

A Comparison of the Capacity of *DSM-IV* and *DSM-5* Acute Stress Disorder Definitions to Predict Posttraumatic Stress Disorder and Related Disorders

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ABSTRACT

Objective: This study addresses the extent to which *DSM-IV* and *DSM-5* definitions of acute stress disorder (ASD) predict subsequent posttraumatic stress disorder (PTSD) and related psychiatric disorders following trauma.

Method: Patients with randomized admissions to 5 hospitals across Australia (N = 596) were assessed in hospital and reassessed for PTSD at 3 (n = 508), 12 (n = 426), 24 (n = 439), and 72 (n = 314) months using the Clinician-Administered PTSD Scale; *DSM-IV* definition of PTSD was used at each assessment, and *DSM-5* definition was used at 72 months. The Mini-International Neuropsychiatric Interview (MINI) was used at each assessment to assess anxiety, mood, and substance use disorders.

Results: Forty-five patients (8%) met *DSM-IV* criteria, and 80 patients (14%) met *DSM-5* criteria for ASD. PTSD was diagnosed in 93 patients (9%) at 3, 82 patients (10%) at 12, 100 patients (12%) at 24, and 26 patients (8%) at 72 months; 19 patients (6%) met *DSM-5* criteria for PTSD at 72 months. Comparable proportions of those diagnosed with ASD developed PTSD using *DSM-IV* (3 months = 46%, 12 months = 39%, 24 months = 32%, and 72 months = 25%) and *DSM-5* (43%, 42%, 33%, and 24%) ASD definitions. Sensitivity was improved for *DSM-5* relative to *DSM-IV* for depression (0.18 vs 0.30), panic disorder (0.19 vs 0.41), agoraphobia (0.14 vs 0.40), social phobia (0.12 vs 0.44), specific phobia (0.24 vs 0.58), obsessive-compulsive disorder (0.17 vs 0.47), and generalized anxiety disorder (0.20 vs 0.47). More than half of participants with *DSM-5*-defined ASD had a subsequent disorder.

Conclusions: The *DSM-5* criteria for ASD results in better identification of people who will subsequently develop PTSD or another psychiatric disorder relative to the *DSM-IV* criteria. Although prediction is modest, it suggests that the new ASD diagnosis can serve a useful function in acute trauma settings for triaging those who can benefit from either early intervention or subsequent monitoring.

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The acute stress disorder (ASD) diagnosis was introduced in *DSM-IV* to describe acute posttraumatic stress reactions that could occur in the month prior to when a potential diagnosis of posttraumatic stress disorder (PTSD) could be made.¹ The diagnosis served 2 purposes: to describe stress reactions in the initial month after a trauma (and thereby facilitate health care by providing patients with a diagnosis) and to identify recently trauma-exposed individuals who will subsequently develop PTSD.² The latter rationale was criticized by some commentators because it was not appropriate to have an independent diagnosis aiming to predict a similar diagnosis.³

Predicting longer term PTSD from acute stress response has traditionally been difficult because there is a well-documented trend for many initial stress responses to be transient and for most people to adapt in the following months.^{4–6} It was hoped in *DSM-IV* that ASD would be able to enhance prediction of subsequent PTSD by emphasizing particular symptoms. Consistent with evidence that dissociative responses after trauma are predictive of later PTSD^{7–9} and with the proposal that acute dissociation impairs processing of traumatic memories and adaptation of traumatic stress,¹⁰ considerable emphasis in ASD was placed on having dissociative responses during and after the trauma with the hope that prediction of PTSD would be enhanced. Specifically, ASD was defined in *DSM-IV* as occurring after a fearful response to experiencing or witnessing a threatening event (Cluster A) and required 3 dissociative symptoms (Cluster B), 1 reexperiencing symptom (Cluster C), marked avoidance (Cluster D), marked anxiety or increased arousal (Cluster E), and evidence of significant distress or impairment (Cluster F). The disturbance must last for a minimum of 2 days and a maximum of 4 weeks (Cluster G) (Table 1).

A systematic review of 22 longitudinal studies has shown that whereas some studies have found that the majority of trauma survivors who display ASD subsequently develop PTSD, other studies have found that only a minority of those with ASD subsequently develop PTSD¹¹; importantly, most people who develop PTSD do not initially experience ASD. For this reason, the approach was taken in *DSM-5* to restrict the goal of the ASD diagnosis to identifying people in the initial month after trauma exposure who are experiencing severe posttraumatic stress reactions with the explicit recognition that these reactions may be transient or may develop into chronic PTSD.¹² The *DSM-5* definition does not require satisfaction of dissociative, reexperiencing, avoidance, and arousal clusters but rather recognizes that acute stress responses can have marked heterogeneity; accordingly, *DSM-5* requires that at least 9 of a potential 14 symptoms be satisfied to meet criteria (Table 1).¹³ The diagnosis can now be made only after 3 days posttrauma rather than the 2 days stipulated in *DSM-IV*.

