Etiology of PTSD: What causes PTSD?

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Although most people will encounter a traumatic stressor at least once, and often several times, in their lives, most people who experience a traumatic stressor do *not* develop PTSD (Breslau, 2002; Kessler et al., 2005; Kessler, Sonnega, Bromet, & Hughes, 1995). Therefore, the question of what causes PTSD cannot simply be answered by referring to its definition: a disorder whose symptoms occur following exposure to a traumatic stressor. In fact, there is controversy as to whether PTSD symptoms really are caused by exposure to traumatic stressors, because all of the symptoms, except for memories, flashbacks, and nightmares of traumatic events, could occur regardless of whether a person has experienced a traumatic stressor. The PTSD symptoms that are not by definition tied to a traumatic stressor—even the two symptoms that are defined as psychological or physical distress due to reminders of past stressful events—actually are also symptoms of other psychiatric disorders as well as of PTSD. Thus, it is important to scientifically examine the assumption that PTSD is caused by exposure to traumatic stressors (Box 3.1). Scientific evidence indicating that PTSD is most likely to occur not just when a traumatic stressor has occurred—but *when the objective severity of exposure to traumatic danger or harm is more extreme*—provides important (albeit not definitive) support for the view that exposure to a traumatic stressor plays a key role in PTSD, as is discussed later in this chapter (see section on the Impact of Stressor Exposure).

In this chapter, a more nuanced view of the causes of PTSD is presented than the commonsense version that PTSD is "caused" simply by exposure to a traumatic stressor. Research demonstrates that PTSD is a "multicausal" phenomenon, meaning that it is the product of a combination of a number of potential causes. Rather than describing the factors that contribute to the development of PTSD as "causes," it is clearer and more factual to describe them as "risk factors" and "protective factors"—that is, things that increase a person's risk of developing PTSD and things that reduce (or protect against) the risk of developing PTSD. As you will see, risk factors include not only exposure to a traumatic stressor but also biological, psychological, and social factors that influence whether PTSD will occur and that can protect against (but not necessarily prevent) the development of PTSD (Box 3.2).

Box 3.1 Key Points

1. The "etiology" of PTSD refers to the study of the "causes of PTSD." Psychological phenomena such as PTSD are rarely, if ever, "caused" by a single factor or any single specific combination of factors (such as specific genes or personality traits or life

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Box 3.1 Continued

experiences) but instead are the product of complex interrelationships among many different biological, environmental, and life experience factors.

2. PTSD is *not* "caused" solely by exposure to traumatic stressors. Although, by definition, PTSD can only occur if a person has been exposed to a traumatic stressor, most people—about 80–90%—who are exposed to traumatic stressors do *not* develop PTSD. Therefore, exposure to a traumatic stressor is a *necessary* but *not sufficient* cause that contributes to—but does not alone account for—PTSD.
3. PTSD is the result of large number of different combinations of "risk" and "protective" factors that influence a person's life trajectory (see Chapter 2) after exposure to a traumatic stressor. Risk factors increase, and protective factors decrease, the likelihood that PTSD will occur.
4. Some "risk" and "protective" factors begin before a traumatic stressor occurs. These "preevent" factors include the person's genetic characteristics (including gender and ethnocultural background, as well as subtler variations in specific genes associated with memory, learning, motivation, and emotional and behavioral problems) that are just beginning to be understood in relation to PTSD. Preevent risk factors also include age (although both children and older adults actually are at *lower* risk generally, compared to adolescents or young and midlife adults). Other preevent risk factors for PTSD are the person's and their family members' previous exposure to traumatic events and past episodes of PTSD, psychiatric or addiction problems, and interpersonal conflicts. A key preevent protective factor that lowers the risk of PTSD is access to socioeconomic resources, most importantly as a result of a higher education level and social support.
5. Female gender is a risk factor, and male gender is a protective factor, for PTSD. However, it is not certain that this is directly related to genetically inborn differences between females and males, because research has shown that females and males may be exposed to different types of traumatic stressors and related risk factors (such as gender bias) as a result of social and political factors that are independent of their biological sex.
6. Persons from ethnoracial minority backgrounds may be more vulnerable (i.e., at greater risk) for developing PTSD than those of majority ethnocultural backgrounds. However, this may be a by-product of social and economic factors (such as racial stigma and discrimination, or poverty) rather than due to a person's ethnicity per se.
7. The specific experience of psychological trauma differs in many ways that may increase or protect against the risk of developing PTSD. Traumatic stressors that involve betrayal or violation of the basic safety of key relationships (such as physical or sexual abuse, family violence, cruel and inhumane imprisonment, or torture), especially if this occurs during the formative years of childhood, increase the risk of PTSD 75% or more.
8. When traumatic events occur directly to the person, and when severe physical injury or pain result, the risk of or "vulnerability" to PTSD is higher—although witnessing very horrific events or their aftermath, especially if this occurs to someone close or on a mass scale (such as genocide or war), can lead to a high risk of PTSD (see Chapter 11).
9. In addition to the nature of the traumatic event, certain reactions immediately or soon after the traumatic event(s) are risk factors. These are called "peritraumatic" risk factors because they occur during or soon after ("peri") a traumatic event). High levels of initial distress (including large increases in physiological stress, such as

blood pressure) or dissociation (i.e., becoming mentally and physically shut down and profoundly disoriented) are the primary peritraumatic risk factors demonstrated by research.

10. Social support and coping self-efficacy (the belief that it is possible to effectively cope with and overcome adversity) are key postevent protective factors against PTSD.

Box 3.2 "Is PTSD Caused by Traumatic Stress?"

This provocative and timely question was the title of an article by Bodkin et al. (2007) in a special issue of the *Journal of Anxiety Disorders.* They report the results of a study in which adults who were receiving antidepressant medications were assessed for trauma history and PTSD using the *Structured Clinical Interview for DSM-IV Axis I Disorders* (First, Spitzer, Gibbon, & Williams, 1996). More than half of the participants reported experiences that qualified as traumatic stressors, and most of them (78%) also reported sufficient symptoms to be diagnosed with PTSD. However, an equivalent proportion (78%) of the participants who did *not* report any past exposure to a traumatic stressor nevertheless reported sufficient symptoms to be diagnosed with PTSD. The authors conclude that it was not necessary to have experienced a traumatic stressor in order to have substantial symptoms of PTSD and that therefore (at least among people seeking treatment for a psychiatric disorder—depression in this case), there is reason to doubt that traumatic stressors "cause" PTSD.

Another study used a thorough trauma history interview and the *Structured Clinical Interview for DSM-III-R* to assess PTSD symptoms (see Chapter 6) with men and women who were diagnosed with one or more personality disorders and were receiving outpatient mental health treatment (Golier et al., 2003). As expected, participants who reported a history of having been physically assaulted as an adult or abused as a child or adolescent were more likely than other patients to be diagnosed with PTSD and also with borderline personality disorder. Those reporting other (accident, disaster, or war-related) traumatic stressors in adulthood were *not* more likely to be diagnosed with either PTSD or borderline personality disorder, but they were more likely to be diagnosed with a paranoid personality disorder. However, patients with PTSD or either personality disorder were more likely to *not* report having experienced each of these types of psychological trauma than to report each type of psychological trauma. Thus, while traumatic stressors involving assault or abuse were definitely related to PTSD, they did not appear to singularly determine who developed PTSD, and they were equally or more related to the two personality disorders as to PTSD. This is consistent with research showing that risk and protective factors, including genetics, personality, anxiety-proneness, intelligence, education, and social support, influence the likelihood and severity of PTSD in addition to trauma exposure.

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Thus, it is clear that exposure to traumatic stressor(s) does not alone cause PTSD and that PTSD-like symptoms may occur to people who do not report any exposure to traumatic stressors. However, these facts do not add up to a conclusion that PTSD is a pseudo-disorder (i.e., a false and misleading diagnosis). Rosen and Taylor (2007) consider the question of whether "pseudo-PTSD" can occur and note that people can pretend to have a trauma history and PTSD symptoms when they really do not ("malingering")—often in an attempt to get benefits such as a disability pension—or can genuinely but falsely believe that they experienced traumatic stressors and are suffering from PTSD symptoms when they really are not ("factitious PTSD"). Weeding out these individuals is very important for clinical, legal, and research purposes (e.g., to prevent misuse of workers compensation, veterans' benefits, and civil law systems), although in practice, even with the best tools (see Chapter 10), it is "almost impossible" (Rosen & Taylor, 2007, p. 204; see also Spitzer, First, & Wakefield, 2007, for a discussion of preventing malingering in PTSD).

McHugh and Treisman (2007) press the point that PTSD is not caused by exposure to traumatic stressors by examining historical accounts (see also Chapter 1; Jones & Wessely, 2007) of stress reactions that preceded the first formal diagnoses of traumatic stress disorders (Gross Stress Reactions in the 1952 *Diagnostic and Statistical Manual*; Posttraumatic Stress Disorder in the 1980 *Diagnostic and Statistical Manual*, 3rd ed.). They argue that the PTSD diagnosis should be eliminated because the symptoms reflect either natural processes of adaptation following exposure to a traumatic stressor (and therefore should not be considered a psychiatric "disorder") or a misclassification of another psychiatric disorder whose symptoms overlap with those of PTSD (such as major depressive disorder or anxiety disorders).

In response, Spitzer et al. (2007) wrote a rejoinder on "saving PTSD from itself in *DSM*-5" recommending several specific changes in the wording of PTSD criteria to clarify the link between exposure to a traumatic stressor and the PTSD symptoms. Spitzer et al. (2007) more fundamentally propose eliminating symptoms from the list of 17 that make up PTSD that are not specific to PTSD—that is, that are identical or highly similar to symptoms of other psychiatric disorders: "irritability, insomnia, difficulty concentrating, and markedly diminished interest" (p. 237). Subsequently, research studies testing the effects of removing these items (and combining PTSD avoidance, emotional numbing, and hyperarousal items into a single set) have found that there would be at most small changes in how often PTSD was diagnosed and in how often PTSD occurred in combination with depression or anxiety disorders (Elhai & Palmieri, 2011; see also Ford et al., 2000 for an earlier study).

