again. Such highly idiosyncratic, excessively negative appraisals distinguish well between trauma survivors with and without PTSD currently (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999), as well as between survivors who are and are not likely to develop PTSD in the future. For example, Dunmore et al. (2001) found that negative appraisals assessed within 4 months posttrauma predicted PTSD symptom severity among assault survivors 6 months and 9 months posttrauma.

Finally, not only does appraisal of the stressor affect its pathogenic impact, but appraisal of acute stress symptoms themselves may influence whether chronic PTSD develops (Ehlers & Clark, 2000). Catastrophic appraisal of symptoms as harbingers of impending psychosis or indicants of moral weakness increase risk for PTSD (e.g., Dunmore et al., 2001; Ehlers et al., 1998). For example, appraisal of intrusive thoughts as meaning that one is about to lose one's mind, rather than as a temporary and expectable response to trauma, may foster attempts to suppress intrusive thoughts, which in turn may lead to a paradoxical increase in their frequency. As this example shows, excessively negative appraisals of the trauma and its consequences motivate trauma survivors to engage in behaviors that maintain the problem. Likewise, some safety behaviors are strong predictors of PTSD. These behaviors include taking excessive precautions (e.g., Dunmore et al., 2001), excessively avoiding trauma reminders (e.g., Harvey & Bryant, 1998a), and ruminating about the trauma and its effects on one's life (e.g., Murray et al., 2002).

ACUTE STRESS DISORDER

The diagnosis acute stress disorder (ASD) made its first appearance in DSM-IV. ASD arises from the same set of traumatic stressors deemed capable of causing PTSD, and is characterized by many of the same symptoms (see Table 2). According to DSM-IV, ASD can occur after exposure to a threatening event, and is diagnosed if the individual exhibits at least three dissociative symptoms, one reexperiencing symptom, marked avoidance, and marked hyperarousal. ASD differs from PTSD in two critical ways. First, the disturbance must last for a minimum of 2 days and a maximum of 4 weeks (after which time a diagnosis of PTSD could be made). Second, the ASD criteria emphasize dissociative reactions. According to DSM-IV, the diagnosis of ASD requires the presence of at least three of the following dissociative symptoms: a sense of emotional numbing or detachment, reduced awareness of one's surroundings, derealization, depersonalization, and amnesia for aspects of the traumatic event. In contrast, the PTSD criteria do not require the individual to display dissociative symptoms.

ASD advocates advanced three arguments for including the disorder in DSM-IV. First, although a diagnosis of PTSD required 1 month of persistent symptoms (so that transient stress reactions would not be classified as pathological), it is inhumane to make highly distressed survivors wait 1 month before diagnosing and treating a severe stress reaction. Indeed, doctors would not wait 1 month to diagnose and treat a broken

Table 2. *Diagnostic criteria for acute stress disorder*

A. The person has been exposed to a traumatic event in which both of the following were present:
   (1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
   (2) the person's response involved intense fear, helplessness, or horror
B. Either while experiencing or after experiencing the distressing event, the individual has three (or more) of the following dissociative symptoms:
   (1) a subjective sense of numbing, detachment, or absence of emotional responsiveness
   (2) a reduction in awareness of his or her surroundings (e.g., "being in a daze")
   (3) derealization
   (4) depersonalization
   (5) dissociative amnesia (i.e., inability to recall an important aspect of the trauma)
C. The traumatic event is persistently reexperienced in at least one of the following ways: recurrent images, thoughts, dreams, illusions, flashback episodes, or a sense of reliving the experience; or distress on exposure to reminders of the traumatic event.
D. Marked avoidance of stimuli that arouse recollections of the trauma (e.g., thoughts, feelings, conversations, activities, places, people).
E. Marked symptoms of anxiety or increased arousal (e.g., difficulty sleeping, irritability, poor concentration, hypervigilance, exaggerated startle response, motor restlessness).
F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or impairs the individual's ability to pursue some necessary task, such as obtaining necessary assistance or mobilizing personal resources by telling family members about the traumatic experience.
G. The disturbance lasts for a minimum of 2 days and a maximum of 4 weeks and occurs within weeks of the traumatic event.
H. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition, is not better accounted for by Brief Psychotic Disorder, and is not merely an exacerbation of a preexisting Axis I or Axis II disorder.

Note. From *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, by the American Psychiatric Association, 1994, pp. 431–432. Copyright 1994 by the American Psychiatric Association. Reprinted with permission of the author.

arm. Second, an ASD diagnosis may predict subsequent PTSD. Hence, early diagnosis and treatment of ASD may reduce the likelihood of later chronic pathology. Third, a benefit of including ASD would be to stimulate research on acute stress reactions.

