highly symptomatic individual could be sufficient for a diagnosis of DOPTSD. Definition 2 DOPTSD should be relatively more common, therefore, leading to the prediction that the definition adopted will have a major impact on the estimated prevalence of the condition.

Only two prospective studies investigating DOPTSD explicitly interpret the DSM definition as ruling out any prior symptoms (2, 3), although both provide data on delayed onset of full PTSD with prior symptoms. There are, however, some retrospective studies where it is unclear how the definition has been interpreted (e.g., reference 8). Given this lack of clarity in the formal definition and in most research studies, we did not exclude studies from our review

that permitted DOPTSD cases to have experienced some prior symptoms. Nevertheless, evidence in any way suggestive of a prior episode meeting full criteria for PTSD, or insufficient information to rule this out, was taken into account in the selection of case reports.

A further issue concerns the length of the delay between trauma exposure and onset. A number of studies have not adhered to the specified delay of at least six months in their research design but have investigated a shorter period. We have excluded these from our review. Other studies have specified onset delays of 12 months or more, and these are included in the review as a separate category.

Retrospective studies of DOPTSD typically assess lifetime episodes with standardized clinical interviews to track the onset and offset of post-trauma symptoms over time. One disadvantage of this method is the problem of memory reliability, particularly when a long time has elapsed between trauma and interview. In consequence, a growing number of researchers have conducted prospective studies using either clinical interviews, questionnaires, or a combination of the two at pre-determined assessment points. This method also has its disadvantages as individuals may have had onsets of PTSD after one assessment that then remitted before the next assessment. If, as has often been the case, there has been no retrospective assessment between time points, this can lead to unreliable estimates of DOPTSD. An overestimate is likely if these undetected remitted onsets occurred within six months post-trauma, whereas an underestimate is likely if they occurred later than six months post-trauma.

To summarize data on the prevalence of DOPTSD it is crucial to be able to compare rates between studies using the same metric. Three different ways of expressing frequency are apparent from the literature. The first is as a proportion of all respondents, the second as a proportion of all respondents with no prior (acute) PTSD, and the third as a proportion of all

respondents with PTSD. Estimates using the first two calculations appear to be most favored but are dependent on the overall rate of PTSD in the study sample. As PTSD rates vary considerably according to the characteristics of the sample and the type of trauma experienced (9), valid comparisons cannot be made with their use. For review purposes the optimal way to express the rate of delayed onset PTSD is as a proportion of all those with PTSD.

The purpose of this review is to resolve the uncertainty about how to interpret existing data on DOPTSD. We use rigorous selection criteria to identify the case reports and group studies that can tell us most about its existence, prevalence, and characteristics. Case studies are uniquely valuable in establishing whether a condition exists and in generating hypotheses about its causes and characteristics. Group studies yield less detailed information but provide a basis for making prevalence estimates and comparisons between immediate onset, DOPTSD, and no-PTSD cases.

Method

Selection of Articles

English-language articles published since 1980 and including data on delayed onset PTSD (defined in a way consistent with the DSM-III, DSM-III-R or DSM-IV) were located from bibliographic databases, recent journal issues, and secondary sources such as journal articles and book chapters. The Social Science Citation Index, PsycINFO, PUBMED and the PILOTS database managed by the National Center for PTSD were searched using the terms PTSD or Posttraumatic Stress Disorder and delayed onset or delay. In a further search the terms longitudinal and prospective were used with PTSD or Posttraumatic Stress Disorder. Articles describing group studies were included if they met the following criteria:

- Delayed onset referred to syndromal (i.e. meeting full DSM criteria) or subsyndromal PTSD rather than non-specific symptoms or behaviours such as help-seeking.
- The study included a valid PTSD assessment such as a diagnostic interview, a questionnaire with all PTSD criteria symptoms included and a designated/valid diagnostic cut-off score, or retrospective assessment of case records as long as PTSD was the explicit focus when the case record was taken.
- Delayed onset of symptoms was able to be assessed as occurring more than 6 months post trauma.
- Time to onset was from the time of specific trauma exposure or the end of potential exposure, such as the end of deployment to a war zone.
- The study included a clinical, community, military, or specific trauma sample (e.g., motor vehicle accident survivors).
- The study sample did not comprise a special population such as litigants or delayed help-seekers.

