An Update of Acute Stress Disorder

It is nearly 20 years since DSM-IV introduced the diagnosis of acute stress disorder (ASD). At the time there was relatively little research substantiating the role or definition of diagnosis, but its introduction promoted an unprecedented amount of research into acute stress reactions after trauma. As we approach the introduction of DSM-5, it is timely to review what we now know about acute traumatic stress and how this is influencing the new definition of the ASD diagnosis.

ASD was initially introduced for two reasons: (a) to describe severe acute stress reactions that occur in the initial month after a trauma that could not be described as PTSD, which can only be diagnosed after a month has transpired since the trauma; and (b) to identify acutely traumatized people who will subsequently develop PTSD as opposed to experiencing a transient stress reaction (Spiegel et al., 1996). To satisfy criteria for an ASD diagnosis in DSM-IV, one needed to experience a traumatic event and respond with fear, horror, or helplessness (Criterion A), and similarly to PTSD, needed to satisfy re-experiencing (Criterion C), avoidance (Criterion D), and arousal (Criterion E) symptom clusters. ASD was markedly differentiated from PTSD by a strong emphasis on acute dissociation, such that one needed to have at least three of the following symptoms: emotional numbing, derealization, depersonalization, reduced awareness of surroundings, or dissociative amnesia (Criterion B). This emphasis on dissociation was based largely on the perspective that dissociative responses to trauma are pivotal in longer-term psychopathology (Harvey and Bryant, 2002).

Longitudinal Evidence for Acute Stress Disorder

A series of longitudinal studies have assessed the relationship between ASD and subsequent PTSD. One recent review of 22 studies concluded that overall the ASD diagnosis is sensitive in predicting PTSD; that is, the majority of individuals with a diagnosis of ASD do subsequently develop PTSD (Bryant, 2011). In contrast, the ASD diagnosis has low specificity; that is, most people who eventually experience PTSD do not initially display ASD. These studies suggest that although trauma survivors with ASD do experience stress reactions that are likely to persist, the current criteria is overly narrow in identifying people at risk.

What is the reason for the poor predictive ability of ASD? Some studies suggest that the emphasis on dissociation may be overly limiting because many people at risk may not display acute dissociative responses (Bryant, Creamer, O’Donnell, Silove, and McFarlane, 2008; Dalgleish et al., 2008; Harvey and Bryant, 1998; Kassam-Adams and Winston, 2004). This finding is consistent with other evidence concerning the relationship between acute dissociation and subsequent PTSD. Although there are numerous studies attesting to the predictive capacity of acute dissociation and subsequent PTSD, Murray, Ehlers, and Mayou, 2002, other analyses suggest that peritraumatic dissociation is not an independent predictor of PTSD (Breh and Seidler, 2007; van der Velden et al., 2006). One study found that dissociation mediates the relationship between acute arousal and subsequent PTSD (Bryant, Brooks, et al., 2011), suggesting that it may be the arousal rather than dissociation at the time of trauma that is pivotal in PTSD development.

There are only a few studies of the relationship between ASD and PTSD in children (Bryant, Salmon, Sinclair, and Davidson, 2007; Dalgleish et al., 2008; Kassam-Adams and Winston, 2004). Although limited by the small number of studies, and the focus of all of them on traumatic injury, these studies indicate that the ASD diagnosis has poor capacity to predict PTSD in injured children. We know much less about the trajectories of trauma response in children than...
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Although the definition of ASD has changed, the existing measures of ASD are still useful because the symptom composition is largely unchanged. Scoring of the measures would need to be modified because the new definition no longer requires that each cluster of symptoms is satisfied; instead, each measure would need to determine the presence of ASD on the basis of 9 symptoms being present. There are three measures of ASD to choose from. The first measure developed for ASD was the Stanford Acute Stress Reaction Questionnaire, which is a self-report measure that indexes 30 possible symptoms of ASD (Cardena, Koopman, Classen, Waelde, and Spiegel, 2000). An alternate measure is the Acute Stress Disorder Interview (Bryant, Harvey, Dang, and Sackville, 1998), which possesses good sensitivity (92%), and specificity (93%) relative to independent clinical diagnosis. The Acute Stress Disorder Scale (Bryant, Moulds, and Guthrie, 2000) is a self-report version of the Acute Stress Disorder Interview, which also has sound psychometric properties.