Although the inclusion of ASD in DSM-IV has, indeed, stimulated much research, the validity of the diagnosis, and especially its emphasis on dissociation, has been seriously questioned (for reviews, see Bryant & Harvey, 2000a; Harvey & Bryant, 2002; R.D. Marshall, Spitzer, & Liebowitz, 1999). The emphasis on dissociative responses rests on the belief that dissociation reflects pathological cognitive avoidance that impedes emotional processing and recovery from trauma (see van der Kolk & van der Hart, 1989). However, a conflicting view posits that dissociation during a traumatic experience may serve a protective function by attenuating the emotional impact of trauma (Horowitz, 1986; Noyes & Kletti, 1977). Critics also worry that the ASD diagnosis amounts to inappropriate classification of a normative human response to overwhelming trauma as a medical disorder. According to this critique, the reactions embodied in the ASD criteria do not arise from underlying psychobiological dysfunction and therefore do not reflect mental illness, but rather arise from the expectable workings of evolved cognitive and emotional mechanisms for responding to trauma (see Wakefield, 1992, 1996).

The ASD diagnosis not only has these conceptual problems, but also has a weak empirical foundation, at best. Indeed, the diagnosis was included in DSM-IV without having undergone the empirical scrutiny required of other candidate diagnoses (Bryant, 2000). Even its advocates acknowledged that the predictive relation between ASD and PTSD was "based more on logical arguments than on empirical research" (Koopman, Classen, Cardeña, & Spiegel, 1995, p. 38). (The notion that something ought to be elevated to the status of a mental disorder because it supposedly increases risk for another disorder is an odd idea. Should high levels of cholesterol constitute a disorder because they increase risk for heart disease?) For these reasons, the diagnosis has been the subject of lively debate (e.g., Bryant & Harvey, 2000b; R.D. Marshall et al., 1999; R.D. Marshall, Spitzer, & Liebowitz, 2000; Spiegel, Classen, & Cardeña, 2000).

Given that the modal outcome following trauma is recovery, can the ASD diagnosis identify persons destined to remain impaired? To date, 12 prospective studies have addressed whether the presence of ASD predicts later PTSD (Brewin et al., 1999; Bryant & Harvey, 1998; Creamer, O'Donnell, & Pattison, in press; Difede et al., 2002; Harvey & Bryant, 1998b, 1999, 2000b; Holeva, Tarrier, & Wells, 2001; Kangas, Henry, & Bryant, in press; Murray et al., 2002; Schnyder et al., 2001; Staab, Grieger, Fullerton, & Ursano, 1996; see Table 3). Marked methodological variability across studies likely contributes to discrepant findings regarding the relation between ASD and PTSD. For example, very low incidence rates of ASD and PTSD in certain studies may have arisen from extremely restrictive inclusion criteria, such as including only patients with very severe physical injuries (e.g., Creamer et al., in press: Schnyder et al., 2001). There is also marked variability in the types of trauma studied, ranging from motor vehicle accidents to assaults, natural disasters, and burns. As Table 3 indicates, there are two ways to evaluate the results of these prospective studies. When one looks at the proportion of people who initially displayed ASD and subsequently developed PTSD, the ASD diagnosis appears to predict PTSD reasonably well (e.g., Brewin et al., 1999; Bryant & Harvey, 1998; Harvey & Bryant,

Table 3. *Summary of prospective studies of acute stress disorder*

Trauma type	Study	Percentage of people with ASD who develop PTSD	Percentage of people with PTSD who had ASD
Motor vehicle accident	Harvey & Bryant (1998b)	78	39
Brain injury	Bryant & Harvey (1998)	83	40
Assault	Brewin, Andrews, Rose, & Kirk (1999)	83	57
Motor vehicle accident	Holeva, Tarrier, & Wells (2001)	72	59
Motor vehicle accident	Creamer, O'Donnell, & Pattison (in press)	30	34
Accidents	Schnyder, Moergeli, Klaghofer, & Buddeberg (2001)	34	10
Typhoon	Staab, Grieger, Fullerton, & Ursano (1996)	30	37
Cancer	Kangas, Henry, & Bryant (in press)	53	61
Motor vehicle accident	Harvey & Bryant (1999)	82	29
Brain injury	Harvey & Bryant (2000b)	80	72
Motor vehicle accident	Murray, Ehlers, & Mayou (2002)	77[a]	34
Burns	Difede et al. (2002)	87	78

Note. ASD = acute stress disorder; PTSD = posttraumatic stress disorder.

[a]This rate is based on assessments conducted 4 weeks after the trauma; the proportion of participants with ASD who developed PTSD was 32% when ASD was assessed 1 week after the trauma.

1998b, 1999, 2000b). Across these studies, approximately three quarters of trauma survivors with ASD subsequently developed PTSD.