The fact that a diagnosis can be faked or mistakenly asserted and incorrectly documented does not render the diagnosis unusable or unnecessary—these problems affect every medical and psychiatric diagnosis (Rosen & Taylor, 2007). Nor should overlap in symptoms of PTSD with those of other diagnoses

disqualify the diagnosis, as this would eliminate many other medical and psychiatric diagnoses that share symptoms. The fundamental issue raised by the study by Bodkin et al. (2007) is not whether the PTSD symptoms could constitute a meaningful diagnosis but whether the diagnosis should include the requirement of a relationship between the symptoms and exposure to a traumatic stressor.

However, a major uncertainty in that study is whether the absence of *reporting* a traumatic stressor can be considered evidence of an absence of *actual exposure* to a traumatic stressor. The assessment instrument used, the *SCID*, is widely used for the diagnosis of PTSD but has been criticized as flawed in several ways. First, *SCID* questions used to ascertain exposure to traumatic stressors are brief and vague and do not systematically and specifically ask about the wide range of potentially traumatic stressors. By comparison, thorough and precise trauma history interviews or questionnaires such as the TESI (see Chapter 6) provide a complete survey of potentially traumatic events that reduces the risk of inadvertent underreporting.

Second, *SCID* questions do not guide the evaluator or interviewee in distinguishing between events that are objectively life-threatening or sexual violations versus those that are distressing or stressful but not technically traumatic. This could lead to either an overreporting of traumatic exposure or an underreporting—in the latter case, because events that may not clearly involve death or severe injury may nevertheless involve substantial danger or life threat that is not evident to either the respondent or the interviewer. Third, *SCID* symptom questions do not enable the interviewer to precisely assess the frequency and intensity of each PTSD symptom; instead, symptoms must be judged as "clinically significant," "subthreshold" (present but not serious), or not present. The distinction between clinically significant and subthreshold symptoms often is very difficult and can lead to over- or underreporting of the PTSD symptoms by the interviewer. For these reasons, it is unclear whether the determination of who had experienced a traumatic stressor and of who warranted a PTSD diagnosis was accurate in Bodkin and colleagues' study.

Evidence from an unexpected source—a study with laboratory rats—suggests that PTSD can occur despite the individual having no conscious memory of traumatic events. This is especially relevant for people who were exposed to maltreatment or family or community violence in the first years of life and subsequently develop PTSD symptoms. Although brain development in infancy is not sufficient at that preverbal stage to support more than fragmentary sensory memories—because words can at most be repeated or named but not yet used to form narrative story-like descriptions of complete events—infants definitely form nonverbal memories that can be virtually permanent. Traumatic events are likely to involve intense sensory experiences and thus to evoke strong and potentially long-lasting memories that could result in any of the PTSD symptoms except verbally describable recall of the traumatic events themselves (the first type of intrusive reexperiencing symptom). Consistent with this view, the

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animal study showed that rats that were exposed to painful foot shocks at an early age (19 days old, comparable to infancy for humans) did not react in adulthood with fear in the context in which the shocks occurred. There was no evidence of what in humans would be a conscious memory of the traumatic event. However, they had extreme startle reactions when given foot shocks as adults, and they displayed several biological and behavioral signs of persistent anxiety, including abnormal fluctuations in stress hormones, a surplus of stress-related (glucocorticoid) receptors in the amygdala, a high level of sensitivity to threats, avoidance of open areas in their environment, and even an aversion to odors that had been present when they were shocked in early life (Poulos et al., 2014). Thus, without evidence of remembering the foot shocks, these rats had changes in their behavior and in the areas in their brains and bodies that match those found in humans who have PTSD.

However, the question still remains whether people can develop PTSD despite never actually having been exposed to traumatic stressor(s). Studies have shown that people report levels of PTSD-like symptoms that are as high, or higher, in relation to distressing but not traumatic life events (such as divorce, job loss, burglary, or chronic illness) as to traumatic events (Anders, Frazier, & Frankfurt, 2011; Mol et al., 2005) and that PTSD symptoms occur along a continuum of severity from relatively mild reactions to nontraumatic stressors to severe reactions to traumatic stressors (Mulder, Fergusson, & Horwood, 2013). Results suggesting that milder "PTSD" symptoms may be quite different than symptoms of true PTSD comes from a study of adult twin pairs. The severity of psychiatric symptoms reported by men who had PTSD related to having been in military combat was substantially worse than for either his identical twin who had not been in combat (ruling out the effects of genetically based vulnerability to psychiatric problems) or for other combat-exposed men who did not have PTSD (Gilbertson et al., 2010). Thus, although people may report high levels of symptoms consistent with PTSD despite not having experienced a traumatic stressor, the symptoms appear to reflect a general sense of distress that has a less severe impact on the person's overall mental state (and potentially also their brain's and body's stress response systems; Poulos et al., 2014) than when PTSD follows exposure to traumatic stressor(s).

Further evidence of the role of traumatic stressors in the development of PTSD comes from a study that addressed the question of whether PTSD symptoms should be assessed with reference to only one traumatic stressor (presumably the past event that was the worst, or that is currently most distressing to recall) or to all traumatic stressors that have ever occurred in the individual's life (Elhai et al., 2009). The study included three groups of college students, all of whom rated their own PTSD symptoms, based on (i) all past traumatic events in their life; (ii) the one past traumatic event they found most distressing to recall currently; and (iii) other nontraumatic stressors in their life (this group had not

experienced a traumatic stressor). The key finding was that the PTSD symptoms reported by the first two groups (all past trauma and one worst past trauma) tended to be organized in a consistent manner, but the symptoms reported by the third group (no past trauma) were more scattered and did not fit a single PTSD profile. The profile of PTSD symptoms also differed between the all past trauma group and the one worst past trauma group in an interesting way. The all past trauma group reported a symptom profile similar to the *DSM-IV* definition of PTSD, except that avoidance and emotional numbing symptoms formed two separate factors instead of being a single combined group. The one worst past trauma group reported a symptom profile more similar to that of the *DSM*-5 PTSD structure, with a separate factor for persistently altered negative emotions ("dysphoria"). The take-home message is that PTSD symptoms "hold together" better for people who actually have been exposed to traumatic stressors than others who have not, which suggests that traumatic exposure may play an important role in the development of PTSD that can be distinguished from the mere presence of PTSD-like symptoms as reported by people who have never been traumatized. Thus, the overall evidence suggests it is unlikely that true PTSD can occur without a traumatic stressor.

Understanding etiology: causes? Or risk and protective factors?

"Etiology," derived from the Greek word for "cause," is the study of the causes of diseases. Psychiatric disorders and psychosocial problems such as PTSD are "multi-determined"—that is, they are the product not of any single "cause" but instead of a complex interplay (Carlson, Dalenberg, & Muhtadie, 2008) of "nature," which is a person's inborn or acquired biological and psychological characteristics, and "nurture," which involves the influences in the physical and social environments that promote or interfere with healthy adaptation or illness, learning or disability, and growth or developmental delays. Regardless of the role played by exposure to traumatic stressors in PTSD, there is convincing scientific evidence that PTSD is caused in part by certain other "risk factors"—or at least that PTSD is more likely to develop when such "risk factors" have occurred.

The term "risk factor" is used because it is virtually impossible scientifically to isolate the exact "cause" of any phenomenon. As an example from the medical field, we know that infection by the human immunodeficiency virus (HIV) can lead to acquired immunodeficiency syndrome (AIDS); thus, HIV could be considered the "cause" of AIDS. However, not everyone who is exposed to HIV develops AIDS, and even among those who do contract AIDS, the timing of developing the illness, its symptoms, and the severity of the symptoms vary greatly from person to person and for the same person at different times. Several "risk factors" can hasten the onset of AIDS and increase its severity and lethality, particularly other conditions that compromise

the body's defenses against infection (the immune system)—for example, other sexually transmitted diseases such as syphilis or herpes. Thus, the "cause" of AIDS, which usually is described as contracting HIV through unprotected sex, needle use or sticks, infected blood transfusions, or mother-child transmission, is more complicated than this. In order to understand the full causes of AIDS and to prevent the disease, it is essential to know about risk factors that may "tip the balance" between having HIV and developing AIDS.

The analogy to PTSD is fairly direct. Exposure to traumatic stressors is the counterpart of exposure to HIV, leading to the risk of developing, respectively, PTSD or AIDS. Therefore, in order to understand, prevent, or treat PTSD, it is essential to know the risk factors that contribute to the development of PTSD and "tip the balance" in a positive or negative way when people are exposed to traumatic stressors. However, it is not enough to know what places a person at risk for a disease, because even when risk factors are present, some individuals still do not develop the disease or do so more slowly or with lesser severity. This suggests that some people may have a kind of "resistance" or even "invulnerability" to disease even when exposed to its causes or risks (Figure 3.1).

The positive side of disease prevention and treatment is that there are "protective factors" that reduce the likelihood of developing diseases. The most important protective factor is any condition or attribute that prevents (or reduces the likelihood) of a person encountering the active biological agent(s) (called "pathogens") that are involved in the disease. Thus, key protective factors for the prevention of AIDS include knowledge about risky behaviors (such as unprotected sex or sharing needles) and safer alternatives, societal and peer-group norms and relationships that support the use

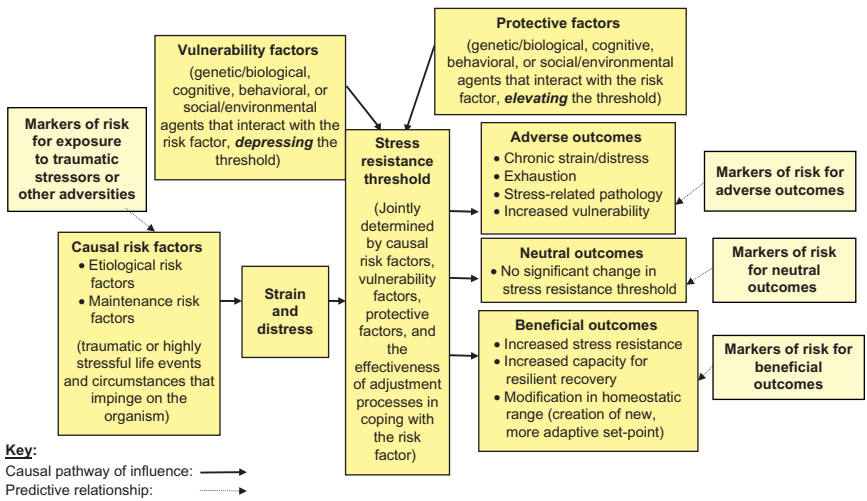

Figure 3.1 The role of risk and protective factors in the etiology of psychological "outcomes" such as PTSD.
Reprinted with permission of the author and publisher from Layne et al. (2008).

of safer alternative practices, and access to activities and practical tools that are necessary in order to engage in safer alternative practices. Other protective factors increase a person's resistance to the disease if infected, such as a particular gene (*CCL3L1*) that has been identified in HIV-resistant individuals, or timely access to prophylactic (preventive) and therapeutic medicines (such as antiretroviral drugs for HIV).

