2002, Galea's group did a follow-up study on another group of adults living south of 110th Street, and found that only 1.7% of the sample had PTSD related to the attacks (Galea, Boscarino, Resnick, & Vlahov, in press). The 7.5% rate obtained within weeks of the attacks may have reflected temporary distress rather than mental illness. This study, like many others in the field (e.g., Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), demonstrates that most people are resilient and recover from early posttrauma symptoms. The authors of another post-September 11 survey of New Yorkers concluded that "those with severe symptoms were far fewer than what we expected, given the magnitude and amount of personal exposure to this disastrous event" (DeLisi et al., 2003, p. 782).

Exposure to Trauma and PTSD

Many therapists have conceptualized PTSD as a normal, expectable reaction to an extraordinary stressor, despite its classification as a mental disorder. The traumatic event itself has been awarded overriding causal significance in producing PTSD; personal vulnerability factors have been minimized. These assumptions have been increasingly questioned in recent years, however (Yehuda & McFarlane, 1995). Epidemiological studies have shown that many American adults have been exposed to DSM-defined traumatic stressors, such as physical assault, rape, or automobile accidents, yet few of them have developed PTSD. The National Comorbidity Survey revealed that 60.7% of randomly sampled adults reported exposure to DSM traumatic stressors (Kessler et al., 1995). But of these trauma-exposed people, only 20.4% of the women and 8.2% of the men had ever developed PTSD. Among adults living in metropolitan Detroit, 89.6% reported exposure to DSM traumatic stressors, yet only 13% of the women and 6.2% of the men had developed PTSD (Breslau, Davis, Andreski, & Peterson, 1991).

Among traumatic stressors, those involving intentional acts of violence are especially likely to produce PTSD (Yehuda, 2002b). In one epidemiological survey, PTSD developed in 11.6% of respondents who had experienced a sudden injury or accident, but in 22.6% of those who had experienced physical assault and in 80% of female rape victims (Breslau et al., 1991). In another study, the single most frequent event causing PTSD was learning about the unexpected death of a loved one; 26.5% of female cases and 38.5% of male cases of PTSD were attributed to this very common event (Breslau, Chilcoat, Kessler, Peterson, & Lucia, 1999). (Being "confronted with an event" qualifies as a PTSD-inducing traumatic stressor, according to DSM-IV; APA, 1994, p. 427. Thus, individuals who, for example, receive a phone call informing them of the unexpected death of a loved one qualify as having been exposed to a traumatic stressor.) Breslau, Chilcoat, Kessler, Peterson, and Lucia found that men are more likely to be exposed to trauma than women, but that trauma-exposed women develop PTSD at twice the rate as do trauma-exposed men, mainly because exposure to criminal violence precipitates PTSD at a much higher rate in women than in men (35.7% vs. 6%).

The Time Course of Posttrauma Symptoms

Most people recover from acute symptoms within 3 months posttrauma, even if they do not receive any treatment (e.g., Kessler et al., 1995). For example, Riggs, Rothbaum, and Foa (1995) reported that 71% of women and 50% of men met symptomatic criteria for PTSD (the requirement of 1-month duration was waived) approximately 19 days after a nonsexual assault. Four months posttrauma, the rate of PTSD had dropped to 21% for women and 0% for men. This research group also reported that 94% of rape victims interviewed an average of 2 weeks posttrauma met criteria for (acute) PTSD; 11 weeks later, the rate dropped to 47% (Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992). About half of trauma survivors who are still symptomatic at 3 months recover over the next few years (Ehlers, Mayou, & Bryant, 1998; Kessler et al., 1995; Schnyder & Moergeli, in press).

RISK FACTORS FOR PTSD

Researchers have endeavored to identify variables that heighten risk for PTSD, studying both who is most likely to be exposed to trauma and who among the trauma exposed is most likely to develop the disorder (Brewin, Andrews, & Valentine, 2000; Yehuda, 1999).

Risk Factors for Trauma Exposure

The most important risk factor for PTSD is, of course, exposure to trauma. People vary considerably in this risk. Certain occupations clearly increase risk (e.g., soldier, firefighter). Some studies suggest that the people who choose these occupations possess other characteristics (e.g., psychological hardiness) that counteract the risk of PTSD that exposure to trauma entails.