A question that remains is whether the new definition of ASD has utility as a predictor of subsequent PTSD. Although it is no longer

purportedly intended to discriminate between transient reactions and precursors to PTSD, there is potential clinical utility in understanding if the new diagnosis has better capacity to predict PTSD than its predecessor in *DSM-IV*. The potential benefit of being able to identify those in the acute phase who are likely to develop longer term PTSD is highlighted by the evidence that early interventions for people with ASD have been shown to be efficacious in reducing subsequent PTSD.^{14–16} There is a need to understand the relative strengths of the ASD diagnosis to describe acute stress reactions and to identify those who are at high risk for later PTSD because this will clarify the utility of the diagnosis in directing interventions to acute distress management or secondary prevention for longer term disorder. For example, if we could better identify people who would eventually develop PTSD, we could allocate mental health resources to either early intervention or monitoring to enhance mental health care. To this end, we report here a longitudinal multisite study of survivors of traumatic injury that assessed for ASD in the initial month after hospital admission and for PTSD at 3, 12, 24, and 72 months later. There is also evidence that ASD has some predictive capacity in identifying people who develop other psychiatric disorders following trauma,¹⁷ and accordingly we also assessed the relative strengths of the *DSM-IV* and *DSM-5* definitions of ASD to predict other affective and substance use disorders.

METHOD

Participants

Randomized admissions to 5 level-1 trauma centers across Australia were recruited into the study between April 2004 and April 2005. These patients formed the basis of previous studies involving prevalence of ASD and the longitudinal course of traumatically injured patients.^{18–20} The study was approved by the research and ethics committee at each hospital. Inclusion criteria included hospital admissions following traumatic injury who were between 18 and 70 years of age, could understand and speak English proficiently, and had a hospital admission of greater than 24 hours following traumatic injury. This last inclusion criterion was adopted because of the difficulty in locating and recruiting patients who remained in hospital for less than a day. Individuals were excluded from the study if they had moderate or severe head injury, were currently psychotic or suicidal, were non-Australian visitors, were cognitively impaired, or were under police guard. Individuals who met entry criteria were randomly selected using an automated, random assignment procedure stratified by length of stay. This approach was adopted to ensure that we did not differentially recruit patients who had

- The *DSM-5* definition of acute stress disorder results in better identification of recently trauma-exposed people who will develop subsequent posttraumatic stress disorder than the *DSM-IV* definition.
- Acute stress disorder in *DSM-IV* and *DSM-5* has modest predictive capacity for posttraumatic stress disorder.
- Over half of traumatically injured patients with acute stress disorder subsequently develop a psychiatric disorder.

Table 1. *DSM-IV* and *DSM-5* Diagnostic Criteria for Acute Stress Disorder

Criterion	<i>DSM-IV</i>	<i>DSM-5</i>
Stressor	<i>Both:</i> Threatening event Fear, helplessness, or horror	Threatening event
Dissociation	<i>Minimum 3 of:</i> Emotional numbing Reduced awareness Depersonalization Derealization Amnesia	<i>Minimum 9 of:</i> Intrusive distressing memories Recurrent distressing dreams Flashbacks Intense reactivity to reminders Emotional numbing Depersonalization/derealization Avoidance of memories/feelings Avoidance of external reminders Sleep disturbance Irritable behavior Hypervigilance Concentration deficits Elevated startle response
Reexperiencing	<i>Minimum 1 of:</i> Intrusive distressing memories Recurrent distressing dreams Flashbacks Intense reactivity to reminders	
Avoidance	<i>Marked avoidance of:</i> Memories, feelings, reminders	
Arousal	<i>Marked arousal, including:</i> Sleep disturbance Irritability Concentration deficits Hypervigilance Elevated startle response Motor restlessness	
Duration	At least 2 days and less than 1 month posttrauma	At least 3 days and less than 1 month posttrauma
Impairment	Impairs functioning	Impairs functioning

longer hospital stays because they may be more accessible. Of the 792 patients approached, 596 (75%) agreed to participate. Participants comprised 427 men and 169 women of the mean age of 37.74 years ($SD = 14.66$). A mild traumatic brain injury²¹ was experienced by 253 participants, and the mean Injury Severity Score (ISS²²) was 10.75 ($SD = 7.96$). Participants spent a mean of 12.33 ($SD = 12.82$) days in hospital. Seventy-five patients were admitted to intensive care units (ICUs). Types of injury included transport accidents ($n = 370$), falls ($n = 96$), assaults ($n = 31$), work-related accidents ($n = 45$), and other injuries ($n = 55$). Individuals who refused to participate in the current study did not differ from participants in terms of gender ($\chi^2_1 = 1.10$, $P = .13$), days in hospital ($t_{772} = 0.07$, $P = .67$), injury severity score ($t_{666} = 0.74$, $P = .34$), or presence of an ICU admission ($\chi^2_1 = 2.71$,

Table 2. Demographic and Clinical Characteristics at Time of Initial Assessment (N = 596)