Alternate Options for Assessing High-risk Trauma Survivors

The modest predictive accuracy of the ASD diagnosis raises questions concerning alternate means to identify recently traumatized people who will progress to PTSD. In short, we currently have little knowledge in terms of acute markers that we can rely on to predict who will develop the disorder. In light of the previously reviewed complexity of the trajectories that we see following trauma, it is perhaps not surprising that acute markers do not perform very well as predictors. This qualification notwithstanding, several acute biological markers have been shown to have a statistical relationship with longer-term PTSD severity. These include elevated resting heart rate, elevated respiration rate, elevated cortisol, low Gamma-Amino Butyric Acid (GABA) plasma levels, and FKBP5 mRNA expression, indicating glucocorticoid activation. Cognitive predictors include maladaptive appraisals about the experiences and one’s responses, over general retrieval of autobiographical memories, shame, and rumination. It must be emphasized, however, that although each of these factors significantly predict subsequent PTSD symptoms, the sensitivity and specificity is modest. That is, if we rely on these factors to screen recently traumatized people who may require secondary prevention, we are likely to either be overly inclusive of many people who are only experiencing a transient stress response or we will be excluding many people who may develop chronic PTSD: for reviews, see Bryant, 2003.

Conclusions

Although it is tempting to seek a simple means to screen people shortly after trauma and to whom we can offer early intervention, the increasing evidence points to this being an unrealistic goal. The course of posttraumatic stress is fluctuating, impeding attempts to accurately identify most people at risk for PTSD. Accordingly, ASD now limits its goal to describing those who are suffering immediate severe reactions and can benefit from intervention in the short-term. This is worthy goal in its own right because it can allow the distress of many people to be addressed with short-term evidence-based treatments, which can alleviate distress that can otherwise be impairing.
In this article, we consider the limitations of these perspectives and argue for a broader theoretical approach that takes into account the natural heterogeneity of trauma reactions over time. To that end, we review recent attempts to identify prototypical patterns or trajectories of trauma reaction that include chronic dysfunction, but also delayed reactions, recovery, and psychological resilience. We consider the advantages but also the limitations and ongoing controversies associated with this approach. Finally, we introduce promising new research that uses relative sophisticated advances in latent growth mixture modeling as a means of empirically mapping the heterogeneity of trauma responses and consider some of the implications of this approach for existing trauma theories.

Breh, D.C., and Seidler, G.H. (2007). Is peritraumatic dissociation a risk factor for PTSD? Journal of Trauma and Dissociation, 8, 53-69. doi:10.1300/J229v08n01_04 In the literature, peritraumatic dissociation is frequently considered to be a risk factor for PTSD. In the last few years, a large number of studies have investigated the connections between PTSD and peritraumatic dissociation. A meta-analysis was conducted, including 35 empirical studies that discuss the connections between peritraumatic dissociation and PTSD. Meta-analysis makes it possible to undertake a systematic integration of findings produced by primary studies of this kind to date. The average effect size was $r = 0.36$, indicating a significant positive correlation between the two dimensions. In this article, the authors make a distinction between correlate and risk factor. Therefore, subgroup analyses of quasi-prospective and retrospective studies were undertaken with a view to establishing whether peritraumatic dissociation represents a risk factor for the development of PTSD following a traumatic event. The average effect size in the quasi-prospective studies was $r = 0.34$, which was significant. From a methodological viewpoint, the results show that peritraumatic dissociation is a moderate risk factor for PTSD. Nonetheless, the conceptualization of peritraumatic dissociation in the framework of psychotraumatic stress syndromes—is it a predictor, a symptom, or something else entirely—needs to be addressed by future research.

Bryant, R.A. (2003). Acute stress reactions: Can biological responses predict posttraumatic stress disorder? CNS Spectrums, 8, 668-674. What biological responses characterize those acute trauma reactions that develop into chronic psychiatric disorder? The need to understand the genesis of posttraumatic psychological disorders has resulted in much attention on biological reactions in the initial aftermath of trauma exposure. This review outlines the prevailing biological models of acute stress reaction and critiques the available evidence concerning biological responses to trauma that are associated with subsequent psychological disorder. The roles of peritraumatic dissociation and vulnerability factors for acute stress reaction are also reviewed. The major challenges for research on psychobiological responses to trauma are highlighted.