Inclusion criteria for case studies are listed separately:

- As it was uncommon for standardised diagnostic interviews to have been used, we required explicit mention that a formal PTSD diagnosis was made using DSM criteria.
- The study provided clear information to establish onset of full PTSD for the first time more than 6 months after the trauma; earlier symptoms suggestive of full PTSD were explicitly ruled out.

The search yielded 74 relevant studies of which 39% met our inclusion criteria. Of 31 case studies, 10 met inclusion criteria. Of the 21 that did not, 16 did not report a formal PTSD diagnosis, 7 reported evidence indicative of a reactivation of an immediate post-trauma

condition, and 7 did not provide sufficient information to rule this out (numbers sum to more than 21 due to overlapping categories). Of the 43 group studies, 19 met inclusion criteria. The cohort of prisoners of war used in Engdahl et al (10) which was included, was also used in Speed et al (11) and Kluznick et al (8) which were excluded due to overlap. Of the 22 other excluded studies, 8 did not include a valid PTSD assessment to detect delayed onset, 12 included onsets with a delay of less than 6 months post- trauma, and 2 described special populations. Authors of group studies were contacted for clarification of assessment procedures where relevant.

Results

Case Studies

Table 1 provides details of the 10 studies that met inclusion criteria for review. Seven reported DOPTSD in relation to war experiences and 3 in relation to motor vehicle accidents (MVA). Four of the studies used DSM-IV diagnostic criteria which includes impairment (criterion F). Close inspection of the text indicated clear evidence of impairment in all studies including the 6 that used earlier DSM versions not requiring impairment. Overall, the war experience articles described 23 cases, with 15 accounted for by one article (12). With one exception (13) the cases were elderly World War II or Korean War veterans with PTSD onsets delayed by at least 30 years. The young soldier described by Solomon et al (13) had an onset around 12 months after his tour of duty. In contrast, all 4 cases described in the MVA articles were younger (age range 23 – 45) with onset delays ranging from 10 months to 4 years. The evidence used to rule out earlier post-trauma episodes is described in Table 1 for each reported case. Probably the most convincing evidence was from 3 MVA cases who were under continuing medical care for their physical injuries prior to onset, making detection of symptoms in the post-

trauma period more likely if present (14, 15). In the 6 studies describing elderly war veterans with very long durations to first onset, 18 of the 22 cases were corroborated by someone else, mostly relatives and usually the spouse (12, 16, 17). Given the age of the veterans this does not rule out the possibility of episodes in the early months or years post-trauma that might have gone undisclosed or been forgotten, a limitation noted in 2 studies (12, 16). The corroborative evidence suggests at least that the veterans in question had very long relatively symptom-free periods before onset in old age.

All the case studies reported events or circumstances that could have triggered onset (Table 1). Over half of all the cases (15/27) were attributed to an onset of a physical illness, and they all involved the elderly veterans (12, 16, 18, 19). With one exception (19), all were neurological illnesses or conditions that may affect cognition. In nearly a quarter of the cases (6/27) the triggers described were reminders of the original trauma (14, 18, 20, 21). Two examples were a subsequent near-miss accident experienced by an MVA victim (14), and a severe head injury to the son of a veteran who had himself sustained a head injury as part of his traumatic experiences (18).

Group Studies

In 13 of the 19 group studies, delayed onset was able to be defined as at least 6 months after trauma exposure. Four of these yielded data on the proportion of respondents for whom no posttraumatic symptoms at all were observed in this period (corresponding to Definition 1 of DOPTSD above), whereas the other 9 studies reported the proportion of people who only met full criteria for PTSD after this period (corresponding to Definition 2 above). In the remaining 6 studies, delayed onset of full PTSD was able to be defined as 1 year or more after trauma exposure (also corresponding to Definition 2). Only 2 of the 19 studies (27,31) used DSM-IV

criteria that includes the impairment. criterion. None of the other studies used this criterion in the relevant analyses.