Here, again, there is a direct analogy to protective factors for PTSD. Just as the best way to prevent AIDS is to increase safe practices, PTSD is best prevented by reducing people's exposure to traumatic stressors. Although exposure to traumatic stressors cannot be entirely eliminated (just as it is impossible to absolutely ensure that accidental exposure to HIV will never happen), it is important to create conditions that reduce the risk of traumatic stressors (such as programs aimed at preventing violence or abuse; see Chapter 9). As with HIV, traumatic stressors also are less likely to be encountered when a person's family, peer-group, community, work or school, and societal support systems promote safety and discourage risky behaviors such as violence or actions that lead to serious accidents (such as drunk driving or driving without a seatbelt). In addition to social norms and support, there are protective factors that reduce the risk that a person will develop PTSD after encountering a traumatic stressor. These include biological protective factors (although, unlike HIV/AIDS, no specific genes have been identified that are protective against PTSD—but research is headed in that direction; see Chapter 6), as well as other person-specific protective factors, and social and environmental protective factors.

Thus, the best way to understand the "cause(s)" of PTSD is not to look for a single (or few) villains that can be defeated or enemies that can be eradicated, but instead to develop a broader understanding of how risk and protective factors lead to the posttraumatic trajectories (see Chapter 2) that result in either PTSD or freedom or recovery from PTSD. Figure 3.2 provides an overview of a "causal model" (i.e., the

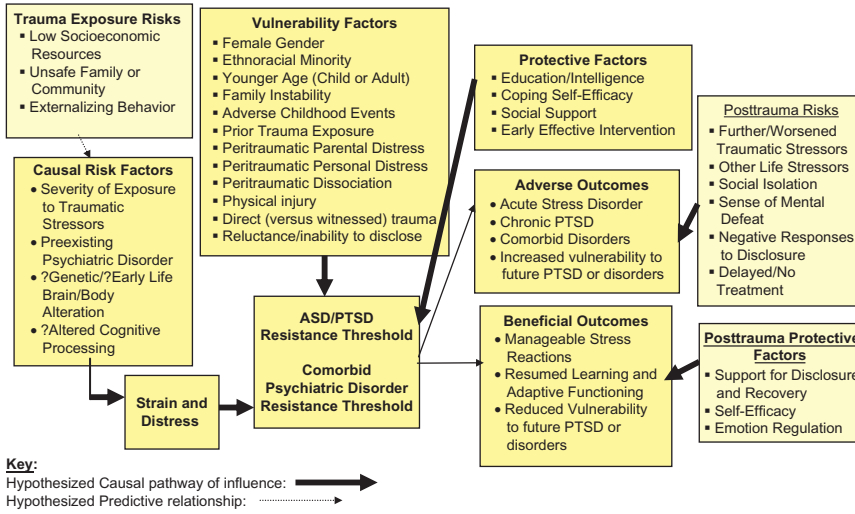

Figure 3.2 Factors influencing PTSD following stressful or traumatic life events.

theoretical connections among the potential "causes") of PTSD developed by Layne et al. (2008). Three types of risk factors are distinguished. The first are "causal risk factors," which are presumed to be directly responsible for a disease or problem (such as HIV with AIDS and exposure to traumatic stressors with PTSD). Causal risk factors have a direct negative effect on the individual biologically and psychologically, increasing the "strain" on the person's resources (e.g., depleting the immune system's capacities or leading to a psychological sense of hopelessness) and the "distress" experienced by the person (e.g., the physical illness symptoms of AIDS or the anxiety of PTSD).

"Vulnerability factors" are the second type of risk factor, including aspects of the individual or the environment that make a person more likely to either experience a causal risk factor or to develop the disease or problem after having experienced the causal risk factor. For AIDS, engaging in unprotected sex or needle use and having a compromised immune system are vulnerability factors. Several vulnerability factors for exposure to traumatic stressors and PTSD are discussed later in the chapter. An interesting bimodal vulnerability factor for PTSD—age—is highlighted in Box 3.3. Vulnerability factors often do not increase strain or distress, but instead contribute to disease or problems by decreasing resistance to the adverse outcomes.

Box 3.3 Age as a Risk or Protective Factor for PTSD

Age may be either a risk or protective factor for PTSD, depending on the stage of emotional, social, and cognitive development of a person exposed to a traumatic stressor. Chronological age appears to be less important in determining risk of PTSD than the developmental stage and the challenges facing the individual at that stage. In addition, at certain points in the life span, there are *transitions* between stages of biological and psychological development that temporarily shake up a person's familiar ways of handling stressors. The upheaval that inevitably occurs in developmental transitions (such as in early latency, adolescence, or midlife adulthood) increases both the risk of exposure to traumatic or other stressors (because of the inevitable change in routines and relationships) and of developing PTSD (due to the demand that simply traversing the developmental transition places on the coping capacities that are needed to recover from the impact of exposure to traumatic stressors). As each transition is completed, the individual gains additional social resources (e.g., the wider array of relationships that develops as a person expands his or her social network) and coping capacities (e.g., increased knowledge and skills that come with maturation, independence, and learning from life experiences). Yet, the transitions also are times of uncertainty and change, which can amplify the impact of a traumatic stressor and lead to increased vulnerability to PTSD regardless of the person's chronological age.

Consistent with this view that developmental transitions confer risk for PTSD, childhood generally is a period of less risk for PTSD than adolescence (Carlson et al., 2008). Childhood is a developmental period when the biological and

cognitive capacities needed for complete narrative (story-like) memories that shape our core beliefs about self/identity and the world/relationships are incomplete and rapidly evolving. Children also tend not to question beliefs and expectations that they hold at any particular moment, while adolescents are constantly reconsidering their view of themselves, relationships, and the future. In addition, children tend to both rely upon and receive more immediate help from primary caregivers than adolescents in coping with stressors. Hormonal changes, accelerated brain development, and increased social, educational, and personal independence and responsibility make adolescence an often tumultuous time in which stress reactivity commonly increases drastically (albeit temporarily). Adolescents also more often report exposure to interpersonal violence than younger children or adults (Boney-McCoy & Finkelhor, 1996; Kessler et al., 2005, 1995), and more than 20% of both girls and boys report new incidents of victimization by interpersonal violence in as short a time period as 15 months (Boney-McCoy & Finkelhor, 1996) and 33% report multiple forms of exposure to interpersonal violence ("polyvictimization," Finkelhor, Ormrod, & Turner, 2007).

Therefore, although children have less well-developed coping capacities, their relatively greater degree of developmental continuity compared to adolescents may actually protect them from developing PTSD—especially if they have reliable and emotionally attuned primary caregiving to support their recovery from exposure to stressors (traumatic and otherwise).

However, young children can be profoundly impacted emotionally and behaviorally when exposed to traumatic stressors (Briggs-Gowan et al., 2010; Briggs-Gowan, Carter, & Ford; 2012; Grasso, Greene, & Ford, 2013). This is particularly true when traumatic stressors occur on an ongoing basis or as a series of events that occur over a lengthy period of time. Such chronic or cumulative traumatic exposure (see Box 3.4) can place a young child at high risk for PTSD or related psychosocial problems as a result of overwhelming their (and their caregivers') capacities to cope and also lead them to develop core beliefs and expectations that are tainted by anxiety, hypervigilance, and despair. Young children are particularly vulnerable to PTSD when they experience chronic or recurrent traumatic stressors at the same time that major disruptions are occurring in their relationships with primary caregivers (such as sexual or physical abuse or severe neglect), placing them at risk for severe emotional and behavioral problems, including PTSD (D'Andrea et al., 2012; English, Graham, Litrownik, Everson, & Bangdiwala, 2005; Ford et al., 2000; Kaplow, Dodge, Amaya-Jackson, & Saxe, 2005; Kaplow & Widom, 2007; Keiley, Bates, Dodge, & Pettit, 2001).

Transitional vulnerability may play a role in adults' vulnerability to PTSD as well. Adults are more likely to develop PTSD if they are exposed to psychological trauma in middle adulthood than either as young adults or in older adulthood (Ford, Adams, & Dailey, 2007). For example, in the National Comorbidity Study-Replication, the prevalence of PTSD among adults rose from 6% among 18–29 year olds (which was very similar to the prevalence among teenagers,

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McLaughlin et al., 2013) to 8% among 30–44 year olds, and 9% among 45–59 year olds (Kessler et al., 2005). Two in three cases of PTSD had an initial onset between age 15 and 53, indicating that late adolescence and midlife adulthood appear to be particularly high-risk developmental periods for PTSD. Beyond the turmoil of adolescence, in middle adulthood additional hormonal changes (such as menopause and decreasing levels of testosterone) and an accumulation of work, family, and financial responsibilities (such as job promotion and midlife career challenges and changes, raising children and caring for aging parents, and increasing costs due to providing for a growing family's needs and children's educations and health care) also may increase stress reactivity and reduce coping resources.

Culture and ethnicity also may play a role in influencing the risk of PTSD for persons of different ages. Norris, Friedman, and Watson (2002) surveyed adults following a hurricane in the United States, a different hurricane in Mexico, and a flood in Poland. In the United States sample, midlife adults were most likely to report PTSD symptoms; in Mexico, younger adults were most likely to report PTSD symptoms; and in Poland, older adults were most likely to report PTSD symptoms. Thus, there was no single relationship between age and PTSD symptoms, suggesting that the different ethnoracial backgrounds, cultures, and social support resources of the three cohorts of disaster survivors may have led to the differences reported by adults of different ages within each cohort resist or recover from PTSD symptoms.