Bryant, R.A. (2011). Acute stress disorder as a predictor of posttraumatic stress disorder: A systematic review. Journal of Clinical Psychiatry, 72, 233-239. doi:10.4088/JCP.09r05072br. The utility of the ASD diagnosis to describe acute stress reactions and predict subsequent PTSD was evaluated. A systematic search was conducted in the PsycINFO, MEDLINE, and PubMed databases for English-language articles published between 1994 and 2009. Studies were selected that assessed for ASD within 1 month of trauma exposure and assessed at a later time for PTSD using established measures of ASD and PTSD. For each study, capacity of the ASD diagnosis to predict PTSD was calculated in terms of sensitivity, specificity, and positive and negative predictive power. For studies that reported subsyndromal ASD, the same analyses were calculated for cases that initially satisfied subsyndromal ASD criteria. Twenty-two studies were identified as suitable for analysis (19 with adults and 3 with children). Diagnosis of ASD resulted in half the rate of distressed people in the acute phase being identified relative to including cases with subsyndromal ASD. In terms of prediction, the ASD diagnosis had reasonable positive predictive power (proportion of people with ASD who later developed PTSD). In contrast, the sensitivity was poor (proportion of people who developed PTSD who initially met criteria for ASD). The ASD diagnosis does not adequately identify the majority of people who will eventually develop PTSD. There is a need to formally describe acute stress reactions but this may be achieved more usefully by describing the broad range of initial reactions rather than attempting to predict PTSD.

Bryant, R.A., Friedman, M.J., Spiegel, D., Ursano, R., and Strain, J. (2011). A review of acute stress disorder in DSM-5. Depression and Anxiety, 28, 802-817. doi:10.1002/da.20737 ASD was introduced into DSM-IV to describe acute stress reactions that occur in the initial month after exposure to a traumatic event and prior to the possibility of diagnosing PTSD, and to identify trauma survivors in the acute phase who are high risk for PTSD. The review considers ASD in relation to other diagnostic approaches to acute stress responses, critiques the evidence of the predictive power of ASD, and discusses ASD in relation to Adjustment Disorder. The evidence suggests that ASD does not adequately identify most people who develop PTSD. This review presents a number of options and preliminary considerations to be considered for DSM-5. It is proposed that ASD be limited to describing severe acute stress reactions (that are not necessarily precursors of PTSD). The evidence suggests that the current emphasis on dissociation may be overly restrictive and does not recognize the heterogeneity of early posttraumatic stress responses. It is proposed that ASD may be better conceptualized as the severity of acute stress responses that does not require specific clusters to be present.
Bryant, R.A., Harvey, A.G., Dang, S.T., and Sackville, T. (1998). Assessing acute stress disorder: Psychometric properties of a structured clinical interview. Psychological Assessment, 10, 215-220. doi:10.1037//1040-3590.10.3.215 This study presents the development of a structured clinical interview to diagnose ASD. The Acute Stress Disorder Interview (ASDI) is a 19-item dichotomously scored interview schedule that is based on DSM-IV criteria. It was validated against clinician-based diagnoses of ASD on 65 trauma survivors assessed between 1 and 3 weeks posttrauma. It possessed good internal consistency (Cronbach’s α = 0.90), sensitivity (91%), and specificity (93%). Test-retest reliability was evaluated on 60 trauma survivors between 1 and 3 weeks posttrauma, with a re-administration interval of 2 to 7 days. Test-retest reliability of ASDI severity scores was strong (Cronbach’s α = 0.88), and diagnostic agreement for presence (88%) and absence (94%) of ASD diagnosis was high. The ASDI appears to be a useful tool to identify those individuals who suffer ASD and are at risk of long-term posttraumatic stress disorder.