Characteristic	Assessed (n = 314)	Dropped Out (n = 282)	P
Age, mean (SD), y	38.11 (14.63)	35.63 (14.73)	.14
Time since injury, mean (SD), d	7.39 (19.56)	6.72 (7.75)	.72
Injury Severity Score, total, mean (SD)	10.94 (8.04)	9.59 (7.40)	.17
ASD severity, ^a mean (SD)	5.21 (4.13)	6.40 (5.25)	.02
Type of injury			
Motor vehicle accident	62%	64%	.81
Assault	5%	5%	
Fall	16%	13%	
Industrial	8%	7%	
Other	9%	11%	
Gender, n (%)			
Male	226 (72)	200 (71)	.83
Female	88 (28)	82 (29)	
Educational level, n (%)			
High school	135 (43)	164 (58)	.06
Technical	69 (22)	39 (14)	
Tertiary	110 (35)	79 (28)	

^aMeasured by Acute Stress Disorder (ASD) Interview total score.

$P = .09$). Refusers were younger than participants ($t_{781} = 3.25$, $P = .001$).

In terms of those who participated in the study, 508 participants completed the 3-month assessment (representing 85% of the initial sample), 426 were assessed at 12 months (72% of the initial sample), 439 were assessed at 24 months (74% of the initial sample), and 314 were assessed at 72 months (53% of the initial sample). Patients at the 72-month assessment did not differ from those who did not participate in terms of gender ($\chi^2_1 = 2.60$, $P = .11$), mild traumatic brain injury ($\chi^2_1 = 0.96$, $P = .33$), length of hospital admission ($t_{1080} = 0.88$, $P = .38$), or ISS ($t_{1080} = 0.22$, $P = .83$). Those who were lost to follow-up were younger (36.08 years ± 13.57 vs 39.51 ± 13.47 ; $t_{1088} = 4.24$, $P = .001$) and had higher ASD severity scores than those who did participate (6.00 ± 4.79 vs 4.87 ± 3.81 ; $t_{1088} = 4.24$, $P = .001$) (Table 2).

Procedure

Following written informed consent, a trained clinician assessed for ASD based on symptoms present at the time of the assessment utilizing the Acute Stress Disorder Interview (ASDI).²³ The ASDI is a structured clinical interview that is based on *DSM-IV* criteria and possesses sound test-retest reliability ($r = 0.95$), sensitivity (92%), and specificity (93%) relative to independent clinician diagnosis. Consistent with the *DSM-IV* definition, assessments were initially commenced at 2 days after the injury and within the initial month; in the context of *DSM-5* altering the minimum timeframe for the ASD diagnosis, it is worth noting that 113 patients (18.9%) were assessed at 2 days posttrauma, and the remainder were assessed after this time. All assessments of PTSD were conducted using the Clinician-Administered PTSD Scale-IV (CAPS-IV²⁴) and were anchored to the traumatic injury that precipitated the hospitalization. The CAPS possesses good sensitivity (0.84) and specificity (0.95) relative to the Structured Clinical Interview for *DSM-IV* Disorders (SCID) PTSD diagnosis and also possesses sound

test-retest reliability (0.90–0.98). In terms of assessing PTSD, the *DSM-IV* criteria were used for the 3-, 12-, and 24-month assessments via telephone structured clinical interviews, and both *DSM-IV* and *DSM-5* criteria²⁵ were used for the 72-month assessment (because *DSM-5* criteria were not available at the times of the earlier assessments); a modified version of the CAPS for *DSM-5*²⁶ developed by the CAPS' authors was used at the 72-month assessment. The ASD criteria for *DSM-5* could be calculated for each time point because, whereas the derivation of the diagnosis has changed in *DSM-5*, the specific items that constitute the diagnosis have remained the same. Five percent of all CAPS interviews were rescored blind to the original scoring to test interrater reliability. Overall, the PTSD diagnostic consistency for the CAPS ranged from 0.98 to 1.00 across the assessments.

To index other affective and substance use disorders, we administered the Mini-International Neuropsychiatric Interview (version 5.5; MINI)²⁷ at each assessment. The MINI is a short, structured diagnostic interview based on the *DSM-IV* and the *ICD-10* classification of mental illness. We used the MINI to identify major depressive episode, panic disorder, agoraphobia, social phobia, specific phobia, obsessive-compulsive disorder, generalized anxiety disorder, alcohol abuse and dependence, and marijuana abuse and dependence.

Data Analysis

To determine diagnostic incidence at each time point, and accordingly derive predictive capacity of ASD, we based all analyses on data that were collected at each time point rather than imputing missing values. We calculated sensitivity (defined as the probability that someone diagnosed with a subsequent disorder initially had ASD), specificity (the probability that someone without a subsequent psychiatric disorder did not initially have ASD), positive predictive power (the probability that someone with ASD developed a subsequent psychiatric disorder), and negative predictive power (the probability that someone without ASD did not develop a psychiatric disorder).