In contrast, the predictive ability of the ASD diagnosis is less promising when one calculates the proportion of people who eventually developed PTSD and who initially displayed ASD. This approach reveals that although some reports indicated that the majority of people with PTSD initially displayed ASD, most studies found that only a minority of people with PTSD suffered ASD within the initial month after trauma exposure. That is, the capacity of the ASD diagnosis to accurately identify most people who will eventually develop PTSD appears limited. The limitations of the ASD diagnosis as a reliable and sensitive predictor of subsequent PTSD have also been underscored by recent evidence that the ASD diagnosis may not be superior to PTSD criteria (employed within the initial month after trauma exposure) as a means of identifying people who will subsequently develop PTSD (Brewin, Andrews, & Rose, 2003). Further, although Difede et al. (2002) found that 87% of burns survivors with ASD subsequently developed PTSD, they also reported that applying the PTSD criteria (except duration of symptoms) 2 weeks after burn injury identified the same individuals as developing PTSD.

One major reason for the variability in prospective studies of ASD and PTSD may be the timing of assessments of ASD. Although DSM-IV stipulates that ASD can be diagnosed after 2 days have elapsed since trauma exposure, it is likely that attempting a diagnostic decision this soon will increase the likelihood that a transient stress reaction will be incorrectly classified as a case of ASD. Indeed, Murray et al. (2002) found that the predictive value of the ASD diagnosis depended on when the patients were assessed. Among survivors of motor vehicle accidents, 77% of those who met ASD criteria at 4 weeks developed PTSD, compared with only 32% of those who met ASD criteria at 1 week after trauma exposure. The rapidly changing nature of stress reactions in the initial weeks following trauma exposure is underscored by evidence from studies of civilians involved in the Gulf War, in which many people who suffered immediate stress reactions in the initial days displayed marked adaptation in the following weeks (Solomon, Laor, & McFarlane, 1996). Attempts to distinguish between transient stress reactions and harbingers of chronic disorder on the basis of symptoms expressed within days of trauma exposure will likely be very difficult.

It appears that the major reason why the ASD diagnosis fails to identify many people who eventually develop PTSD (see the right-most column in Table 3) is that the requirement that three dissociative symptoms be present excludes many people who nonetheless develop PTSD. For example, Harvey and Bryant (1998b) reported that 60% of trauma survivors who displayed acute reexperiencing, avoidance, and hyperarousal, but no dissociation, developed PTSD. This pattern undermines the claim that acute dissociation is a necessary harbinger of subsequent pathology.

PREVENTING POSTTRAUMATIC PSYCHOPATHOLOGY

Although many people experience acute stress-related symptoms in the wake of traumatic events, only a minority develop ASD, PTSD, or both. Most people recover from traumatic events without any professional assistance. But given that a significant minority of people exposed to trauma do develop lasting psychological problems, what sort of interventions should be offered, when should they be offered, and to whom? When considering these issues, one should be mindful of important distinctions between different kinds of interventions. *Primary prevention* of PTSD and other posttraumatic problems (e.g., ASD, depression, substance abuse) entails taking steps to reduce the frequency of traumatic events (e.g., restricting adolescents' access to firearms to diminish risk of school violence). These steps usually fall within the bailiwick of law and public health rather than clinical psychology and psychiatry. *Secondary prevention* comprises crisis intervention techniques, such as psychological debriefing, that are delivered within days of the trauma and designed to mitigate distress and prevent the emergence of posttraumatic psychopathology. *Early treatment interventions* are delivered soon after posttraumatic disorders have emerged, but early in the course of the disorders.

In this review, we concentrate on whether secondary prevention, especially the widely used psychological debriefing, and early treatment interventions promote recovery from posttraumatic stress. We acknowledge that survivors and communities have many needs in the aftermath of trauma, and that the prevention of persistent symptoms of psychological distress is only one of them. It is, however, beyond our scope here to review the many different targets of crisis intervention and their effectiveness.

PSYCHOLOGICAL DEBRIEFING

Psychological debriefing has its roots in World War I (Litz, Gray, Bryant, & Adler, 2002). Following a major battle, commanders would meet with their men to debrief them. The objective was to boost morale by having combatants share stories about what had happened during the engagement. This historical group debriefing method was also used by American troops during World War II and continues to be used by the Israeli army today (Shalev, Peri, Rogel-Fuchs, Ursano, & Marlowe, 1998).

Drawing parallels between the stress of combat and the stress of emergency medical service, Mitchell (1983) reasoned that a similar approach might diminish stress reactions among firefighters, police officers, emergency medical technicians, and other people exposed to what he referred to as "critical incidents" (i.e., traumatic events). A former firefighter and paramedic, Mitchell obtained a Ph.D. in human development and developed the most widely used method of psychological de-