Bryant, R.A., Mastrodomenico, J., Felmingham, K.L., Hopwood, S., Kenny, L., Kandris, E., et al. (2008). Treatment of acute stress disorder: A randomized controlled trial. Archives of General Psychiatry, 65, 659-667. doi:10.1001/archpsyc.65.6.659 ASD can identify recent trauma survivors who are likely to subsequently develop chronic PTSD. Cognitive behavior therapy for ASD may prevent PTSD. Trauma survivors may not tolerate exposure-based therapy in the acute phase. There is a need to compare nonexposure therapy techniques with prolonged exposure for ASD. To determine the relative efficacy of exposure therapy or trauma-focused cognitive restructuring in preventing chronic PTSD relative to a wait-list condition, a randomized controlled trial started of civilian trauma survivors (N = 90) attending an outpatient clinic between March 2002 and June 2006 who met criteria for ASD. Patients were randomly assigned to receive 5 weekly 90-minute sessions of either imaginal and in vivo exposure (n = 30), cognitive restructuring (n = 30), or a wait-list condition (n = 30). Measures were taken of PTSD at 6 months with a follow-up clinical interview, and self-report measures were accepted of PTSD, depression, anxiety, and trauma-related cognition patients. Intent-to-treat analyses indicated that at posttreatment, fewer patients in exposure had PTSD than those in cognitive restructuring or wait-list (33% vs. 63% vs. 77%, P = .002). At follow-up, patients in exposure were more likely to not meet diagnostic criteria for PTSD than those in cognitive restructuring (37% vs. 63%; odds ratio, 2.10; 95% confidence interval, 1.12-3.94, P = .05) and to achieve full remission (47% vs. 13%; odds ratio, 2.78; 95% confidence interval, 1.14-6.83, P = .005). On measures of PTSD, depression, and anxiety, exposure resulted in markedly larger effect sizes at posttreatment and follow-up than cognitive restructuring.

Bryant, R.A., Moulds, M., and Guthrie, R. (2000). Acute Stress Disorder Scale: A self-report measure of acute stress disorder. Psychological Assessment, 12, 61-68. doi:10.1037//1040-3590.12.1.61 This study presents the development of a self-report inventory to (a) index ASD and (b) predict subsequent development of PTSD. The Acute Stress Disorder Scale (ASDS) is a 19-item inventory that is based on DSM-IV criteria and is scored on a 5-point scale. It was validated against the ASDI on 99 civilian trauma survivors assessed between 2 and 10 days posttrauma. Using a formula to identify ASD caseness, the ASDS possessed good sensitivity (95%), and specificity (83%). Test-retest reliability was evaluated on 107 bushfire survivors 3 weeks posttrauma, with a re-administration interval of 2 to 7 days. Test-retest reliability of the ASDS scores was strong (Cronbach’s α = 0.94). Although the factor structure of the ASDS differed somewhat across the two samples, dissociative symptoms loaded on a separate factor from other acute stress symptoms in both samples. Predictive ability of the ASDS was investigated in 82 trauma survivors who completed the ASDS and were subsequently assessed for PTSD 6 months posttrauma. A cut-off score of 6 on the ASDS predicted 91% of those who developed PTSD and 93% of those who did not. One-third of those scoring above the cut-off did not develop PTSD, however. The ASDS shows initial promise as an initial screening instrument to identify acutely traumatized individuals who warrant more thorough assessment for risk of developing long-term PTSD.

Cardena, E., Koopman, C., Classen, C., Waelde, L.C., and Spiegel, D. (2000). Psychometric properties of the Stanford Acute Stress Reaction Questionnaire (SASRQ): A valid and reliable measure of acute stress. Journal of Traumatic Stress, 13, 719-734. doi:10.1023/A:1007822603186 A reliable and valid measure is needed for assessing the psychological symptoms experienced in the aftermath of a traumatic event. Previous research suggests that trauma victims typically experience dissociative, anxiety and other symptoms, during or shortly after a traumatic event. Although some of these symptoms may protect the trauma victim from pain, they may also lead to acute stress, posttraumatic stress, or other disorders. The Stanford Acute Stress Reaction Questionnaire (SASRQ) was developed to evaluate anxiety and dissociation symptoms in the aftermath of traumatic events, following DSM-IV criteria for acute stress disorder. We present data from multiple datasets and analyses supporting the reliability and construct, convergent, discriminant, and predictive validity of the SASRQ.