RESULTS

Incidence of ASD and Psychiatric Disorders

At the initial assessment, 45 patients (8%) met *DSM-IV* criteria for ASD and 80 patients (14%) met *DSM-5* criteria for ASD (an additional 11 patients met *DSM-5* criteria for ASD based on a 2-day minimum requirement). In terms of the impact of removing the subjective response to the traumatic response from the stressor criterion in *DSM-5* (ie, the A2 criterion), 7 patients (9%) would not have received the ASD diagnosis if the subjective requirement of fear, horror, or helplessness was required. At 3 months posttrauma, 93 patients (9%) met criteria for PTSD and 288 patients (29%) met criteria for any disorder. At 12 months posttrauma, 82 patients (10%) met criteria for PTSD and 278 patients (34%) met criteria for any disorder. Patients who satisfied the *DSM-5* criteria for ASD did not differ from those who met the *DSM-IV* criteria on any demographic, injury-related, or

Table 3. Frequency, Sensitivity, Specificity, and Positive and Negative Predictive Power of DSM-IV and DSM-5 Acute Stress Disorder (ASD) Predicting Posttraumatic Stress Disorder (PTSD)^a

Follow-Up Assessment	PTSD Incidence, n (%)	ASD DSM-IV				ASD DSM-5			
		Sensitivity	Specificity	Positive Predictive Power	Negative Predictive Power	Sensitivity	Specificity	Positive Predictive Power	Negative Predictive Power
3 Months	49 (10)	0.31	0.96	0.46	0.93	0.51	0.93	0.43	0.95
12 Months	43 (10)	0.26	0.96	0.39	0.92	0.45	0.93	0.42	0.94
24 Months	51 (12)	0.18	0.95	0.32	0.90	0.33	0.92	0.33	0.92
72 Months (DSM-IV)	26 (8)	0.19	0.95	0.25	0.93	0.27	0.92	0.24	0.93
72 Months (DSM-5)	19 (6)	0.19	0.95	0.25	0.93	0.26	0.92	0.17	0.95

^aSensitivity = probability that someone diagnosed with PTSD initially had ASD. Specificity = probability that someone without PTSD did not initially have ASD. Positive predictive power = probability that someone with ASD develops PTSD. Negative predictive power = probability that someone without ASD does not develop PTSD.

Table 4. Frequency, Sensitivity, Specificity, and Positive and Negative Predictive Power of DSM-IV and DSM-5 Acute Stress Disorder (ASD) Predicting Affective or Substance Use Disorders at 12 Months^a

Diagnosis	Incidence, n (%)	ASD DSM-IV				ASD DSM-5			
		Sensitivity	Specificity	Positive Predictive Power	Negative Predictive Power	Sensitivity	Specificity	Positive Predictive Power	Negative Predictive Power
Major depression	77 (18)	0.18	0.96	0.48	0.84	0.30	0.92	0.48	0.86
Panic disorder	27 (6)	0.19	0.94	0.17	0.94	0.41	0.91	0.23	0.96
Agoraphobia	49 (12)	0.14	0.94	0.24	0.90	0.40	0.92	0.40	0.92
Social phobia	33 (8)	0.12	0.94	0.14	0.93	0.44	0.91	0.29	0.95
Specific phobia	25 (6)	0.24	0.94	0.21	0.95	0.58	0.92	0.29	0.97
OCD	18 (4)	0.17	0.94	0.10	0.96	0.47	0.90	0.17	0.98
GAD	49 (12)	0.20	0.95	0.35	0.90	0.47	0.93	0.48	0.93
Substance use disorders	41 (10)	0.02	0.93	0.03	0.90	0.15	0.89	0.13	0.91

^aSensitivity = probability that someone diagnosed with affective and substance use disorders initially had ASD. Specificity = probability that someone without a psychiatric disorder did not initially have ASD. Positive predictive power = probability that someone with ASD develops a psychiatric disorder. Negative predictive power = probability that someone without ASD does not develop a psychiatric disorder. Abbreviations: GAD = generalized anxiety disorder, OCD = obsessive-compulsive disorder.

Table 5. Frequency, Sensitivity, Specificity, and Positive and Negative Predictive Power of DSM-IV and DSM-5 Acute Stress Disorder (ASD) Predicting Any Posttraumatic Stress Disorder (PTSD), Affective, or Substance Use Disorders^a

Follow-Up Assessment	Any Diagnosis Incidence, n (%)	ASD DSM-IV				ASD DSM-5			
		Sensitivity	Specificity	Positive Predictive Power	Negative Predictive Power	Sensitivity	Specificity	Positive Predictive Power	Negative Predictive Power
3 Months	147 (29)	0.14	0.96	0.61	0.73	0.25	0.94	0.64	0.75
12 Months	140 (33)	0.14	0.97	0.71	0.70	0.24	0.96	0.76	0.72
24 Months	141 (32)	0.10	0.95	0.50	0.69	0.20	0.93	0.58	0.71
72 Months (DSM-IV)	90 (29)	0.10	0.95	0.45	0.72	0.13	0.92	0.41	0.72

^aSensitivity = probability that someone diagnosed with affective and substance abuse disorders initially had ASD. Specificity = probability that someone without a psychiatric disorder did not initially have ASD. Positive predictive power = probability that someone with ASD develops a psychiatric disorder. Negative predictive power = probability that someone without ASD does not develop a psychiatric disorder.

acute stress severity factors. At 24 months posttrauma, 100 patients (12%) met criteria for PTSD and 273 patients (34%) met criteria for any disorder. At 72 months posttrauma, 26 patients (8%) met DSM-IV criteria for PTSD, 19 patients (6%) met DSM-5 criteria for PTSD, and 91 patients (29%) met criteria for any disorder.