For example, North et al. (2002) assessed 176 male firefighters approximately 34 months after they had done rescue and recovery work at the site of the Oklahoma City terrorist bombing. The rate of disaster-related PTSD (13%) was significantly lower among the firefighters than among 88 male primary victims of the bombing itself (23%). Among firefighters with any psychiatric disorder after the bombing, 82% had preexisting psychopathology. Unfortunately, North et al. were unable to interview all of the firefighters, and volunteers for the study constituted less than 25% of those working at the site.

North et al. (2002) speculated that psychological preparedness for dealing with gruesome aspects of firefighting may have been one variable fostering resilience among the men who worked at the Oklahoma bombing site. Similarly, Başoğlu,

Mineka, Paker, Livanou, and Gök (1997) reported that psychological preparedness for trauma buffers individuals against subsequent symptoms. These researchers found significantly lower rates of current PTSD among left-wing Turkish political activists who had been tortured by the military regime than among nonactivist Turks who had been arrested for nonpolitical crimes and tortured (18% vs. 58%). These findings are especially striking because the activists had been exposed to more torture than the nonactivists. Combining both activist and nonactivist groups for further analyses, Başoğlu et al. found that the more psychologically prepared victims were (e.g., knowing about torture methods, being aware that torture often followed arrest, being trained in stoicism techniques), the less severe were their torture-related PTSD symptoms.

Realizing that severe stressors often do not strike people at random, Breslau's research group endeavored to identify risk factors for traumatic exposure in the general population. In a retrospective study, they found that adults reporting having had childhood conduct problems were more likely than other adults to be exposed to trauma in adulthood (Breslau et al., 1991). People reporting mental illness in their family members were also at heightened risk for exposure to trauma. In their subsequent prospective epidemiological study, Breslau, Davis, and Andreski (1995) found that higher scores on questionnaire measures of extraversion and neuroticism were associated with increased likelihood of subsequent exposure to traumatic events. Men were at greater risk than women; individuals lacking a college degree were at greater risk than college graduates; and Blacks were at greater risk than Whites.

Risk Factors for PTSD Among People Exposed to Trauma

Among people exposed to traumatic events, what factors are associated with increased risk for PTSD? These factors might include both variables operative prior to the trauma (e.g., high vs. low cognitive ability) and variables that come into play posttrauma (e.g., high vs. low levels of social support).

Cross-sectional studies

Some cross-sectional studies on people with PTSD have revealed correlates of the disorder that constitute plausible risk factors, even though these variables were measured after individuals had developed PTSD. People who are already suffering from anxiety or mood disorders are at heightened risk for PTSD, as are those with a family history of these disorders (Breslau et al., 1991). Relative to other trauma-exposed adults, those with PTSD report higher rates of having been sexually (Nishith, Mechanic, & Resick, 2000) or physically (Breslau, Chilcoat, Kessler, & Davis, 1999) abused during childhood. Retrospective reports of instability in one's family during childhood are likewise associated with PTSD (King, King, Foy, & Gudanowski, 1996).

Cognitive ability is related to risk for PTSD among people exposed to trauma. For example, McNally and Shin (1995) found that lower intelligence was associated with greater severity of PTSD symptoms among Vietnam veterans even after controlling for the extent of combat exposure (measured by the veterans' self-reports). Other researchers have replicated this finding (Silva et al., 2000; Vasterling, Brailey, Constans, Borges, & Sutker, 1997; Vasterling et al., 2002). For example, studying inner-city children and adolescents, Silva et al. (2000) found that IQ was the best predictor of resilience against PTSD among those exposed to trauma (e.g., witnessing violence, being sexually abused). Of those with above-average IQ scores, 67% had neither PTSD nor subthreshold PTSD. Of those with below-average IQ scores, only 20% had no PTSD symptoms. Note, however, that in these studies, researchers administered tests of cognitive ability among people who had already been exposed to trauma and who had already (in many cases) developed PTSD.

Neuroticism—a personality trait reflecting proneness to experience negative emotions (irritability, anxiety, depression)—is higher among trauma-exposed people with PTSD than among those without the disorder (e.g., Breslau et al., 1991; McFarlane, 1989). Unfortunately, it is unclear whether heightened neuroticism results from trauma (or PTSD), is a vulnerability factor for PTSD, or both.