Harvey, A.G., and Bryant, R.A. (2002). Acute stress disorder: A synthesis and critique. Psychological Bulletin, 128, 886-902. doi:10.1033/0033-2909.128.6.886 The diagnosis of ASD was introduced to describe initial trauma reactions that predict chronic PTSD. This review outlines and critiques the rationales underpinning the ASD diagnosis and highlights conceptual and empirical problems inherent in this diagnosis. It is concluded that there is little justification for the ASD diagnosis in its present form. The evidence for and against the current emphasis on peritraumatic dissociation is discussed. The range of biological and cognitive mechanisms that potentially mediate acute trauma response are reviewed. The available evidence indicates that alternative means of conceptualizing acute trauma reactions and identifying acutely traumatized people who are at risk of developing PTSD need to be considered.

diagnosed in 13% of participants, and a further 20.7% suffered subclinical levels of ASD. At follow-up, 77.8% of ASD participants and 60% of subclinical ASD participants met criteria for PTSD. The strong predictive power of acute numbing, depersonalization, a sense of reliving the trauma and motor restlessness, which contrast to the low to moderate predictive power for other symptoms, indicate that only a subset of ASD symptoms are strongly related to the development of chronic PTSD. These findings support the utility of the ASD diagnosis but suggest that the dissociative and arousal clusters may require revision.

Spiegler, D., Koopman, C., Cardeña, E., and Classen, C. (1996). Dissociative symptoms in the diagnosis of acute stress disorder. In L.K. Michelson and W.J. Ray (Eds.), Handbook of dissociation: Empirical, theoretical, and clinical perspectives (pp. 367-380), New York: Plenum. After giving a brief description of ASD, where the essential component is the presence of dissociative symptomatology during or shortly after traumatic events, we review the following converging lines of evidence: (1) the conceptual and empirical association between PTSD and dissociation, (2) the evidence for the presence of dissociative responses during or shortly after trauma in a substantial percentage of the population, (3) the association between level of exposure to trauma and dissociative response, and (4) the association between peritraumatic dissociative responses and later full-fledged PTSD. We make the case that dissociative symptomatology is a frequent accompaniment of trauma that, if untreated, may lead to short- and long-term distress and malfunction. We analyze the dissociative and anxiety symptoms reported among respondents in the immediate aftermath of the 1991 Oakland/Berkeley fires.

### ADDITIONAL CITATIONS

Bryant, R.A., Creamer, M., O’Donnell, M.L., Silove, D., and McFarlane, A.C. (2008). A multisite study of the capacity of acute stress disorder diagnosis to predict posttraumatic stress disorder. *Journal of Clinical Psychiatry, 69*, 923-929. doi:10.4088/JCP.v69n0606 Objective: Previous studies investigating the relationship between ASD and PTSD have reported mixed findings and have been flawed by small sample sizes and single sites. This study addresses these limitations by conducting a large-scale and multisite study to evaluate the extent to which ASD predicts subsequent PTSD. Method: Between April 2004 and April 2005, patients admitted consecutively to four major trauma hospitals across Australia (N = 597) were randomly selected and assessed for ASD (DSM-IV criteria) during hospital admission (within 1 month of trauma exposure) and were subsequently reassessed for PTSD 3 months after the initial assessment (507). Results: Thirty-three patients (6%) met criteria for ASD, and 49 patients (10%) met criteria for PTSD at the 3-month follow-up assessment. Fifteen patients (45%) diagnosed with ASD and 34 patients (7%) not diagnosed with ASD subsequently met criteria for PTSD. The positive predictive power of PTSD criteria in the acute phase (0.60) was a better predictor of chronic PTSD than the positive predictive power of ASD (0.46). Conclusions: The majority of people who develop PTSD do not initially meet criteria for ASD. These data challenge the proposition that the ASD diagnosis is an adequate tool to predict chronic PTSD.