Relationship Between ASD and PTSD

In terms of those diagnosed with ASD according to the DSM-IV criteria, the minority of participants subsequently met criteria for PTSD (range, 25%–46%) (Table 3). Comparable positive predictive power was observed for the DSM-5 criteria. In terms of those diagnosed with PTSD at each assessment, only a small proportion met DSM-IV criteria initially (range, 19%–31%). In contrast, the DSM-5

criteria resulted in markedly higher rates of participants who eventually developed PTSD being identified with ASD initially (range, 27%–53%). The prediction of PTSD at 72 months was much weaker for both DSM-IV and DSM-5 criteria for ASD, regardless of whether DSM-IV or DSM-5 criteria were employed. The negative predictive power for both DSM-IV and DSM-5 ASD definitions was consistently high, suggesting that the absence of an ASD diagnosis is strongly predictive of not subsequently developing PTSD.

Relationship Between ASD and Affective and Substance Use Disorders

Table 4 presents the specific relationships between ASD and each psychiatric disorder at 12 months. The sensitivity for the DSM-IV diagnosis was very poor across diagnoses,

however, it markedly increased for all disorders when the *DSM-5* definition was applied. For example, sensitivity increased for depression (0.18 vs 0.30), panic disorder (0.19 vs 0.41), agoraphobia (0.14 vs 0.40), social phobia (0.12 vs 0.44), specific phobia (0.24 vs 0.58), obsessive-compulsive disorder (0.17 vs 0.47), and generalized anxiety disorder (0.20 vs 0.47).

Table 5 presents the capacity of ASD to predict the presence of any affective or substance use disorder at each time point. In terms of those diagnosed with ASD according to the *DSM-IV* criteria, at least half of participants subsequently met criteria for any disorder (range, 45%–71%). Comparable predictive power was observed for the *DSM-5* criteria (range, 41%–76%). In terms of those diagnosed with any disorder at each assessment, approximately one-tenth of participants met *DSM-IV* ASD criteria initially (range, 10%–14%). The *DSM-5* criteria led to higher rates of participants who eventually developed any disorder being identified with ASD initially (range, 13%–25%).

DISCUSSION

More people met criteria for ASD using the *DSM-5* criteria (14%) than the *DSM-IV* criteria (8%). This is not surprising considering that the criteria were intentionally broadened to remove the restriction of having to meet criteria for all the clusters. The increase in incidence suggests that the new diagnosis is meeting the need to identify more of those trauma survivors who are experiencing marked acute stress reactions. The finding that the rate increased only 6% suggests that the increase is reasonable, and the *DSM-5* diagnosis does not appear to be overidentifying cases.

The primary purpose of this study, however, was to index the predictive power of the different definitions of ASD. The finding that comparable rates of people with *DSM-IV* and *DSM-5* ASD subsequently developed PTSD at 3 (46% vs 43%), 12 (39% vs 42%), 24 (32% vs 33%), and 72 (25% vs 24%) months posttrauma suggests that the modification of the criteria has not radically altered the positive predictive power of the diagnosis. That is, the ASD diagnosis falls short in achieving strong screening capacity insofar as the minority of people with ASD develop chronic PTSD. In contrast, of those who eventually developed PTSD, nearly twice the proportion developed PTSD using the *DSM-5* criteria than the *DSM-IV* criteria at 3 (31% vs 51%), 12 (26% vs 45%), and 24 (18% vs 33%) months. Again, this may not be surprising because the more relaxed criteria in *DSM-5* resulted in more people being eligible for the diagnosis and permits more people who may be high risk for PTSD to meet the ASD criteria. This finding is consistent with previous reports that subsyndromal ASD, defined as meeting only 3 of the 4 symptom clusters in *DSM-IV*, improved prediction of PTSD in previous longitudinal studies.^{20,28–30}