Of the 4 studies that provide evidence relevant to Definition 1 (Table 2), 2 report numbers of respondents with full PTSD (24, 25) and 2 report numbers of respondents with both syndromal or subsyndromal PTSD after 6 months (22, 23). Of all the group studies reviewed, these were the only ones to include subsyndromal cases. In Prigerson et al's study 37 of the 172 PTSD cases (22%) were subsyndromal. The majority met all DSM-III-R symptom cluster criteria, but lacked the symptom duration criterion (Maciejewski personal communication). In Helzer et al's study PTSD was rare and nearly all (94%) of the 422 cases were subsyndromal, defined as having any PTSD symptoms. Given that all these studies investigating onset of posttraumatic symptoms used retrospective interviews and reported the course of symptoms in the context of delayed onset, it was assumed that earlier, now remitted, onsets were taken into account in the assessment. All of these studies are distinguished from other group studies reviewed inasmuch as they are epidemiological investigations of a variety of traumas with varying lengths of time between trauma and assessment . Helzer et al. (22) and Prigerson et al. (23) both reported higher rates of delayed symptom onset in combat trauma than in civilian traumas. In Helzer et al's (22) study, there were no delayed symptom onsets among those with civilian traumas. In two studies Breslau and colleagues (24, 25) similarly found minimal or no evidence for delayed onset in studies of young mothers and of adults too young to have been Vietnam conscripts. In the young mothers study, these authors reported that "With the exception of one case, PTSD symptoms began within days of exposure" (25, p. 84).

Table 3 presents details of the 9 studies with 6-month delayed onset of full PTSD. Studies are arranged by the length of the period covered post-trauma which ranged from 9 months to 50

years. Six of the studies were longitudinal and the three with the longest assessment periods were retrospective in design. With one exception, (26) all used clinical interviews to assess PTSD. We looked for factors that might affect delayed onset rates, the first being the length of the post-trauma assessment period. This was on the assumption that the longer the period assessed beyond the first 6 months, the more opportunity there would be for respondents to develop DOPTSD. Table 3 shows that with two exceptions (10, 27), the pattern of delayed onset rates broadly fitted our assumptions. The study with the shortest duration of 9 months (28) found no onsets delayed by more than 6 months. It should be noted that Yule et al's (27) study differed from the others in that the respondents were all schoolchildren at the time of the trauma. Also notable is that 2 of the 3 studies with military samples reported similar delayed onset rates that were much higher than the civilian studies (26, 29) despite differences in design and duration times (2 and 18 years). However, Engdahl et al's (10) retrospective investigation of veterans who were all prisoners of war is inconsistent with this pattern. These authors found only two cases of delayed onset out of 140 PTSD cases.

Another factor considered was if the studies assessed whether respondents could have had onsets of PTSD that then remitted before the next assessment point. As already noted, lack of such an assessment could lead to both over- and underestimates of delayed onset cases. The last two columns in Table 3 show whether the studies assessed remitted onsets between time points as well as whether earlier episodes of PTSD within 6 months of the trauma could be ruled out. The table shows that although 3 of the longitudinal studies were negative on both counts (2, 26, 30), this did not appear to affect the delayed onset rates relative to the other studies that had included these more detailed assessments. For example, both North et al (3) and Sungur & Kaya (30) with similar study durations reported delayed onset rates of 21%, although only the former

assessed symptoms between time points. One possible explanation is that the under- and overestimates that might be a consequence of assessment omissions cancel each other out.