Bryant, R.A., O’Donnell, M.L., Creamer, M., McFarlane, A.C., and Silove, D. (in press). A multi-site analysis of the fluctuating course of posttraumatic stress disorder. *JAMA Psychiatry.* Context: Delayed-onset PTSD accounts for approximately 25% of PTSD cases. Current models do not adequately explain the delayed increases in PTSD symptoms following trauma exposure. Objective: To test the roles of initial psychiatric reactions, mild traumatic brain injury (MTBI) and ongoing stressors on delayed-onset posttraumatic stress. Design: Prospective design cohort study. Setting: Patients were drawn from recent admissions to 4 major trauma hospitals across Australia. Participants: 1,084 traumatically injured patients were assessed during hospital admission and followed up at 3 months, 12 months, and 24 months (N = 785; 72%) after injury. Main Outcome Measure: PTSD severity was assessed at each assessment with the Clinician-Administered PTSD Scale (CAPS). Results: Of those who met PTSD criteria at 24 months, 44% reported no PTSD at 3 months and 56% had subsyndromal or full PTSD. In those who displayed subsyndromal or full PTSD at 3 months, PTSD severity at 24 months was predicted by prior psychiatric disorder, initial PTSD symptom severity and type of injury. In those who displayed no PTSD at 3 months, PTSD severity at 24 months was predicted by initial PTSD symptom severity, MTBI, length of hospitalization, and the number of stressful events experienced between three and 24 months. Conclusions: These data highlight the complex trajectories of PTSD symptoms over time. This study also points to the roles of ongoing stress and MTBI in delayed cases of PTSD, and suggest the potential of ongoing stress compounding initial stress reactions and lead to a delayed increase in PTSD symptom severity. This study also provides initial evidence that MTBI increases risk for delayed PTSD symptoms, particularly in those with no acute symptoms.
subsequent PTSD over and above the other PTSD symptom severity at 6 months. Dissociative symptoms predicted persistent dissociation 4 weeks after the accident, predicted chronic symptoms.

6 months. Assessments included measures of dissociation, memory road traffic accident survivors were recruited. Patients were assessed prospectively the relationship between dissociative predictors, and chronic PTSD. Murray, J., Ehlers, A., and Mayou, R.A. (2002). PTSD: The relationship between acute stress disorder and PTSD in injured children. Journal of the American Academy of Child and Adolescent Psychiatry, 43, 403-411. doi:10.1097/00004583-200404000-00006 To examine the prevalence of ASD and PTSD in injured children and to evaluate the utility of ASD as a predictor of PTSD. Children hospitalized for injuries sustained in a traffic crash were enrolled in a prospective study. ASD was assessed in 243 children within 1 month after injury, and PTSD was assessed in 177 of these children 3 or more months after injury. The relationship between ASD and PTSD was examined via correlations between symptom severity scores and calculation of sensitivity, specificity, and positive and negative predictive values for categorical prediction of PTSD from ASD or subsets of ASD symptoms. Eight percent of children met the symptom criteria for ASD and another 14% had subsyndromal ASD; 6% met the symptom criteria for PTSD and another 11% had subsyndromal PTSD. ASD and PTSD symptom severity were associated. Sensitivity was low for prediction of child PTSD from child ASD. Subsyndromal ASD was a more effective predictor of PTSD. A substantial minority of injured children are affected by traumatic stress disorders. ASD in children may not be an optimal categorical predictor of PTSD. With increasing attention to early posttrauma services for children, empirically valid assessment/ triage models deserve further study.

Murray, J., Ehlers, A., and Mayou, R.A. (2002). Dissociation and post-traumatic stress disorder: Two prospective studies of road traffic accident survivors. British Journal of Psychiatry, 180, 363-368. doi:10.1192/bjp.180.4.363 Background: Dissociative symptoms during trauma predict PTSD, but they are often transient. It is controversial whether they predict chronic PTSD over and above what can be predicted from other post-trauma symptoms. Aims: To investigate prospectively the relationship between dissociative symptoms before, during and after a trauma and other psychological predictors, and chronic PTSD. Method: Two samples of 27 and 176 road traffic accident survivors were recruited. Patients were assessed shortly after the accident and followed at intervals over the next 6 months. Assessments included measures of dissociation, memory fragmentation, data-driven processing, rumination and PTSD symptoms. Results: All measures of dissociation, particularly persistent dissociation 4 weeks after the accident, predicted chronic PTSD severity at 6 months. Dissociative symptoms predicted subsequent PTSD over and above the other PTSD symptom clusters. Memory fragmentation and data-driven processing also predicted PTSD. Rumination about the accident was among the strongest predictors of subsequent PTSD symptoms.