Some retrospectively assessed risk factors tap variables apparently linked to the posttrauma environment rather than pretrauma antecedents. For example, self-reported low social support is associated with PTSD among Vietnam combat veterans (e.g., Boscarino, 1995; Keane, Scott, Chavoya, Lamparski, & Fairbank, 1985) and among civilians (see meta-analysis by Brewin et al., 2000). (Meta-analysis is a statistical technique for combining the results of similar studies, hence enhancing the reliability of any conclusion about the hypothesis under consideration.) Thus, people who are exposed to trauma, but who lack social support, may be at heightened risk for developing PTSD. Stated differently, a supportive posttrauma environment might hasten reduction in acute symptoms, thereby reducing risk for PTSD. Unfortunately, it is difficult to tell what mechanism is responsible for the association between low social support and risk of PTSD. It may be that posttrauma symptoms, such as anger outbursts and emotional withdrawal, alienate potential sources of social support. In addition, impoverished social-support networks make it difficult for people to overcome the effects of trauma. Nevertheless, negative perceptions of other people's responses (e.g., "I feel that other people are ashamed of me now") predict PTSD beyond what can be predicted from initial symptom levels (Dunmore, Clark, & Ehlers, 2001).

Prospective studies

The ideal method for identifying risk factors for PTSD is to take pretrauma measurements on a large number of individu-

als, perhaps those likely to encounter traumatic events (e.g., firefighters, military personnel), and to follow them over time. One can then identify those who are exposed to trauma and determine the putative risk factors that seem to predict who develops the disorder and who does not, as well as who recovers. Such prospective, longitudinal studies are expensive and therefore difficult to conduct, however. Another approach is to comb archival records of trauma-exposed individuals and identify variables, measured pretrauma, that correlate with PTSD.

Studying predeployment military archives of inductees who later fought in Vietnam, researchers found that those who developed PTSD had reported more school difficulties, had lower arithmetic aptitude, and (paradoxically) had lower heart rate than did combat veterans who did not develop PTSD (Pitman, Orr, Lowenhagen, Macklin, & Altman, 1991). Because of multiple statistical comparisons, Pitman et al. urged readers to interpret these significant differences as "trends" (p. 418). Schnurr, Friedman, and Rosenberg (1993) obtained premilitary scores from the Minnesota Multiphasic Personality Inventory (MMPI; a self-report measure of normal and abnormal personality variation) for a group of Dartmouth College undergraduates who later served in Vietnam. After controlling for extent of combat exposure, Schnurr et al. found that elevations on several MMPI scales (Hypochondriasis, Psychopathic Deviate, Paranoia, Femininity) predicted PTSD symptoms. In another study, Bramsen, Dirkzwager, and van der Ploeg (2000) found that predeployment negativism—a personality trait similar to neuroticism—predicted subsequent PTSD symptoms among Dutch peacekeepers serving in the former Yugoslavia.

Two prospective studies of Vietnam veterans and Israeli soldiers indicate that above-average intelligence may buffer people against the traumatic effects of stressors (Kaplan et al., 2002; Macklin et al., 1998). In both studies, higher precombat IQ scores predicted lower risk for PTSD following combat. Lower precombat intelligence predicted greater severity of PTSD symptoms at the time of the studies (Macklin et al., found that this was true even after controlling statistically for amount of self-reported combat exposure). Macklin et al. found no significant association between severity of PTSD symptoms at the time of the study and change in IQ score since predeployment. In other words, the stress of chronic PTSD did not lower scores on IQ tests taken at the time of the study.

Peritraumatic risk factors

Another approach to studying risk factors is to identify peritraumatic responses—those occurring during the trauma itself or shortly after it—that predict later PTSD. Studies that take this approach are longitudinal in the sense that traumatized people are followed over time, but they are not truly prospective because measurements are taken after trauma has occurred. They are prospective in the sense that measurements are taken shortly after the event, before a month has elapsed (the duration requirement for PTSD).