In the final category of 6 studies, listed in Table 4, delayed onset is defined as occurring at least one year after trauma exposure. Four studies were longitudinal and these all defined delayed onset as one year post trauma (31, 32, 33, 34). The remaining 2 were retrospective and defined onset delay as 2 years post-trauma (35), or 5 years post-trauma (36). The period covered post-trauma ranged from 3 to 40 years in the 6 studies. Three studies were of war-related trauma and included 2 military samples (34, 35) and a sample of resistance fighters (36). The other 3 studies involved motor vehicle accidents (31, 32) and a mass shooting (33). As with the studies listed in Table 3 it was expected that the longer the post-trauma period covered, the higher the rate of delayed onset would be. However, this might be moderated by the defined post-trauma delay: the longer the delay the less chance there would be of delayed onsets developing subsequently.

The 4 longitudinal studies with defined delays of 1 year confirmed the previous pattern found in the 6-month delayed onset studies that the longer the period covered, the higher the rate of delayed onsets. The 2 studies covering 3 years post trauma had very similar delayed onset rates (31, 33) despite the fact that prior episodes were not ruled out in one (31). Solomon & Mikulincer (34), with the longest period covered of the 4 studies (20 years), reported a particularly high rate of 63% in Israeli war veterans assessed as having no combat stress reaction during the 1982 Lebanon war. This study is of particular interest as it had four assessment points at 1, 2, 3 and 20 years and reported delayed onset rates at each time point. The study showed that the majority of delayed onsets (12/20) occurred in the relatively short interval between 1 and 2 years post trauma. It was possible to compare this study with Bremner et al.’s (35) investigation

of American war veterans that used a retrospective interview to cover a similar time period and had delayed onset defined as 2 years post trauma. As shown in Table 4, expressed as a proportion of all PTSD cases, 2-year delayed onset rates were remarkably similar (25% and 23%).

The last study in this category is that of Op den Velde et al. (36) who reported that around two-thirds of all PTSD cases in their sample of Dutch World War II resistance fighters were delayed by as much as 5 years. This investigation used a retrospective clinical interview. The authors distinguished onsets of PTSD which gradually developed from an earlier subsyndromal condition from delayed PTSD in which the first symptoms did not appear until at least 5 years after the war. It is of interest to contrast the delayed onset rate in all PTSD cases in this study (84/123: 68%) with that of Engdahl et al. (10) (2/140: 1.4% - Table 3), as both involved male combatants and used retrospective interviews to cover 40 years or more (the longest time periods in the studies reviewed). It is not immediately clear why there should be such a discrepancy. It cannot be explained by the element of captivity as over half of the resistance fighters had been in concentration camps.

To summarize the frequency of DOPTSD for the 6 military and 9 civilian studies in Tables 3 and 4, we calculated the mean rates weighting for sample size, length of delay and length of follow-up period. The weighted means were 38.2% and 15.3% respectively for military and civilian studies. These were similar to the unweighted means (42.4% and 15% respectively). Considering these 15 studies overall, 8 reported information on prior symptoms and in 6 of these 8 all delayed onset cases had prior symptoms (information for reference 10 supplied by personal communication with first author). However none systematically reported information to indicate how many symptoms were involved in the transition to full syndromal criteria.

Evidence for differences between groups with delayed onset, acute onset and no PTSD

We examined the studies reviewed to identify those that had conducted systematic group comparisons and found three. Watson et al's (29) retrospective study of combat veterans found no significant differences between 32 acute and 31 delayed onset cases on current symptom profiles or measures of current repression or life-stress. However all respondents had current PTSD. The two longitudinal studies of civilian trauma did not always compare the same groups and numbers in the delayed onset groups were very small (5 and 7) making interpretation of non-significant differences questionable (2, 37). Both studies compared groups on at least some initial post-trauma PTSD symptom clusters and heart rate and prior psychiatric history. Bryant and Harvey (2) compared delayed onset cases with both acute cases and those without PTSD. The purpose of Buckley et al.'s (37) study was primarily to compare initially symptomatic delayed onset cases with symptomatic trauma victims who did not develop PTSD. The only symptom cluster assessed in both studies was avoidance symptoms and the only group comparison they had in common was between delayed onset and no PTSD groups. Neither study found differences between delayed onset and no PTSD groups on prior psychiatric history. However, Buckley et al. (37) found a significant group difference on PTSD avoidance symptoms, but no group difference on initial heart rate, the reverse of what was found by Bryant & Harvey (2). Given the small numbers with DOPTSD no further description of individual findings is warranted.