Conclusions: Persistent dissociation and rumination 4 weeks after trauma are more useful in identifying those patients who are likely to develop chronic PTSD than initial reactions.

Shalev, A.Y., Ankri, Y., Israeli-Shalev, Y., Peleg, T., Adessky, R., and Freedman, S. (2012). Prevention of posttraumatic stress disorder by early treatment: Results from the Jerusalem Trauma Outreach and Prevention Study. Archives of General Psychiatry, 69, 166-176. doi:10.1001/archgenpsychiatry.2011.127 To compare early and delayed exposure-based, cognitive, and pharmacological interventions for preventing PTSD. Consecutively admitted survivors of traumatic events were assessed by use of structured telephone interviews a mean (SD) 9.61 (3.91) days after the traumatic event. Survivors with symptoms of ASD were referred for clinical assessment. Survivors who met PTSD symptom criteria during the clinical assessment were invited to receive treatment. Twelve weekly sessions of prolonged exposure (PE; n = 63), or cognitive therapy (CT; n = 40), or double blind treatment with two daily tablets of either escitalopram (10 milligrams) or placebo (selective serotonin reuptake inhibitor/ placebo; n = 46), or 12 weeks in a waiting list group (n = 93). Treatment started a mean (SD) 29.8 (5.7) days after the traumatic event. Waiting list participants with PTSD after 12 weeks received PE a mean (SD) 151.8 (42.4) days after the traumatic event (delayed PE). Proportion of participants with PTSD after treatment, as determined by the use of the CAPS were treated 5 and 9 months after the traumatic event. Treatment assignment and attendance were concealed from the clinicians who used the CAPS. At 5 months, 21.6% of participants who received PE and 57.1% of comparable participants on the waiting list had PTSD (odds ratio [OR], 0.21 [95% CI, 0.09-0.46]). At 5 months, 20.0% of participants who received CT and 58.7% of comparable participants on the waiting list had PTSD (OR, 0.18 [CI, 0.06-0.48]). The PE group did not differ from the CT group with regard to PTSD outcome (OR, 0.87 [95% CI, 0.29-2.62]). The PTSD prevalence rates did not differ between the escitalopram and placebo subgroups (61.9% vs. 55.6%; OR, 0.77 [95% CI, 0.21-2.77]). At 9 months, 20.8% of participants who received PE and 21.4% of participants on the waiting list had PTSD (OR, 1.04 [95% CI, 0.40-2.67]). Participants with partial PTSD before treatment onset did similarly well with and without treatment. Prolonged exposure, CT, and delayed PE effectively prevent chronic PTSD in recent survivors. The lack of improvement from treatment with escitalopram requires further evaluation. Trauma-focused clinical interventions have no added benefit to survivors with subthreshold PTSD symptoms.

van der Velden, P.G., Kleber, R.J., Christianse, B., Gersons, B.P.R., Marcelissen, F.G.H., Drogendijk, A.N., et al. (2006). The independent predictive value of peritraumatic dissociation for postdisaster intrusions, avoidance reactions, and PTSD symptom severity: A 4-year prospective study. Journal of Traumatic Stress, 19, 493-506. doi:10.1002/jts.20140 This 4-year prospective study (N = 662) of victims of a fireworks disaster examines the independent predictive value of peritraumatic dissociation for self-reported intrusions, avoidance reactions, and PTSD symptom severity at both 18 months (T2) and approximately 4 years postdisaster (T3). Peritraumatic dissociation was measured 2 to 3 weeks after the disaster (T1). Hierarchical multiple regression analyses revealed that peritraumatic dissociation was not a strong independent predictor for intrusions and avoidance reactions and PTSD symptom severity at T2 or T3 above initial intrusions, avoidance reactions, and psychological...
Results suggest that an early screening procedure for peritraumatic dissociation, which is aimed at identifying disaster victims who are at risk for long-term psychological disturbances can be omitted.