Although the *DSM-5* ASD criteria did result in better sensitivity in predicting PTSD than the *DSM-IV* criteria, it should be noted that both the *DSM-IV* and *DSM-5* formulae resulted in only modest prediction of PTSD. Although there is a demonstrated relationship between a number of

acute psychological (eg, peritraumatic dissociation, anxiety sensitivity^{31,32}) and biological (eg, heart rate³³) variables, the overall poor capacity to predict longer term PTSD can probably be attributed to the increasingly well-documented finding that there is not a linear relationship between acute stress response and longer term PTSD; it appears that there is a fluctuating course of posttraumatic stress in the months and years after trauma exposure.¹⁹ Further, there is not a single trajectory of response as studies that have employed latent growth mixture modeling approaches have mapped a variety of courses that may be characterized as chronic distress, worsening stress, recovery, or consistently without stress symptoms.^{34–36} It appears that a range of factors can impact on the subsequent development of PTSD that may be independent of acute stress response, including stressors that occur subsequent to the initial trauma,^{19,37,38} maladaptive coping behaviors,³⁹ and appraisals that exacerbate the stress response.⁴⁰ In this context, it is apparent that any attempt to predict chronic PTSD from the acute stress response will be limited because these subsequent factors cannot necessarily be predicted in the acute phase. This appears to be the case particularly in prediction of longer term PTSD; the predictive ability of initial ASD in relation to subsequent PTSD appears to have become weaker as the follow-up period was extended. This is consistent with the conclusion that as more time elapses since the initial trauma exposure, the greater influence factors other than initial response may have on PTSD severity. It is important to note that we have previously reported from this data set (up to the 24-month assessment) that there is great instability in terms of diagnostic status across time.¹⁹ That is, approximately half of patients who report PTSD at 1 assessment do not report it at the subsequent assessment, and this pattern changes again at the following assessment. This volatility raises serious challenges for predicting PTSD status at any time because the individuals who are being identified are not necessarily the same people at 2 different time frames.

Whereas the *DSM-IV* and *DSM-5* criteria had comparable positive predictive power for other affective and substance use disorders, the *DSM-5* had markedly stronger sensitivity for the 3-, 12-, and 24-month assessments. Up to 24 months, the *DSM-5* criteria for ASD was identifying between 20%–25% of people who developed some psychiatric disorder (depending on the assessment point). We note that a proportion of these cases were likely to be reactivations of preexisting disorders. It is to be expected that the broader definition of ASD in *DSM-5* would capture more people in the acute phase who subsequently develop a disorder because it is less prescriptive. This has potential ramifications for identification of people in the acute phase who may be more likely to develop a subsequent disorder; more than half of people with ASD in hospital were likely to develop a subsequent disorder. Although using ASD as an early marker will not necessarily allow prediction of a subsequent specific disorder, it could have potential to alert clinicians to focus follow-up assessments on those individuals because they are at higher risk of needing mental health intervention for

some disorder subsequent to their injury. In concluding that ASD may function as a predictor of subsequent psychopathology, however, it also needs to be recognized that this form of screening will also miss the majority of people who will develop a subsequent disorder, and hence, it is by no means sufficient as a tool for general primary care screening. It is interesting that the predictive power for different disorders at 12 months was not substantively different between most disorders. Two exceptions were apparent to this pattern. Positive predictive power tended to be stronger for depression than any other anxiety disorder; this may have occurred because of subsequent comorbid PTSD and depression rather than the capacity to predict depression in its own right. The other exception was substance abuse, which was poorly predicted by ASD; this may be attributed to the distinctive symptom clusters of substance use disorder and to the role of preexisting substance use.

The conclusions about the predictive abilities of ASD for both *DSM-IV* and *DSM-5* definitions need to be qualified by the timeframe of the follow-up assessments. Tables 3 and 4 indicate that sensitivity for ASD predicting PTSD gradually reduces at each assessment, and it is most clearly exemplified at the 72-month follow-up at which point the sensitivity was poor. As time elapses after trauma exposure, it is increasingly less likely that the precipitating trauma will have less impact on subsequent PTSD and other life events will have a stronger impact. Longitudinal studies indicate that long-term trajectories of trauma response have very modest relationships with initial stress reactions partly because ongoing stressors negatively impact on the recovery course of trauma survivors.¹¹

These findings have implications for those working in acute settings. One of the major reasons for identifying people in the acute phase after trauma is so that we can provide early intervention to those who are likely to develop PTSD. There is good evidence that cognitive-behavioral therapy for ASD does limit subsequent PTSD^{14,41}; however, we do not want to be providing this to all trauma survivors. Resources demand that we can provide this form of intervention only to those who are less likely to remit in the following months, and so the new ASD definition does appear to provide some better guidance to clinicians regarding who is at high risk for PTSD and may benefit from early therapy than the *DSM-IV* criteria. Moreover, the observation of strong negative predictive power of ASD predicting absence of psychiatric morbidity suggests that the absence of an ASD diagnosis allows us to identify with moderate confidence those trauma survivors in the acute trauma phase who will not require subsequent mental health intervention. In the context of limited resources, this can allow triaging of trauma survivors and makes it more realistic to either provide early intervention or conduct follow-up assessments to determine the mental health needs of the proportion that are identified as being at greater risk.