Third, there are "outcome risk factors," which are factors that increase the likelihood that adverse outcomes will be severe if they occur. Developing an increasing viral "load" (level of HIV in the blood system) or repeated unprotected exposure to HIV are examples of HIV/AIDS outcome risk factors. Later in this chapter several PTSD outcome risk factors (risk factors associated with more severe and prolonged PTSD symptoms) identified by scientific research are discussed.

Protective factors counteract or counterbalance the effects of risk factors by increasing the person's ability to "resist" (prevent) or to be "resilient" in coping with and "recovering" from adverse outcomes. Protective factors may indirectly increase beneficial outcomes, but their primary function is to reduce the resistance threshold—that is, to increase the likelihood that the person will successfully access biological or psychosocial resources that reduce exposure to causal risk factors, vulnerability, and the development of severe disease or problems.

Causal risk factors for PTSD

One causal risk factor for PTSD has been strongly established by scientific research: the "dose" or severity of exposure to traumatic stressors. We next consider the one

necessary—but not sufficient (see Box 3.1)—causal risk factor for PTSD: exposure to traumatic stressor(s). Then we explore the evidence regarding three other types of potential causal risk factors for PTSD that were identified in a review of 54 prospective longitudinal studies of risk factors for PTSD: psychiatric disorders, and associated coping and personality styles and family adversities; vulnerability in brain/body stress response systems related to genetics or early life experiences; and limitations or deficits in cognitive abilities (DiGangi et al., 2013).

Degree or severity of exposure to traumatic stressor(s)

The causal risk factor for PTSD that has been most consistently documented in scientific research is the *degree of exposure to traumatic stressor(s)* (Brewin, Andrews, & Valentine, 2000; Carlson et al., 2008; Ford, 2009; Norris & Slone, 2007; Ozer, Best, Lipsey, & Weiss, 2003; Vogt, King, & King, 2007). This has been termed the "dose response relationship" because the greater the "dose" (in this case of exposure to harm or threat), the greater the likelihood that the exposed person will subsequently suffer PTSD. There is no *a priori* way to define or measure "degree of exposure" to a traumatic stressor. It is not as simple as saying that certain events seem "worse," "more horrible," or "more painful" than others. Exactly how the "dose" or "event exposure" is defined and measured varies greatly, however (Vogt et al., 2007). It may be defined as the severity of the threat to the person's (or someone else's) life (Ozer et al., 2003). It may be defined as the extent of physical injury (Acierno, Resnick, Kilpatrick, Saunders, & Best, 1999) or of the cruelty or violence or destruction inflicted upon affected persons (Kessler et al., 1995). It also may be defined as the extent of destruction to the family, home, or community in the wake of a natural or humanmade disaster or mass-casualty incident (Norris & Slone, 2007). The survivor's subjective response of terror or horror (Brewin et al., 2000; Bryant, 2005) or dissociation (Ozer et al., 2003) at the time of the event or soon afterward (the "peritraumatic" period) also have been used to operationalize trauma exposure and found to be predictive of developing PTSD (see Vulnerability Factors in this chapter).

Aspects of traumatic stressors have been identified and shown to be associated with more severe immediate stress reactions, which in turn are associated with a greater likelihood of developing PTSD or other psychiatric disorders (Brewin et al., 2000, 2002). These include:

- intentional physical or sexual violence perpetrated by another person or group (such as domestic, war, or community violence; terrorist attacks; or torture);
- betrayal by a person or organization responsible for protecting the safety and rights of vulnerable individuals (such as physical or sexual abuse by a caregiver or priest);
- violation of victims' bodies or selves or homes by extreme violence or destruction (such as war atrocities, rape, or destruction of home and community in a disaster);
- coercion used to destroy people's self-respect and will to resist (such as combined physical and emotional abuse, domestic violence, or torture; see Chapter 11);
- prolonged complete isolation from human contact and social interaction (such as solitary confinement of prisoners of war, kidnapping victims, or abused children);

- Lengthy duration or numerous repetitions of exposure to traumatic stressors, or of uncertainty in the face of imminent exposure (such as chronic abuse, violence, or premature deaths, or living in a violent family, war-zone, or disaster-prone area; Boxes 3.4 and 3.5).

Box 3.4 Cumulative Traumatic Stress: Retraumatization, Childhood Adversity, and Polyvictimization

When people experience a series of stressors, simultaneously or one at a time over a period of days, weeks, months, or years, there is a "cumulative" impact (Grasso et al., 2013), similar to that of a boxer who is hit by a rapid sequence of blows and by many blows over the course of a long fight. People who have already been exposed to a traumatic stressor tend to be at risk for exposure to another traumatic stressors. Three distinct lines of theory and research have evolved in an attempt to understand cumulative traumatic stress: retraumatization, childhood adversity, and polyvictimization.

Retraumatization. There is substantial evidence that being multiply exposed to traumatic stressors places an individual at increasing risk of developing PTSD and related psychosocial and physical health problems in childhood and across the life span (Follette & Vijay, 2008). However, exactly what constitutes retraumatization has not been rigorously defined. There is no specified period of time or context in which the subsequent exposure to additional psychological trauma(s) must occur in order for retraumatization to have occurred. Once the initial, or "index," psychological trauma has occurred, any subsequent exposure to traumatic stressors may result in retraumatization. Retraumatization can occur regardless of whether the environmental or contextual factors of the first traumatic exposure are different at the time of the later traumatic exposures or remain consistent. Thus, if the first traumatic experience is childhood sexual abuse (CSA), retraumatization can occur through exposure to very different types of traumatic stressors, such as a natural disaster, war, or interpersonal violence (Whitfield, Anda, Dube, & Felitti, 2003). Whether different combinations (or timings) of index and subsequent traumatic stressors have different causal or vulnerability effects in relation to PTSD has not been established by research.

In some cases the term *retraumatization* has been used to describe acute exacerbations of PTSD-related distress by reminders of past psychological traumas. For example, involvement in the judicial system can prove retraumatizing to crime victims when they are forced to recount their experience or are mandated to testify in trials. In such cases, the person is not exposed to additional psychological trauma but experiences increased posttraumatic distress as a result of encountering reminders of the past traumatic stressors. It is important to distinguish between this usage of the term *retraumatization* from repeated exposure to traumatic stressors usage because, while the results may be similar, the second case does *not* actually involve exposure to traumatic stressors. If this difference is not carefully noted, experiences or events that are stressful but not traumatic may be misconstrued as being traumatic stressors. Such confusion could lead to

the false conclusion that stressful experiences such as court proceedings inevitably are traumatic stressors, as opposed to being highly stressful.

Research has shown that repeated exposures to the same or different psychological traumas is associated with an increased risk of developing PTSD and more severe PTSD symptoms compared to a single exposure to a traumatic event (Follette and Vijay, 2008). Over the course of a lifetime, 67% of women who have been victimized by interpersonal violence will report at least one additional incident of victimization (Cloitre, Cohen, & Koenen, 2006). The factors associated with increased risk for sexual revictimization/retraumatization include having a history of child or adolescent sexual abuse and the characteristics of previous victimization (the severity and frequency of those abuses, the age at which the abuse began, the nature of the abusive sexual contact, the victim's relationship with the abuser, the duration and number of exposures to victimization; Follette and Vijay, 2008). A history of CSA is a risk factor for sexual victimization as an adult: women with a history of CSA have been found to be twice as likely as other women to be assaulted in adulthood (Nishith, Mechanic, & Resick, 2000), and men with a CSA history are 5.5 times more likely than other men to be assaulted as an adult (Classen, Palesh, & Aggarwal, 2005). The combination of CSA and adult victimization is a key risk factor for adult PTSD (Nishith et al., 2000). Follette and Vijay (2008, pp. 588–589) notes:

> *Intrapersonal variables, which include psychological disorders, substance use, and sexual practices, can also put an individual at risk for retraumatization by affecting the ways they are able to respond to subsequent stressful events. … For example, an individual who utilizes dissociation as a form of coping with posttraumatic stress problems is at significantly greater risk to experience another traumatic event.*

Although individual risk factors are an important consideration, this should not be misinterpreted as "blaming the victim." In most such cases, the traumatized individual does not intentionally cause the occurrence of additional traumas, but she or he may be less careful or more prone to taking risks as a result of experiencing posttraumatic distress or dissociation—or the person may simply live in circumstances that are unavoidably dangerous (such as violent communities or families). Environmental or contextual factors also seem to increase risk of retraumatization as well. Factors such as poverty or neglect can increase the likelihood of a person being exposed to multiple potentially traumatic events, including childhood abuse and family and community violence. Poverty may also be a risk factor for retraumatization among people exposed to disasters, because they are more likely than more affluent people to lose (or simply not have access to) vital resources that facilitate postdisaster recovery.

An example of the kind of study needed in order to more definitively establish whether and how repeated exposure to traumatic stressors leads to a greater likelihood or severity of PTSD is the 10-year follow-up conducted by Breslau, Peterson, and Schultz (2008) of a survey of a sample of generally healthy young adults in a midwestern U.S. health maintenance organization. They found that

(*Continued*)

Box 3.4 Continued

retraumatization (i.e., having a subsequent exposure to a traumatic stressor after having had a prior experience of psychological trauma) was associated with an increase in the risk of PTSD only if PTSD had occurred following the first traumatic exposure. Thus, it was not simply the cumulative impact of repeated exposure to traumatic stressors but that "retraumatization" *in combination with vulnerability to PTSD* that resulted in the increased risk of PTSD that has been associated with retraumatization.