Discussion

It is of note that the majority of the relevant articles have not complied with DSM criteria in the assessment and diagnosis of DOPTSD, even though a number of these have been widely cited as evidence for the condition. This is likely to have hindered the field coming to reliable conclusions. The evidence from studies that have complied with DSM criteria clearly indicates that definitional issues are critical in resolving the controversies about the existence of DOPTSD. If the most conservative definition (Definition 1) is taken, namely that the onset of any PTSD symptom is delayed by at least 6 months, DOPTSD appears to be extremely rare outside of military samples. Even within these samples there is disagreement about whether it exists to any appreciable extent, and the higher prevalence estimates have only been found in large-scale surveys where detailed questioning is unlikely to have been possible. If DOPTSD is defined in this way, the scope for any scientific research will be very limited.

There is, on the other hand, no reason for skepticism concerning the existence of DOPTSD when the more inclusive Definition 2 is used. This type of DOPTSD accounts for well over a third of PTSD cases in military samples and 15% of cases in civilian samples according to our estimates. Prevalence seems to be affected, as might be expected, by the length of the delay specified prior to onset and the length of the follow-up period, but adjusting for both these variables, as well as sample size, does not greatly alter the prevalence rates based on existing studies. Likewise, longitudinal studies yield similar prevalence estimates whether or not remitted onsets between assessments are investigated. From the studies that have included relevant information, we can conclude that this type of DOPTSD is preceded by prior symptoms in the great majority of cases. Little information is available on the level of these prior symptoms, but case reports are consistent in indicating that sudden sharp increases in the number and severity of symptoms are possible.

Table 5 presents a description of this type of typical delayed onset. It is based on a composite of individual war veterans studied by us reporting DOPTSD. The phenomenon is sufficiently common to pose important questions about the mechanisms that can explain why in some individuals initial symptoms are not followed by normal recovery but remain vulnerable to reactivation and even exacerbation. These mechanisms will probably include some brought into play by external triggers, some purely internal mechanisms such as suppression and inhibition, and their interaction (38).

With the exception of the only study based solely on a prisoner of war sample (10), all the military studies consistently showed high rates of DOPTSD regardless of whether the studies were retrospective or longitudinal. Although only military samples have been followed over long periods of time, there is evidence that a substantial proportion of DOPTSD occurs soon after return from deployment. Longer follow-up of civilian samples may help to explain this difference in rates. The consistency of findings from a variety of different research designs and settings in different countries and at different times suggests that exaggerated reporting due to the desire for compensation is unlikely to be a major factor. If it does occur, the pattern of findings suggests that it is most likely to involve exaggeration of symptoms previously experienced in a milder form. Further, it is not clear that exaggerated reporting can explain the substantial difference in DOPTSD rates between military and civilian populations as compensation is also likely to be a factor in non-military trauma. In two civilian studies compensation did not predict the development of PTSD although the numbers involved were very small (32, 37). Finally it may be noted that in the post-Vietnam era, with the diagnosis of PTSD firmly established, there is less reason why the search for compensation would specifically involve delayed-onset rather than acute-onset PTSD.