Researchers have tested whether the severity of symptoms at various times posttrauma is correlated with subsequent PTSD. Symptom severity in the first few days following the trauma fails to predict later PTSD (e.g., Shalev, 1992), whereas greater symptom severity from 1 to 2 weeks posttrauma and onward correlates highly with subsequent symptom severity (e.g., Harvey & Bryant, 1998b; Murray, Ehlers, & Mayou, 2002; Shalev, Freedman, Peri, Brandes, & Sahar, 1997). For example, an Israeli study of survivors of motor vehicle accidents indicated that greater severity of PTSD symptoms assessed 1 week after the accident predicted greater risk of a diagnosis of PTSD 1 year later (Koren, Arnon, & Klein, 1999). However, as Shalev et al. pointed out, low initial symptom levels are better at predicting a favorable outcome than high initial symptom levels are at predicting PTSD.

Several studies have suggested that self-reported peritraumatic dissociation (e.g., feeling unreal or experiencing time slowing down during the trauma) predicts subsequent PTSD among trauma survivors. Indeed, a recent meta-analysis indicated that peritraumatic dissociation was the single best predictor of PTSD ($r = .35$) among trauma-exposed individuals (Ozer, Best, Lipsey, & Weiss, 2003). Unfortunately, Ozer et al. failed to include cognitive ability in their meta-analysis, and both cross-sectional (e.g., Vasterling et al., 1997—$r = -.37$) and prospective (e.g., Macklin et al., 1998—$r = -.45$) studies have shown that the predictive power of cognitive ability is at least as great as that of peritraumatic dissociation.

Some researchers have investigated possible links between specific symptoms of peritraumatic dissociation and risk for subsequent PTSD. Among Israeli civilians exposed to motor vehicle accidents, terrorist attacks, and other traumatic events, a sense that what was happening was unreal (derealization) and a sense of time distortion (things happening in slow motion)—measured 1 week posttrauma—predicted who met criteria for PTSD 6 months later (Shalev, Peri, Canetti, & Schreiber, 1996). In another study, depersonalization (feeling disconnected from one's body), emotional numbing, motor restlessness, and a sense of reliving the trauma—measured within 1 month after the trauma—predicted later PTSD among survivors of automobile accidents (Harvey & Bryant, 1998b). However, Murray et al. (2002) found that although peritraumatic dissociation measured within 24 hr or 1 week of the trauma predicted PTSD 6 months after a motor vehicle accident, persistent dissociation at 4 weeks was a more powerful predictor.

It remains unclear whether dissociation in the aftermath of the trauma predicts PTSD over and above what can be predicted from symptoms of reexperiencing, avoidance, and hyperarousal. Brewin, Andrews, Rose, and Kirk (1999) and G.N. Marshall and Schell (2002) found no evidence that peritraumatic dissociation is an independent and unique predictor of subsequent PTSD severity, whereas Murray et al. found that dissociation improved the prediction of subsequent PTSD severity. Among individuals exposed to community violence, peritraumatic dissociation failed to predict later PTSD symp-

toms once Marshall and Schell controlled for initial severity of PTSD symptoms. This study also highlighted the problems associated with earlier studies that required trauma survivors to recall their levels of peritraumatic association at a later time, because Marshall and Schell found that later recall of peritraumatic dissociation was often inaccurate.

Overall, there are mixed findings concerning the extent to which acute dissociative symptoms predict PTSD. In several studies, peritraumatic dissociation was a powerful predictor of subsequent PTSD (e.g., Ehlers et al., 1998; Koopman, Classen, & Spiegel, 1994; Murray et al., 2002; Shalev, Freedman, et al., 1998; see also the meta-analysis of Ozer et al., 2003). However, peritraumatic dissociation failed to predict PTSD in other prospective studies (e.g., Dancu, Riggs, Hearst-Ikeda, Shoyer, & Foa, 1996; G.N. Marshall & Schell, 2002). These findings are consistent with other evidence that peritraumatic dissociation does not necessarily result in later psychiatric disorders (see Harvey & Bryant, 2002). For example, survivors of harrowingly close brushes with death often report having experienced intense dissociation (e.g., depersonalization, time seeming to slow down), but they seldom develop later psychiatric problems (Noyes & Kletti, 1976, 1977). Indeed, among these survivors, "many commented that they had been without frightening dreams or anxiety after their accidents and also that they had not found memory of the accident disturbing" (Noyes & Kletti, 1976, p. 26). Because these close brushes with death produce so "little traumatic aftermath" (Noyes & Kletti, 1977, p. 382), Noyes and Kletti concluded that peritraumatic depersonalization is an adaptive mechanism.