Adverse Childhood Experiences (ACEs). The ACEs Study was a large-scale epidemiological (see Chapter 4) investigation that has gained professional and public recognition by demonstrating that adversities experienced in childhood may substantially increase the risk of many physical and mental health problems in adulthood (Anda, Butchart, Felitti, & Brown, 2010; Felitti et al., 1998). The study originally was based on information obtained from more than 17,000 young and midlife adults who completed a routine health screening while receiving health care services in the California Kaiser Permanente Health Maintenance Organization. (80% white; 54% female; average age 57 years old). Participants completed a 10-item yes-or-no questionnaire telling whether they had experienced each of the following ACEs before the age of 18: physical abuse, verbal abuse, sexual abuse, physical neglect, emotional neglect, an alcoholic parent, witnessing domestic violence of their mother, a family member jailed, a mentally ill family member, and loss of a parent due to divorce, death, or separation.

Each participant was assigned a score based on the number of ACE categories endorsed. Nearly two-thirds of the participants reported at least one ACE, and one in six people experienced four or more ACEs. Women were 50% more likely than men to have experienced five or more ACE categories. Disturbingly high proportions reported traumatic adversity in childhood: 18% of men and 25% of women reported CSA; 22% of men and 20% of women reported childhood physical abuse; 12% of men and 15% of women had witnessed maternal battering. Almost 90% of those who reported any one ACE reported at least one additional ACE, so more than half experienced the cumulative impact of multiple childhood adversities. Three times as many (65%) individuals who had witnessed domestic violence (versus 23% of those not exposed to domestic violence in childhood) also had an alcoholic parent while growing up. Similarly, more than four times as many (81%) people who reported emotional abuse (versus 20% who did not) also reported physical abuse as a child.

Therefore, the ACEs study explored the relationship of *the number of ACEs* experienced to a wide variety of adverse outcomes in adulthood. The result was a "dose-response" relationship where *every additional ACE* reported increased the risk of serious physical and mental health problems in adulthood (including smoking, obesity, physical inactivity, depression, suicide attempts, alcoholism, drug abuse, sexual promiscuity, sexually transmitted disease) and also major medical illnesses (heart disease, cancer, stroke, chronic bronchitis, COPD, diabetes,

hepatitis, and skeletal fractures). Some of the more dramatic and serious public health findings of the ACEs study included:

- People reporting five or more ACEs were *10 times* as likely to have attempted suicide, and those reporting seven or more ACEs were *30 times* more likely. After accounting for other risk factors for suicide (alcoholism, depression, street drug use), people with ACE scores of 7 or higher were still *17 times* more likely to have attempted suicide than people reporting no ACEs.
- Besides the occurrence of psychiatric disorders, their complexity and severity (as measured by the number of psychiatric medications prescribed) were also related to the number of ACEs.
- The risk of developing often fatal or extremely severe medical illnesses, such as heart disease, lung cancer, or autoimmune diseases, was directly related to the number of ACEs reported.
- Subsequently, the 10 ACEs questions were included by the Centers for Disease Control (Bynum et al., 2010) in a random sampling of the adult populations of five states in the United States, and the original findings from the Kaiser Permanente study were replicated; with each additional ACE, physical and mental health risk behaviors and illnesses were several times more likely to occur in childhood or adulthood.

Polyvictimization. The National Survey of Children's Exposure to Violence (NatSCEV) interviewed 2030 10- to 17-year-old children and adolescents who represented a cross section of all youth in the United States. Victimization experienced in the past year was measured using the Juvenile Victimization Questionnaire (JVQ) to survey 34 different types of victimization. Although noninterpersonal events (e.g., accident, natural disaster) could be viewed as "victimizing" survivors or witnesses, in these studies victimization was defined as including only interpersonal events (e.g., physical or sexual assault, abuse, or witnessed violence). Youth were defined as having been "polyvictimized" if they endorsed exposure to four or more victimization types on the JVQ. This is similar to the ACEs score in that it is based on the number of different *types* of victimization rather than the number of times or chronicity or severity of any single type of victimization. However, the NatSCEV assessed a much larger number of more specifically defined types of victimization than the 10 ACEs. Victimization also was assessed based only on the past year, rather than as recalled several decades later, which would be expected to increase the accuracy of the respondent's recall of victimization. Despite these definitional and procedural differences, the findings regarding polyvictimization from the NatSCEV were remarkably similar to the findings from the ACEs studies.

Nearly one in four youths surveyed who had experienced any victimization were polyvictims. Polyvictims tended to be older and male, although a large number of girls also were polyvictims (Finkelhor et al., 2005). Polyvictims were on average four times more likely to be revictimized in the year following the study, and nearly half of polyvictims at baseline were categorized as polyvictims again in the second year (Finkelhor et al., 2007). While referred to as "persistent

(*Continued*)

Box 3.4 Continued

polyvictims" in the NatSCEV, these youths definitely could be considered likely to have experienced retraumatization. In addition, consistent with the ACEs studies' results, the persistently polyvictimized youths tended to live in families in which violence occurred, to have experienced childhood maltreatment, and to have had family members who abused alcohol and drugs or a parent who was unemployed. Thus, persistent polyvictimization of children involves not only exposure to multiple types of potentially traumatizing victimization but also living in a family context of maltreatment and relational adversity. Retraumatized polyvictim children encounter adversity across multiple contexts where violence is pervasive, inflicted by a variety of perpetrators: physically and emotionally maltreatment by caregivers; bullying by peers; sexual abuse by caregivers, mentors, or acquaintances; and witnessing a host of violent and traumatic incidents in the home, school, and community (Cuevas, Finkelhor, Clifford, Ormrod, & Turner, 2010). On the other hand, the more friends a youth has, the lower the risk of persistent polyvictimization (Finkelhor et al., 2007)—except if the friends are involved in risky behaviors or lifestyles, such as juvenile delinquency (Cuevas, Finkelhor, Turner, & Ormrod, 2007; Ford, Elhai, Connor, & Frueh, 2010). The latter findings echo the results of studies showing that social support can be either a protective or risk factor, depending on its nature (see Box 3.5).

Ultimately, four pathways to polyvictimization were identified by the NatSCEV: (i) residing in a dangerous community, (ii) living in a dangerous family, (iii) living in a nondangerous but chaotic and multiproblem family environment, and (iv) having emotional problems that lead to risky behavior, foster antagonism, and increase the likelihood of being victimized (Finkelhor, Ormrod, Turner, & Holt, 2009). The first (dangerous community) pathway to polyvictimization was most common among children who became polyvictims before 10 years of age, and these children most often became polyvictims at about the age of entry to elementary school (i.e., when their contact with the larger community sharply expanded). The other age at which many youth became polyvictims was at about the age of entry into high school, which may be a vulnerable period both due to the turmoil of adolescence and contact with an even broader spectrum of peers that occurs in the typically large and diverse high school context. The vulnerability that occurs at those two key points of developmental transition is consistent with the research evidence showing that such transitions are risky times for PTSD (see Box 3.3).

Box 3.5 Pathways from Victimization to Revictimization

As many as three in four women who report a history of having been victimized by CSA also report experiencing sexual assault in adulthood (Roodman & Clum, 2001), and they are two to three times more likely to report an adult sexual assault

than other women (Classen et al., 2005). Many factors have been hypothesized to account for this high risk of revictimization, including early developmental production of sex hormones, increased sexual activity and engaging in risky sexual behavior, and attempting to use sex to cope with depression or other forms of psychological distress (Noll, Trickett, & Putnam, 2003; Orcutt, Cooper, & Garcia, 2005). However, sexual abuse often occurs in combination with other forms of childhood maltreatment (such as physical or emotional abuse or neglect), each of which has been shown to be associated with adult revictimization (D'Andrea et al., 2012). For example, a prospective study of high-risk children in adulthood found that not only CSA but also physical abuse and neglect predicted physical and sexual revictimization (Widom, DuMont, & Czaja, 2007).

A study with young women found that the relationship between CSA and revictimization over a 6-year period of time could be explained in part by a tendency toward depression, anxiety, and attempts to manage these negative emotions and beliefs by using sex (Orcutt et al. 2005). Pervasive negative emotional states and beliefs are a core PTSD symptom that is consistently associated with all forms of childhood maltreatment (D'Andrea et al., 2012). Adolescents may be at risk for sexual revictimization due to a developmentally normative tendency to use sex to manage these types of distress. For example, a study with a community sample of adolescents who reported their reasons for engaging in sex immediately after sexual encounters found that they reported using sex to cope with negative emotions if they felt a sense of low self-esteem or anxiety, and for boys there was indirect evidence that using sex to manage negative emotions provided a buffer against feeling depressed (Dawson, Shih, de Moor, & Shrier, 2008).

These findings raise the question of whether adolescents and young adults who have been victimized in childhood actually are trapped by posttraumatic distress in ways that might lead them to be vulnerable to revictimization (see Box 3.4). Therefore, a study was done with college women who reported whether they had experienced childhood sexual (CSA), physical (CPA), or emotional (CEA) abuse and then approximately 2 months later reported whether they had been sexually assaulted in that time period (ASA) (Miron & Orcutt, 2014). One in nine of the women reported a sexual assault (11%), and there appeared to be several potential pathways from the childhood abuse to revictimization. EA was directly predictive of ASA. Women who had experienced CSA and also sexual abuse in adolescence also were at risk for ASA. And women who had experienced CPA were at risk for ASA if they were subsequently sexually abused in adolescence and felt depressed in adulthood. In addition, CPA victims also were at risk for ASA even if they had never been previously sexually assaulted but if they used sex to manage negative emotions and were likely to agree to have sex after a casual encounter. The four pathways to sexual revictimization identified in this study may be only a subset of the ways in which survivors of childhood traumatic stressors are at risk for further traumatization. However, despite the brief time frame in which adult victimization was assessed (on average 2 months), the

(*Continued*)

Box 3.5 Continued

findings demonstrate that revictimization is a prevalent problem and point to the role that PTSD—in the form of negative emotions and beliefs, and risky attempts to manage this distress—may play when sexual or physical abuse has occurred in the life of a child or youth.