Other explanations for high rates of DOPTSD in military samples rely on theory and clinical observation rather than systematic evidence, but provide some hypotheses for future investigation. Some authors predicted a plethora of delayed stress reactions after the Vietnam War and pointed to the role of emotional numbing and denial facilitated by troop management and military training (39, 40). Horowitz and Solomon (39) speculated that the nature of modern warfare (short periods of combat interspersed with periods of relative safety) is conducive to the maintenance of denial and numbing reactions. The perception of prolonged safety on repatriation might subsequently result in the relaxation of defensive coping strategies after a period of good functioning, leading to the re-emergence of intrusive trauma recollections. In the same vein, Shatan (40) suggested that delayed stress reactions in Vietnam veterans were secondary to emotional anaesthesia resulting from both the trauma and from military training that discouraged grief and intimacy (40). In the context of the 1982 Lebanon war, other authors have been mindful of the role of subsequent war stressors that might act as reminders of earlier combat stress in triggering DOPTSD (34). Indeed, it cannot be ruled out that immediate reactions to trauma from subsequent tours of duty might account for some cases of supposed DOPTSD.

Review of the relevant case studies provides a further source of explanation specific to DOPTSD in the elderly. However, in this context we cannot rule out the possibility that the very long delays involved may have concealed a reactivation of previous conditions rather than true delayed onset. The most prevalent triggers to DOPTSD in the case studies were related to the onset of neurological and other disorders affecting cognition in elderly combat veterans. As mentioned by some authors (e.g., reference 16) such disorders are likely to involve damage to the prefrontal cortex, leading to disconnections with other brain areas. The inhibiting effect of the prefrontal cortex on regulating thoughts and emotions may therefore be lost (41). A compatible

explanation provided by Grossman et al (16) is that decreased cognitive functioning leads to diminished capacity to utilize previously successful coping strategies to ward off aversive thoughts and symptoms.

In conclusion, this review has gone some way to resolving the uncertainty about how to interpret the existing data on delayed-onset PTSD. However, given the diversity of methodologies in existing studies, some ambiguities and uncertainties remain. Establishing reliable knowledge about DOPTSD will depend on the adoption by future editions of the DSM of a more explicit definition that includes the likelihood of at least some prior symptoms becoming exacerbated or reactivated. This definition implies that there is a need to discriminate between those individuals whose initial symptoms are part of normal recovery and those who remain at risk. In addition, it would be valuable to know whether DOPTSD is equivalent to acute-onset PTSD in terms of risk factors, type of trauma exposure, clinical features, course, and response to treatment. For example, do formal interventions, social support, and subsequent life stress play more of a role in one condition than the other? Finally, it should be borne in mind that few existing studies have had DOPTSD as their primary focus. In consequence they have often had insufficient statistical power, and further research is needed with larger samples of individuals with DOPTSD to identify and shape interventions in vulnerable groups.

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Table 1. Case Studies meeting inclusion criteria for Delayed Onset PTSD

Reference	Trauma Type	N	Age	Gender	Length of time to onset post-trauma	PTSD diagnostic criteria	Evidence to rule out previous episodes	Trigger
Bryant (1996)	MVA (Brain damaged victims with short-term posttraumatic amnesia)	2	27 23	Male	1. 10 months 2. 17 months	DSM-IV	Both under hospital care and appeared un-distressed prior to onset. Timing of symptom onset carefully recorded	1. Near miss accident 2. Police evidence contradicted his report that he had been the driver
Burstein (1985)	MVA (evidence for case 2 only)	1	45	Female	12 months	DSM-III	Referring physician corroborated onset delay. Patient reported abrupt delayed onset	Realising physical injuries were not minor and would require continuing treatment
Cassiday & Lyons (1992)	WWII Torture & head/lung injury	1	63	Male	37 years	DSM-III-R	Good post-war functioning verified by VA	Son's head injury triggered symptom onset. Cerebral vascular accident triggered full diagnosis
Grossman et al. (2004)	WWII Concentration camp survivor and war nurse	2	74 80	Male Female	Many years on	Formal PTSD criteria met	Relatives corroborated lack of prior PTSD symptomatology Authors note caution	Onset of neurological illness: CVA and small vessel disease
Hermann & Eryavec (1994)	WWII combat experiences	2	68 72	Male	30 years +	DSM-III-R	Symptoms not experienced during service and no previous psychiatric history in either case	1. Medical Illness: rheumatoid arthritis 2. Death of wife
Hilton (1997)	WWII combat experiences and liberating concentration camp (evidence for case 1 only)	1	72	Male	50 years	DSM-IV	Lack of previous psychiatric history; discharged A1. Occasional anxiety symptoms.	Seeing Auschwitz documentaries