We recognize several limitations. First, all participants suffered traumatic injury primarily from motor vehicle accidents; PTSD can vary across trauma populations, and

in the context of ASD, it is worth noting that severe and interpersonal trauma is more likely to lead to dissociative symptoms^{42,43}; accordingly, the current findings may not apply to other trauma populations. Second, by assessing ASD in hospital, we may have indexed symptoms in a relatively protective environment, which may not reflect the function of ASD reactions in more naturalistic contexts. Further, by assessing the people approximately 1 week after trauma, the results may not be applicable to more acute trauma settings. Although follow-up assessments were conducted via telephone, telephone and face-to-face interviews can result in comparable responses.⁴⁴ We assessed PTSD at the 3-, 12-, and 24-month assessments using the *DSM-IV* criteria only because the *DSM-5* PTSD definition was not available at the time of these assessments; it is possible that different results may emerge when predicting the *DSM-5* definition of PTSD. Relatedly, although the *DSM-5* retained the same symptoms as *DSM-IV*, the wording of some items was altered (eg, irritability was altered to irritable behavior), and this may have affected the comparability between definitions. We were not able to reliably assess mental health provision throughout the study, so we do not know the potential impact of treatment on predictive function. We also lacked sufficient sample size to detect the impact of comorbidity on results. Finally, there was considerable attrition over the 6 years of the study, and we note that participants with more severe ASD were more likely to drop out; this differential dropout rate may have impacted the predictive findings.

In summary, these results demonstrate that the revised ASD definition in *DSM-5* performs better than the *DSM-IV* version insofar as it is less limiting and also captures more people who subsequently develop PTSD. Although not the intended purpose of the ASD diagnosis in *DSM-5*, it does provide an opportunity to identify with greater confidence those who may develop mental health problems. It should be noted, however, that we should not be relying on the ASD diagnosis as the optimal means of prediction of subsequent PTSD and we need to develop better monitoring processes to identify those in need of mental health interventions following trauma.

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REFERENCES

1. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition. Washington, DC: American Psychiatric Association; 1994.
2. Harvey AG, Bryant RA. Acute stress disorder: a synthesis and critique. *Psychol Bull.* 2002;128(6):886–902.
3. Bryant RA, Harvey AG. New *DSM-IV* diagnosis of acute stress disorder. *Am J Psychiatry.* 2000;157(11):1889–1891.
4. Galea S, Vlahov D, Resnick H, et al. Trends of probable post-traumatic stress