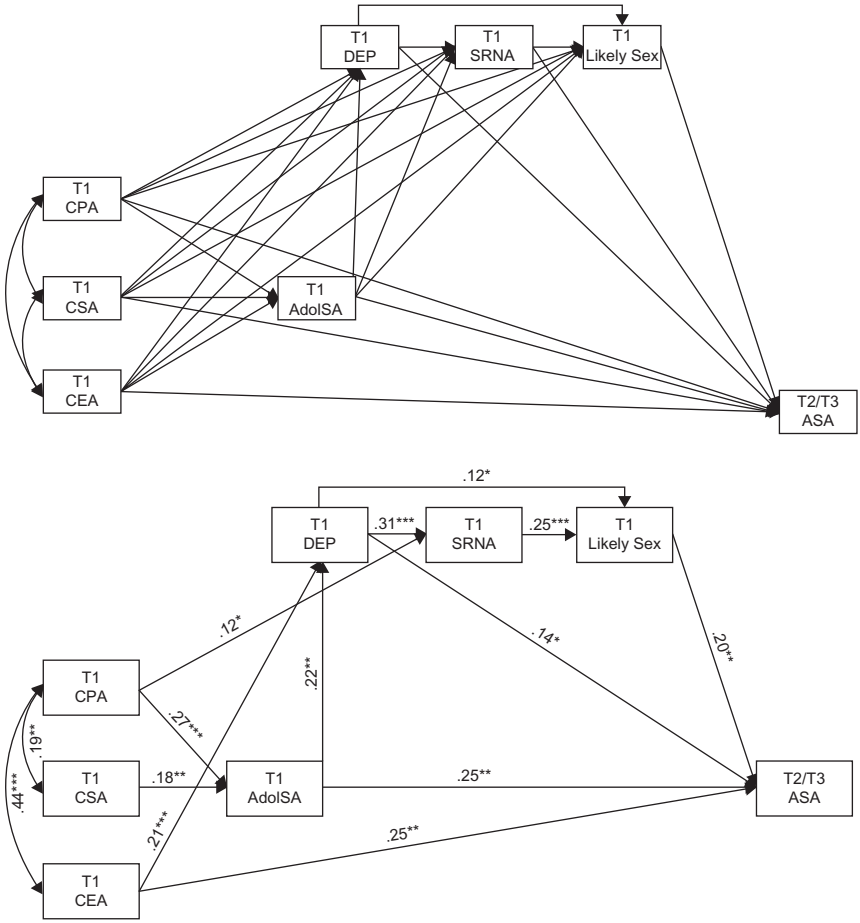

Figure for Box 3.5 reprinted with permission of the author and publisher from Miron and Orcutt (2014).

These "event" or "exposure" risk factors have several common denominators despite their many differences. Actual or imminent severe physical injury or "violation" (by sexual assault or disfigurement or dismemberment) is explicit or implicit in each exposure causal risk factor. In addition, several exposure causal risk factors involve witnessing or suffering the loss of primary relationship(s) due to someone

else's untimely death or experiencing one's own (or witnessing someone else's) imminent death. The reality or imminent threat of death is likely to elicit the biological survival reaction that appears to become fixed and chronic in PTSD (see Chapter 6), and this biological shift could explain how the threat of death or witnessing the death or near death of someone else can have a psychologically "traumatic" impact.

Another common denominator among the exposure causal risk factors is the terror elicited by extreme violence such as war or torture, and the horror elicited by extreme "violation," such as sexual assault or abuse (D'Andrea, Ford, Stolbach, Spinazzola & van der Kolk, 2012). Although individuals' subjective reactions will differ even to what seem to be objectively severely terrifying or horrifying events, a number of scientific studies have shown that more severe violence or violation that is intentionally inflicted on victim(s) is associated with a higher likelihood of directly disclosed or indirectly expressed terror and horror (Marx, Forsyth, Gallup, Fuse, & Lexington, 2008; Pat-Horenczyk, 2008).

A third common denominator among exposure causal risk factors is the extent to which they are "sudden, uncontrollable, and have an extremely negative valence" (Carlson et al., 2008). To this could be added the unpredictability of the stressor (Foa, Zinbarg, & Rothbaum, 1992). A sudden and unpredictable stressor is likely to occur too rapidly and with too little warning for the affected person to "act to either physically protect himself/herself from harm or to psychologically prepare for a negative outcome" (Carlson et al., 2008). If the stressor also is largely or completely beyond the person's ability to control what happens or influence the outcome, the combination of *suddenness, unpredictability, and uncontrollability* of the event is likely to take the affected person by surprise, which may result in a sense of shock, disbelief, and confusion. Such events provide minimal opportunity to defend oneself physically or psychologically, leading to greater risk of being unprotected and injured (and to be unable to protect others from being injured).

When events happen rapidly and without warning or an opportunity to influence their course or outcome, it is more difficult to think clearly because the person has little or no preparation to provide a foundation for a thoughtful appraisal and even less time to think analytically about what is happening, what it means, and what to do. Not surprisingly, therefore, people with PTSD often describe feeling particularly distressed—frightened, helpless, guilty, ashamed—by intrusive memories of traumatic events because they believe that they were powerless to prevent or stop terrible things from happening ("I never saw it coming, and when it did, I couldn't stop it") or that they should have been vigilant and prepared but had failed to do so ("I should have known it was going to happen and been ready so I wouldn't have been so helpless when it happened").

Preexisting psychiatric disorders

A second type of potential causal risk factor is a *preexisting psychiatric illness*. Adults with chronic and severe mood disorders (such as major depression or bipolar disorder), anxiety disorders (such as obsessive-compulsive, panic, or generalized anxiety

disorder), psychotic disorders (such as schizophrenia or schizoaffective disorder), dissociative disorders, eating disorders (such as anorexia or bulimia nervosa), or personality disorders (such as borderline or paranoid personality disorder) often report a history of exposure to psychological trauma and both past (14–66%) and current (i.e., 12–35%) PTSD (see Chapter 4) (Mueser, Essock, Haines, Wolfe, & Xie, 2004; Mueser et al., 2001). Adults with psychiatric disorders report substantial recent exposure to victimization traumas (such as abuse or domestic violence; Goodman et al., 2001; Neria, Bromet, Sievers, Lavelle, & Fochtmann, 2002; Sells, Rowe, Fisk, & Davidson, 2003), but the most common time of first victimization is in childhood (Follette and Vijay, 2008; Neria et al., 2002).

Not only is PTSD prevalent among adults with psychiatric disorders (see Chapter 4), but it also is associated with more severe symptoms of those disorders and worse impairment in relationships and work than is found when adults with psychiatric disorders do not also have PTSD. For example, Mueser et al. (2004) reported that 16% of patients with psychiatric disorders in a study of three vocational rehabilitation programs met research criteria for current PTSD, and those with PTSD had more severe psychiatric symptoms, worse self-reported health, lower self-esteem, lower subjective quality of life, and poorer employment outcomes (lower rates of competitive work, fewer hours worked, fewer wages earned) *even when these patients had received the most effective rehabilitation intervention*. Similarly, a study of more than 1000 depressed adults who had been treated in inpatient psychiatric programs showed that PTSD was associated with more severe hostility and depression at discharge from the hospital and more discharges against medical advice (Holtzheimer, Russo, Zatzick, Bundy, & Roy-Byrne, 2005).

Psychiatric disorders also may be risk factors for PTSD even when the disorders are relatively mild and not severe mental illnesses. For example, Sandweiss et al. (2011) showed that military personnel injured during deployment were 2.5 times as likely to screen positive for PTSD if they had any psychiatric disorder prior to the injury; the severity of the injury also was correlated with the risk of PTSD, but this had only one-tenth as strong a relationship to PTSD as did prior psychiatric disorder. A prospective longitudinal study of children followed over 3 decades into adulthood found that all new cases of PTSD and 93.5% of participants who had ever had PTSD by age 26 also had a psychiatric disorder (most of which began by age 15) between ages 11 and 21 years old (Koenen et al., 2008). This study systematically assessed psychiatric disorders periodically in childhood and adolescence, thus identifying many mild to moderate severity cases that would never have been detected under ordinary circumstances (because most of those individuals would not have sought mental health treatment). While these results do not definitely establish that psychiatric disorders contribute to causing PTSD, they strongly suggest that having a psychiatric disorder makes a person vulnerable to developing PTSD.

One potential explanation for these findings is that PTSD may precede and exacerbate other psychiatric disorders. Consistent with this evidence, patients with psychiatric disorders who report having experienced interpersonal psychological traumas such as abuse in childhood often report particularly severe hallucinations, paranoia, mania, depression, severe anxiety, suicidality, hostility, suspiciousness, and dissociation

(Leverich et al., 2001; Lysaker, Meyer, Evans, Clements, & Marks, 2001; Lysaker, Nees, Lancaster, & Davis, 2004). Perkonigg, Kessler, Storz, and Wittchen (2000) also found that agoraphobia, dysthymic disorder, generalized anxiety disorder, and somatoform disorder were more likely to occur after PTSD had occurred.

However, another possibility is that the symptoms of psychiatric disorders may make people vulnerable to PTSD or may even serve as a causal risk factor and contribute to causing PTSD. Perkonigg et al. (2000) found that several psychiatric disorders *preceded* first exposure to a traumatic stressor and PTSD in most (64–71%) cases, including specific phobia, social phobia, and somatoform disorder. These disorders also increased the likelihood that PTSD would occur following exposure to a traumatic stressor, although major depression was the best predictor of PTSD after exposure to a traumatic stressor, increasing the risk of developing PTSD more than 23 times. Substance use disorders were particularly likely to occur before exposure to a traumatic stressor, but they did not to lead to an increased risk of PTSD (Perkonigg et al., 2000). A number of studies have shown that emotional and behavioral difficulties that are commonly found in psychiatric disorders—anxiety, anger, dysphoria, and rumination (persistent nagging worry or distress)—are risk factors for PTSD (DiGangi et al., 2013). Several other studies have found that a family history of emotional or behavioral problems, which place offspring at risk for psychiatric disorders, are risk factors for PTSD (Copeland, Keeler, Angold, & Costello, 2007; Inslicht et al., 2010; Koenen, Moffitt, Poulton, Martin, & Caspi, 2007). Thus, psychiatric problems both on an individual and intergenerational (see Chapter 2) level may increase trauma-exposed persons' risk of developing PTSD.