Reference	Trauma Type	N	Age	Gender	Length of time to onset post-trauma	PTSD diagnostic criteria	Evidence to rule out previous episodes	Trigger
Lim (1991)	MVA in a military context	1	30	Male	4 years	DSM-III-R.	Detailed history revealed earlier avoidance of accident reminders only	Colleague injured by similar military vehicle
Ørner & de Loos (1998)	WWII Combat & POW	1	71	Male	Many years on	DSM-IV	'Unremarkable medical history' prior to dramatic onset with flashbacks. Wife confirmed.	Re-enlistment triggered anxiety and panic reactions. Argument with work colleague triggered full PTSD diagnosis
Ruzich et al. (2005)	WWII & Korean War Combat-related experiences	15	68-86	Male	56 years approx	DSM-IV	Psychiatric history systematically assessed. Lack of psychiatric history in all cases confirmed by corroboration of family member or local medical officer	Medical conditions that may affect cognition = 9 Dementia = 2 Environmental stressors = 4
Solomon et al. (1989) [Case described in group study]	1982 Lebanon war experiences	1	25	Male	12+ months post combat	DSM-III	Military psychiatric records showed some nightmares immediately after trauma	Next call-up for military service

MVA = Motor Vehicle Accident
WWII = World War II
POW = Prisoner of War

Table 2. Group studies reporting onset of any PTSD symptoms delayed by at least 6 months after trauma exposure

Reference	Trauma Type	N	Age range (M, SD)	Gender	Diagnosis	Design	Period covered post-trauma	Proportion with delayed PTSD symptom onset
Breslau et al (1991)	Mixed	1007	21 - 30	Mixed	DSM-III-R	Retrospective Interview	Not specified	1% (1/93)
Breslau et al. (1997)	Mixed	801	(31)	Women (mothers)	DSM-III-R	Retrospective Interview	Not specified	0% (0/111)
Helzer et al. (1987)	Mixed	2493	Not specified	Mixed	DSM-III	Retrospective Interview	Not specified	16% combat trauma 0% civilian trauma
Prigerson et al. (2001)	Mixed	1703	15 – 24	Men	DSM-III-R	Retrospective Interview	Not specified	22% combat trauma 8% civilian trauma

Table 3. Group studies reporting onset of full PTSD delayed by at least 6 months after trauma exposure (arranged by period covered post-trauma)

Reference	Trauma Type	N	Age Range (M, SD)	Gender	Diagnosis	Design (number of assessment points)	Period covered post-trauma	Proportion of PTSD cases with delayed-onset	Remitted onsets assessed between time points?	Prior episodes within 6 months of trauma ruled out?
Epstein (1993)	Accidental injury	15	20 – 83	Mixed	DSM-III-R	Longitudinal Interview (5)	9 months	0% (0/6)	Yes	Yes
Buckley et al. (1996)	MVA	107	Not specified for whole sample	Mixed	DSM-III-R	Longitudinal Interview (3)	12 months	10% (7/69)	Yes	Yes But all DO initially subsyndromal.
North et al. (2004)	Bombing	137	(44.1, 10.8)	Mixed	DSM-III-R	Longitudinal Interview (2)	17 months	21% (12/56)	Yes	Yes: But all DO sub-syndromal at 6m
Sungur & Kaya (2001)	Riot/Fire	79	Not specified	Mixed	DSM-III-R	Longitudinal Interview (4)	18 months	21% (8/39)	No	No But 1initially subsyndromal DO reported
Bryant & Harvey (2002)	MVA	103	17 – 63	Mixed	DSM-III-R	Longitudinal Interview (3)	2 years	18% (5/28)	No	No But 2 initially subsyndromal DO reported
Southwick et al. (1995)	War: Military	62	Not specified	Mixed	DSM-III-R	Longitudinal Questionnaire (3)	2 years	50% (4/8)	No	No