How can one make sense of the mixed findings about the capacity of peritraumatic dissociation to predict subsequent PTSD? There are several explanations available. One possibility is that peritraumatic dissociation leads to later PTSD because the dissociation is associated with other known risk factors. Keane, Kaufman, and Kimble (2001) noted the evidence suggesting that a history of childhood trauma is associated with subsequent dissociation (Spiegel & Cardeña, 1991), and raised the possibility that peritraumatic dissociation may be linked to PTSD because of its association with childhood trauma, which is a strong risk factor for subsequent PTSD. Keane et al. proposed that more rigorous testing of the link between peritraumatic dissociation and PTSD will be achieved by including dissociative reactions at the time of the recent precipitating trauma in mathematical models that test the relative contributions of pretrauma, peritraumatic, and posttrauma factors. The possible association of acute dissociation with distinct vulnerability factors may explain, to some degree, the discrepant findings about the relationship of peritraumatic dissociation and PTSD.

A related possibility is that dissociation influences the development of PTSD in only a subset of trauma-exposed individuals. Some theorists have proposed diathesis-stress models according to which only people who possess dissociative tendencies may respond to trauma with dissociative reactions (Butler, Duran, Jasiukaitis, Koopman, & Spiegel, 1996). Consistent with this notion is evidence that people who display dissociative reactions following trauma are more hypnotizable (hypnotizability is strongly correlated with dissociative tendencies) than people who develop acute stress reactions without dissociative symptoms (Bryant, Guthrie, & Moulds, 2001). Although both groups may have high risk for developing PTSD, only the subset of people who possess dissociative tendencies appear to respond with acute dissociative symptoms. This pattern may explain why acute dissociation is not uniformly found to be a strong predictor of subsequent PTSD. The proposal that some people will develop PTSD after having acute dissociative symptoms and others will develop PTSD without this initial reaction points to different pathways of PTSD development. Consistent with the view that there are multiple pathways to PTSD is evidence that dissociation is a stronger predictor of subsequent PTSD in women than in men (Bryant & Harvey, 2003).

Another possibility is that the influence of peritraumatic dissociation on subsequent PTSD may be mediated by how individuals appraise their dissociative reactions. Cognitive theorists posit that PTSD develops and is maintained by catastrophic interpretations of both the traumatic event and the individual's resultant responses (Ehlers & Clark, 2000). People who are likely to develop posttraumatic psychopathology exaggerate the probability of future negative events occurring (Smith & Bryant, 2000; Warda & Bryant, 1998). Further, negative appraisals in the initial period after trauma exposure predict subsequent PTSD (Engelhard, van den Hout, Arntz, & McNally, 2002), as do catastrophic attributions of responsibility for a trauma in this period (Andrews, Brewin, Rose, & Kirk, 2000; Delahanty et al., 1997). Negative appraisals of peritraumatic dissociation may predict subsequent PTSD better than do dissociative reactions themselves. For example, an individual who interprets emotional numbing as a normal response to an assault may be less distressed than an individual who interprets numbing as a sign of madness.

For a range of traumas, how distressed a victim was during the trauma or how threatened the person felt (e.g., whether the person believed that he or she was about to die) often has predicted PTSD better than objective measures of stressor magnitude, such as the extent of bodily injury (e.g., Ehlers et al., 1998; Perry, Difede, Musngi, Frances, & Jacobsberg, 1992; Schnyder, Moergeli, Klaghofer, & Buddeberg, 2001). The higher the distress or perceived threat, the more severe PTSD symptoms were likely to be.

The conclusions that people draw from the trauma can be distorted and can contribute to the subsequent distress, making it difficult for survivors to put the event in the past. For example, rape victims may feel very ashamed and blame themselves for their attacks; accident survivors may think they are incompetent because they did not prevent the crash from happening; and assault survivors may feel that there is something about them that will make it very likely that they will be attacked

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.