How might a psychiatric disorder lead a person to be vulnerable to PTSD? A study of 500 male military veterans who had varying degrees of exposure to a natural disaster (Hurricane Katrina) found that those with PTSD were more likely than others to have a psychiatric illness that was diagnosed before the disaster (Constans et al., 2012). After the disaster, veterans who developed PTSD also were more likely than others to endorse trauma-related beliefs (see Altered Cognitive Processes in this chapter). Having trauma-related beliefs also was more strongly related to PTSD than the extent of the individual's exposure to traumatic stressors or damage to his home during the disaster or his social support after the disaster. A trajectory was identified leading from preexisting psychiatric illness to trauma-related beliefs and from those beliefs to PTSD, although the beliefs did not fully account for the relationship between psychiatric illness and postdisaster PTSD. Thus, one possible pathway from a preexisting psychiatric disorder to PTSD may be fearful beliefs that, if apparently confirmed by a traumatic event, could develop into persistent PTSD symptoms such as nightmares, avoidance, or hypervigilance.

The complicated relationship between other psychiatric disorders and PTSD is illustrated by studies that suggest that psychosis may increase the risk of PTSD and that exposure to traumatic stressors increases the risk of psychosis (Morrison, Frame, & Larkin, 2003). As an example of the first point, Shaw, McFarlane, Bookless, and Air (2002) conducted interviews with patients hospitalized for psychotic disorders and concluded that psychotic symptoms were sufficiently distressing to potentially lead to a "post-psychosis PTSD." Whether the psychotic symptoms indeed were

sufficiently distressing to lead to the development of PTSD symptoms was not established. It is possible that psychosis may contribute to PTSD instead by increasing the distress associated with PTSD symptoms that preexisted the psychotic disorder and were due to an actual traumatic stressor that had not been identified—that is, a delayed form of PTSD that psychotic symptoms did not cause but that may have been exacerbated by the psychosis. It also is possible that symptoms co-occurring with psychoses that are thought to represent PTSD actually are symptoms of other disorders such as depression. These two possibilities are illustrated by the findings of a study of adults in pharmacotherapy studies who were diagnosed with depression and reported substantial PTSD symptoms despite not reporting any past exposure to traumatic stressors (Bodkin, Pope, Detke, & Hudson, 2007). The "PTSD" symptoms may have been due to traumatic stressors that they did not recall or chose not to report, or to their depression rather than to PTSD.

Indirect support for the hypothesis that psychiatric disorders may serve as a causal risk factor for PTSD may be found by comparing the results of neuroimaging (brain scan) studies for PTSD with those for other psychiatric disorders such as major depression (Gotlib & Hamilton, 2008), bipolar disorder (Strakowski, Delbello, & Adler, 2005), schizophrenia (Brunet-Gouet & Decety, 2006), obsessive-compulsive disorder (Remijnse, van den Heuvel, & Veltman, 2005), dissociative disorders (Peres, Moreira-Almeida, Caixeta, Leao, & Newberg, 2012), and personality disorders (Reinders et al., 2014). While there are numerous specific differences in the integrity and size and patterns of neural activation in different areas in the brain across these disorders, a consistent finding is impaired activation in the brain areas involved in regulating emotions and making executive decisions (such as the medial and dorsolateral prefrontal cortices) and abnormal size or neural activity patterns in the areas of the brain associated with stress reactivity and emotional distress (such as the amygdala in the limbic system), screening, and organizing perceptual and cognitive information (such as the thalamus, striatum, and hippocampus). As is discussed in greater detail in Chapter 6, these brain areas and psychological functions have been found to be altered or impaired in PTSD.

A study with patients diagnosed with dissociative identity disorder (formerly called multiple personality disorder) shed further light on the possibility that altered patterns of brain activation may underlie both PTSD and other psychiatric disorders (Reinders et al., 2014). When patients were in a state of anxiety ("hyperaroused"), their brains showed high levels of stress response activation and low levels of emotion regulation and executive functioning activation, directly paralleling what occurs in PTSD. However, when they were emotionally and mentally shut down ("hyperaroused") their brains had almost the opposite pattern of activation—directly parallel to neuroimaging findings for the dissociative subtype of PTSD (Lanius et al., 2010).

Thus, it is possible that the fundamental biological and psychological capacities that are compromised in PTSD may already have been compromised by preexisting psychiatric disorders. However, preexisting psychiatric disorders could not be the primary cause of PTSD, because most individuals who develop PTSD, particularly when it begins within a few weeks or months after exposure to a traumatic stressor, do not have a preexisting psychiatric disorder (particularly children (Copeland et al., 2007)

but also including adults (Kessler et al., 2005)). PTSD may precede other psychiatric disorders. Therefore, the alterations in brain structure and activity observed in psychiatric disorders may be a prior causal risk or vulnerability factor for many psychiatric disorders—including but not limited to PTSD. It may not be psychiatric disorders per se but preexisting alterations in brain activation that underlie the psychiatric disorders that contribute to the etiology of PTSD. Given the differences in specific symptoms and specific brain alterations that exist between psychiatric disorders, including PTSD, it remains necessary to determine what other risk or vulnerability factors lead to specific brain alterations and symptoms that distinguish each psychiatric disorder (including PTSD) from all others.

Currently, the evidence for psychiatric disorders as risk factors for PTSD is strong in terms of a vulnerability factor (because people with psychiatric disorders more often report both exposure to traumatic stressors and PTSD than people with no psychiatric disorder) but at best preliminary in terms of psychiatric disorder symptoms serving as a causal risk factor (because it is not clear that psychiatric symptoms either can serve as a traumatic stressor or involve alterations in brain structure or function that may subsequently lead to PTSD). It also is not known whether more basic risk or vulnerability factors (such as altered brain structure or function) may play a role in the etiology of all psychiatric disorders, including PTSD, rather than the psychiatric disorders serving as risk or vulnerability factors for PTSD or vice versa.

Potential genetic or biological causal risk factors

A third potential causal risk factor is a genetic predisposition that may underlie alterations in brain structure and function that may in turn result in specific symptoms of PTSD. Family and twin studies suggest that PTSD may be "heritable" (i.e., inborn genetic differences may predispose people to develop PTSD; Goenjian et al., 2008; Guffanti et al., 2013; Liberzon et al., 2014; Sumner et al., 2014; White et al., 2013). The potential heritability of PTSD does not mean that people who share the same or similar genetic inheritance automatically or inevitably will develop PTSD—only that when one of two twins or family members who share some or all of the same genes develops PTSD, it is more likely that the other twin or family member also has had, or will have, PTSD than if neither of them develop PTSD. Whether this is actually the result of genetic inheritance or other related factors (such as a shared family environment) has not been definitively established, but it does appear that shared genetic inheritance is associated with a correlated risk of both exposure to traumatic stressors (Stein, Jang, Taylor, Vernon, & Livesley, 2002) and developing PTSD (Guffanti et al., 2013; Koenen et al., 2007; White et al., 2013).

A study of 200 members from 12 multigenerational families from Armenia, who 20 years earlier had survived the massive Spitak earthquake (which led to 17,000 deaths and destroyed more than half the city of Gumri), demonstrated that 41% of the risk of developing PTSD was due to genetic factors (Goenjian et al., 2008). A profile of the objective impact of the earthquake was compiled for each person, including

factors such as whether their home was destroyed, deaths of relatives, seeing dead bodies, being injured, and witnessing the injury of another person. After statistically accounting for the effects of these factors, age, and other variables that were related to the likelihood of developing PTSD (female gender, pre-earthquake traumatic experiences), the extent to which family members shared common genes was strongly related to their risk of developing PTSD (as well as to resilience and recovery from PTSD). Exposure to earthquake traumatic stressors also played a role in the risk of developing a depressive or other anxiety disorder, although the contribution of genetic inheritance to those disorders was stronger (66% and 61%, respectively) than for PTSD. While specific genes were not identified, it appeared that the same or very similar genes were responsible for the risk of developing any or all of the PTSD, anxiety, or depressive disorders.

If there is a genetic causal risk factor for PTSD, a specific genetic source of the risk will have to be identified (i.e., specific genes or portions of a genes that are different among people with and without PTSD). This is unlikely to be a single gene or portion thereof because PTSD is a "phenotypically complex" phenomenon—that is, PTSD involves several alterations in behavior, cognition, emotion, and physiology. Groups or complexes of genes are typically needed to orchestrate complex psychobiological conditions such as PTSD. To add to the complication, the same genes that are associated with depression, other anxiety disorders (such as generalized anxiety disorder and panic disorder), and substance or alcohol dependence also appear to be associated with PTSD because the genetic contribution to PTSD seems to share most of its variance with the genetic contribution to depression and to other anxiety disorders (Koenen et al., 2007). Thus, the same or similar genetic alterations might underlie several psychiatric disorders that share anxiety, depression, and emotion dysregulation symptoms—and not just PTSD.

Genetically transmitted biological alterations that might be responsible for an increased risk for PTSD include those involved in emotion regulation (Harrison and Tunbridge, 2008) and cognitive abilities (Kremen et al., 2007). Problems in each of these areas of psychological functioning are associated with specific brain structures and processes (see Chapter 6), and both could either increase the likelihood of exposure traumatic stressors or reduce the ability of the impaired individual to cope emotionally with stressors once exposed. Therefore, in order to fully account for genetic causal risk factors, the many ways in which PTSD alters behavior (such as avoidance of reminders of stressful events, problems with sleep, withdrawal from relationships), cognition (such as hypervigilance, blame of self and others, difficulty sustaining concentration on mental tasks, and difficulty accessing verbal memory to solve problems), emotion (such as anxiety, irritability and anger, and dysphoria), and physiology (such as hyperarousal, tendency to startle easily, difficulty with sleep, and physical reactivity to reminders of stressful experiences) each will have to be linked to specific genetic locations or complexes.