Reference	Trauma Type	N	Age Range (M, SD)	Gender	Diagnosis	Design (number of assessment points)	Period covered post-trauma	Proportion of PTSD cases with delayed-onset	Remitted onsets assessed between time points?	Prior episodes within 6 months of trauma ruled out?
Yule et al. (2000)	Shipping disaster	217	17 – 25	Mixed	DSM-IV	Retrospective Interview	4 -7 years	10% (11/110)	Yes	Yes But all DO had some symptoms prior to onset
Watson et al. (1988)	War: Military	63	Not specified for whole sample (34 approx)	Men	DSM-III	Retrospective Interview	18 years	49% (31/63)	Yes	Yes
Engdahl et al. (1998)	War: Military POW	262	71 (median)	Men	DSM-III-R	Retrospective Interview	50 years	1.4% (2/140)	Yes	Yes But both DO had some symptoms prior to onset

DO = Delayed onset

Table 4. Group studies reporting onset of full PTSD delayed by at least 1 year after trauma exposure (arranged by defined onset delay and period covered post-trauma)

Reference	Trauma Type	N	Age Range (M, SD)	Gender	Diagnosis	Design (Number of assessment points)	Defined onset delay	Period covered post-trauma	Proportion of PTSD cases with delayed-onset	Remitted onsets assessed between time points?	Prior episodes within defined delay period ruled out?
Koren et al. (2001)	MVA	58	(27) (approx)	Mixed	DSM-IV	Longitudinal Interview (2)	1 year	3 years	10% (2/21)	Yes	No
North et al. (2002)	Mass shooting	116	18 – 83	Mixed	DSM-III-R	Longitudinal Interview (3)	1 year	3 years	9% (4/44)	Yes	Yes But all DO initially subsyndromal
Mayou et al. (1997)	MVA	111	17 – 69	Mixed	DSM-III-R	Longitudinal Interview +. Follow-up Questionnaire (3)	1 year	5 years	36% (8/22)	No	No
Solomon & Mikulincer (2006)	War : Military (non-clinical sample)	83	38 – 57 (approx)	Men	DSM-III	Longitudinal Questionnaire (4)	1 year After war	20 years	63% (20/32) [25% 2 years]	No	Partly (no combat stress reaction in war)
Bremner et al. (1996)	War: Military	61	(45, 2.4)	Men	DSM-III-R	Retrospective: Interview.	2 years after combat tour	24 years	23%(14/61)	Yes	Yes But all had some symptoms prior to onset
Op den Velde et al. (1993)	War: Resistance Fighters	147	60 – 65	Men	DSM-III-R	Retrospective Interview	5 years after war	40 years	68% (84/123)	Yes	Yes

DO = Delayed onset

Table 5. Vignette of a war veteran with delayed onset PTSD

Jim served as a private in the army for five years in his early 20s.During this time he was deployed to Northern Ireland where he witnessed the shooting and death of his room mate who was a close friend. Subsequently he reported an occasion when two bombs went off in quick succession in his camp. He suffered hearing loss and had to clean up the aftermath, including the blood of injured friends.

He developed symptoms of avoidance and arousal soon after this last trauma, but did not have any reexperiencing symptoms until 18 months later which was 8 months after his army discharge. It was at this stage that he met criteria for full PTSD. He says it took a long time for all the memories to come back. But nothing really sparked them, because he avoided thinking about it and tried to lead a normal life: “I’m a bottler, always have been, I bottle things up.” His intrusive thoughts and memories have constricted his normal activities, “It has a knock-on effect in terms of how you feel about yourself, so yeah, it’s really held me back and still does”.

The first time he showed any emotions to anyone else was when he received psychiatric help 18 months after he left the forces: “When I started to speak about the events I was really a physical and emotional wreck.”