- disorder in New York City after the September 11 terrorist attacks. *Am J Epidemiol*. 2003;158(6):514–524.
5. Rothbaum BO, Foa EB, Riggs DS, et al. A prospective examination of post-traumatic stress disorder in rape victims. *J Trauma Stress*. 1992;5(3):455–475.
 6. Riggs DS, Rothbaum BO, Foa EB. A prospective examination of symptoms of posttraumatic stress disorder in victims of nonsexual assault. *J Interpers Violence*. 1995;10(2):201–214.
 7. Murray J, Ehlers A, Mayou RA. Dissociation and post-traumatic stress disorder: two prospective studies of road traffic accident survivors. *Br J Psychiatry*. 2002;180(4):363–368.
 8. Koopman C, Classen C, Spiegel DA. Predictors of posttraumatic stress symptoms among survivors of the Oakland/Berkeley, Calif., firestorm. *Am J Psychiatry*. 1994;151(6):888–894.
 9. Shalev AY, Freedman S, Peri T, et al. Predicting PTSD in trauma survivors: prospective evaluation of self-report and clinician-administered instruments. *Br J Psychiatry*. 1997;170(6):558–564.
 10. Spiegel D, Koopman C, Cardena C, et al. Dissociative symptoms in the diagnosis of acute stress disorder. In: Michelson LK, Ray WJ, eds. *Handbook of Dissociation*. New York, NY: Plenum Press; 1996:367–380.
 11. Bryant RA. Acute stress disorder as a predictor of posttraumatic stress disorder: a systematic review. *J Clin Psychiatry*. 2011;72(2):233–239.
 12. Bryant RA, Friedman MJ, Spiegel D, et al. A review of acute stress disorder in DSM-5. *Depress Anxiety*. 2011;28(9):802–817.
 13. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. Fifth Edition. Washington, DC: American Psychiatric Association; 2013.
 14. Bryant RA, Mastrodomenico J, Felmingham KL, et al. Treatment of acute stress disorder: a randomized controlled trial. *Arch Gen Psychiatry*. 2008;65(6):659–667.
 15. Bryant RA, Sackville T, Dang ST, et al. Treating acute stress disorder: an evaluation of cognitive behavior therapy and supportive counseling techniques. *Am J Psychiatry*. 1999;156(11):1780–1786.
 16. Bisson JJ, Shepherd JP, Joy D, et al. Early cognitive-behavioural therapy for post-traumatic stress symptoms after physical injury: randomised controlled trial. *Br J Psychiatry*. 2004;184(1):63–69.
 17. Bryant RA, Creamer M, O'Donnell M, et al. The capacity of acute stress disorder to predict posttraumatic psychiatric disorders. *J Psychiatr Res*. 2012;46(2):168–173.
 18. Bryant RA, O'Donnell ML, Creamer M, et al. The psychiatric sequelae of traumatic injury. *Am J Psychiatry*. 2010;167(3):312–320.
 19. Bryant RA, O'Donnell ML, Creamer M, et al. A multisite analysis of the fluctuating course of posttraumatic stress disorder. *JAMA Psychiatry*. 2013;70(8):839–846.
 20. Bryant RA, Creamer M, O'Donnell ML, et al. A multisite study of the capacity of acute stress disorder diagnosis to predict posttraumatic stress disorder. *J Clin Psychiatry*. 2008;69(6):923–929.
 21. American Congress of Rehabilitation Medicine. Definition of mild traumatic brain injury. *J Head Trauma Rehabil*. 1993;8(3):86–87.
 22. American Association for Automotive Medicine. *The Abbreviated Injury Scale 1990-Revision*. Des Plaines, IL: American Association for Automotive Medicine; 1990.
 23. Bryant RA, Harvey AG, Dang ST, et al. Assessing acute stress disorder: psychometric properties of a structured clinical interview. *Psychol Assess*. 1998;10(3):215–220.
 24. Blake DD, Weathers FW, Nagy LM, et al. The development of a clinician administered PTSD scale. *J Trauma Stress*. 1995;8(1):75–90.
 25. Friedman MJ, Resick PA, Bryant RA, et al. Considering PTSD for DSM-5. *Depress Anxiety*. 2011;28(9):750–769.
 26. Weathers FW, Blake DD, Schnurr PP, et al. (2013). The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5). Interview available from the National Center for PTSD at www.ptsd.va.gov.
 27. Sheehan DV, Lecrubier Y, Sheehan KH, et al. The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *J Clin Psychiatry*. 1998;59(suppl 20):22–33, quiz 34–57.
 28. Harvey AG, Bryant RA. The relationship between acute stress disorder and posttraumatic stress disorder: a prospective evaluation of motor vehicle accident survivors. *J Consult Clin Psychol*. 1998;66(3):507–512.
 29. Bryant RA, Harvey AG. Relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury. *Am J Psychiatry*. 1998;155(5):625–629.
 30. Dalgleish T, Meiser-Stedman R, Kassam-Adams N, et al. Predictive validity of acute stress disorder in children and adolescents. *Br J Psychiatry*. 2008;192(5):392–393.
 31. Marshall GN, Miles JN, Stewart SH. Anxiety sensitivity and PTSD symptom severity are reciprocally related: evidence from a longitudinal study of physical trauma survivors. *J Abnorm Psychol*. 2010;119(1):143–150.
 32. Marshall GN, Schell TL. Reappraising the link between peritraumatic dissociation and PTSD symptom severity: evidence from a longitudinal study of community violence survivors. *J Abnorm Psychol*. 2002;111(4):626–636.
 33. Bryant RA, Creamer M, O'Donnell M, et al. A multisite study of initial respiration rate and heart rate as predictors of posttraumatic stress disorder. *J Clin Psychiatry*. 2008;69(11):1694–1701.
 34. Bonanno GA, Mancini AD, Horton JL, et al; Millennium Cohort Study Team. Trajectories of trauma symptoms and resilience in deployed US military service members: prospective cohort study. *Br J Psychiatry*. 2012;200(4):317–323.
 35. Lam WW, Bonanno GA, Mancini AD, et al. Trajectories of psychological distress among Chinese women diagnosed with breast cancer. *Psychooncology*. 2010;19(10):1044–1051.
 36. deRoos-Cassini TA, Mancini AD, Rusch MD, et al. Psychopathology and resilience following traumatic injury: a latent growth mixture model analysis. *Rehabil Psychol*. 2010;55(1):1–11.
 37. Solomon Z, Kotler M, Shalev A, et al. Delayed onset PTSD among Israeli veterans of the 1982 Lebanon War. *Psychiatry*. 1989;52(4):428–436.
 38. Green BL, Lindy JD, Grace MC, et al. Buffalo Creek survivors in the second decade: stability of stress symptoms. *Am J Orthopsychiatry*. 1990;60(1):43–54.
 39. Guthrie R, Bryant R. Attempting suppression of traumatic memories over extended periods in acute stress disorder. *Behav Res Ther*. 2000;38(9):899–907.
 40. 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.
 41. Bryant RA, Harvey AG, Dang ST, et al. Treatment of acute stress disorder: a comparison of cognitive-behavioral therapy and supportive counseling. *J Consult Clin Psychol*. 1998;66(5):862–866.
 42. Zatzick DF, Marmar CR, Weiss DS, et al. Does trauma-linked dissociation vary across ethnic groups? *J Nerv Ment Dis*. 1994;182(10):576–582.
 43. Marmar CR, Weiss DS, Schlenger WE, et al. Peritraumatic dissociation and posttraumatic stress in male Vietnam theater veterans. *Am J Psychiatry*. 1994;151(6):902–907.
 44. Aziz MA, Kenford S. Comparability of telephone and face-to-face interviews in assessing patients with posttraumatic stress disorder. *J Psychiatr Pract*. 2004;10(5):307–313.