Two biological characteristics that have been linked to PTSD are dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and altered size and function of the hippocampus in the brain (see Chapter 6). The most consistent HPA axis alteration in PTSD is low levels of cortisol (hypocortisolism), which is the brain chemical that

"turns down" the body's stress response. These findings suggested that PTSD may be caused in part, biologically, by a stress response system that cannot be slowed down. A study of Swedish soldiers found that those with lower prewar levels of cortisol were at risk for developing PTSD (Aardal-Eriksson, Eriksson, & Thorell, 2001). However, a metaanalysis of dozens of studies concluded that there were no systematic differences in cortisol levels between people with PTSD and controls (Meewisse, Reitsma, De Vries, Gersons, & Olff, 2007). It also is not clear whether cortisol levels of children (Yehuda et al., 2007) or adults (Delahanty, Nugent, Christopher, & Walsh, 2005) are altered as the result of: (i) genetic inheritance, (ii) early life parent-child or family interactions, (iii) exposure to a traumatic stressor (such as in the first hours after a severe accident or assault), (iv) developing PTSD, or (v) some combination of these factors. Thus, neither genetics nor an abnormality in the body's stress response system can be assumed be a causal risk factor for PTSD; either genetics or biological abnormalities may contribute to PTSD or may be only indirectly or secondarily related to PTSD.

A second example of the potential but as yet uncertain role of genetics and biology in PTSD is provided by studies on the hippocampus, a brain region that is thought to play an important role in creating and retrieving personal ("autobiographical") memories, and numerous studies have shown evidence of smaller hippocampi among people PTSD (and one recent study showed a correlated reduction in the activation of the hippocampus in laboratory experiences; Astur et al., 2006; see Chapter 6). Findings of smaller hippocampi in PTSD, and evidence that chronic stress is associated with loss of neurons in animals' hippocampi (McEwen, 2006) led to speculation that traumatic stressor exposure or having PTSD might cause atrophy in the hippocampus. However, rigorous scientific studies did not support the hypothesis that PTSD caused atrophy of the hippocampus (Neumeister, Henry, & Krystal, 2007). Studies of the size (volume) of the hippocampus in twins indicate that smaller hippocampi may be a risk factor for PTSD that is not inborn but results from adverse prenatal and early childhood environmental factors such as poor nutrition, exposure to toxins, or imbalances of maternal hormones during the early period of brain development in utero and in infancy (Woodward et al., 2007). Thus, what appear to be genetically based alterations a brain area involved in memory and PTSD may actually be due to environmental factors that are causal risk or vulnerability factors for PTSD.

Longitudinal studies of genetics and exposure to adverse experiences have shown that genetic factors may have a causal risk effect by interacting with potentially traumatic experiences rather than directly causing adverse outcomes such as PTSD. For example, a prospective study from birth to adulthood of a large sample of males in New Zealand (Segman, Shalev, & Gelernter, 2007) found that a particular genetic characteristic interacted with maltreatment experiences such that boys with the characteristic who were maltreated showed more aggressive violence as adults than those who were not maltreated (Caspi et al., 2002; Segman et al., 2007). A different gene variant was found to interact with stressful life experiences (including childhood abuse) in determining the risk of depression in adulthood (Kaufman et al., 2004), and to interact with the extent of exposure to Hurricane Katrina and adequacy of social support to determine the risk of PTSD (Kilpatrick et al., 2007; White et al., 2013).

Most interestingly, variants of genes that are directly involved in the body's stress response systems have been particularly likely to be associated with risk of PTSD. Studies showed that genes that control the production and effects of cortisol and related HPA stress hormones were associated with PTSD risk in hurricane survivors (White et al., 2013) and interacted with a history of childhood abuse trauma (but not with childhood exposure to other types of traumatic stressors) in determining the risk of PTSD symptoms in a large sample of adults in nonpsychiatric health care (Binder et al., 2008). Another gene variant that is involved in adrenaline responses to stressors was found to interact with childhood exposure to traumatic stressors in determining risk versus resilience to PTSD in two very different samples: primarily Caucasian male military veterans and African American women (Liberzon et al., 2014). Consistent with these findings, the pretrauma exposure physiological characteristics that have been shown to be risks for PTSD involve stress reactivity: startle reactivity (Orr et al., 2012; Sijbrandij, Engelhard, Lommen, Leer, & Baas, 2013), muscle tension (EMG) reactivity (Guthrie & Bryant, 2006; Sijbrandij et al., 2013), high levels of a metabolite of adrenaline (MHPG) in saliva (which also were related to peritraumatic distress; Apfel et al., 2011), difficulty in extinguishing conditioned fear responses (Lommen, Engelhard, Sijbrandij, van den Hout, & Hermans, 2013), and the number of brain receptors in for stress hormones (glucocorticoids) (van Zuiden, Geuze, et al., 2012; van Zuiden et al., 2011; van Zuiden, Heijnen, et al., 2012). Only one physiological marker for stress reactivity—cortisol levels—has not been found to be a PTSD risk factor, and it also was not related to peritraumatic distress *but* was predictive of dissociative symptoms and acute stress disorder (Inslicht et al., 2011). Thus, the path to PTSD may begin with a biological tendency to stress reactivity, except among individuals who are more prone to dissociation (who may instead develop the "shut-down" form of dissociative PTSD).

Genetic factors related to biological stress reactivity thus play a crucial role in determining the risk of or resilience to PTSD when people are exposed to traumatic stressors. The specific type of stress reactivity (e.g., hypo- versus hyperarousal) may be greatly influenced by these genetic characteristics. Moreover, it is not genetics alone but the interaction of genes with different types of exposure to traumatic stressors—particularly childhood maltreatment, but also adult exposure to violence or severe natural disasters—that is crucial in PTSD etiology (and related disorders such as depression) and will be the subject of study for many years to come.

Cognitive processing capacities

Clinicians and scientists (as well as philosophers, historians, and writers) have long observed that life-altering experiences often are followed by profound changes in a person's beliefs that include a sense of alienation from and loss of faith in self, other people, social institutions, and spirituality (Frankl, 1946). Psychological trauma can shatter the assumptions of invulnerability (Janoff-Bulman, 1992) and trust (Freyd, 1994) that are psychologically sustaining "positive illusions" (Taylor, 1989). More

specifically, recent theories have hypothesized that exposure to traumatic stressors may alter not just beliefs but the underlying cognitive processes that are the basis for creating, sustaining, or changing a person's knowledge and fundamental beliefs.

Psychological research has demonstrated that beliefs are based on "schemas," which are ideas ("mental representations") that organize everything that a person knows (the "knowledge base") (Fiske & Taylor, 1991). Schemas are like filters or "blueprints" (Dalgleish, 2004) that people use to match what they experience on a sensory level with what they know from prior experience in order to create perceptions ("what I see, hear, touch, taste, and feel"), emotions ("how I feel"), and thoughts ("what I know, wish, hope, believe, or plan"). For example, "safety" is a schema that includes different elements for different people, involving family and friends for one person or self-reliance and physical or mental prowess for another person. Schemas are always in the background when people perceive, feel, think, and act, and rarely are actually observed except in activities that encourage intensive self-reflection (such as some approaches to psychotherapy or psychological research, philosophical inquiry, or spiritual renewal). Traumatic stressors tend to contradict most commonly held schemas (such as safety, trust, and self-confidence), potentially leading to rapid radical shifts in not just beliefs but basic assumptions about self and the world (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999; Janoff-Bulman, 1992). However, schemas are relatively resistant to change, so it has been hypothesized that the contradictory information provided by psychological trauma may be sequestered separately from existing schemas in what Horowitz (1997) describes as "active memory." This could account for the common experience described by persons with PTSD that it seems as if they have two divided consciousnesses—one in which they experience themselves and the world as they were before a traumatic stressor occurred and the other in which they experience a more threatening and dangerous world and powerless or ineffective self.

This potential posttraumatic psychological conflict between long-held schemas and newly learned contradictory—and emotionally highly charged and negative—information also could account for the cardinal symptoms of PTSD. Intrusive reexperiencing could be due to periodic episodes of unwanted awareness of new information in "active memory." Hyperarousal could be the physical and emotional distress due to not being able to integrate the traumatic information that is in "active memory" into existing schemas. Avoidance and numbing of emotions could be automatic attempts to remove trauma-related knowledge and associated emotional distress from "active memory" in order to reduce or eliminate awareness of these painful thoughts, images, and feelings (Horowitz, 1997). Thus, the psychological conflict between the security provided by preexisting schemas and the traumatic stress reactions triggered by memories of traumatic events could be an underlying cause or major contributor to PTSD.

However, numerous research studies suggest that persons with PTSD have such profoundly altered beliefs that it appears more accurate to characterize them as having adopted new schemas rather than experiencing a psychological conflict between preexisting schemas and traumatic information (Foa et al., 1999). Foa and Rothbaum (1998) hypothesize that the underlying mental structure of knowledge—what they call the "associative network," which is the interconnected ("associated") collection of perceptions, emotions, and thoughts that each person develops based on life

experiences—can be altered by exposure to traumatic stressors. A new associative network is believed to emerge when PTSD develops, organized around distressing (principally either anxiety or anger) or absent ("numbed") emotions (Foa & Rothbaum, 1998) and corresponding new schemas (or "appraisals"; Ehlers & Clark, 2000) that are organized around core guiding beliefs that the world is dangerous and the self is powerless or ineffective.

The possibility that altered knowledge ("active memory"; Horowitz, 1997) or schemas ("fear network" or fear-based appraisals; Ehlers & Clark, 2000; Foa & Rothbaum, 1998) might cause or contribute to the development or persistence of PTSD raises a basic question: how does exposure to traumatic stressors differ from other types of life experiences sufficiently to create not just new knowledge or modifications in existing schemas but new knowledge that is painfully intrusive or new schemas that are completely based on fear rather than other positive (such as confidence or hope) or negative (such as sadness or guilt) emotions and guiding beliefs?

Brewin (2001) and Dalgleish (2004) attempt to answer this question by postulating that fundamental changes in how a person processes information cognitively occur as a result of demands placed upon the mind by psychologically traumatic events. Brewin (2001) theorizes that human beings have two modes of information processing, one of which enables us to make sense of and recall autobiographically (i.e., as a personal life "story") ordinary experiences in life based on conscious awareness and brief verbal summaries of experiences; this is described as "verbally accessible memory" (VAM). The second form of information processing—"situationally accessible memory" (SAM)—is viewed as a largely automatic nonconscious intake and storage of memories in the form of sensory and bodily reactions to experiences (such as in the form of images, sounds, or bodily feelings). Both VAM and SAM are unique and complementary sources of information processing that together provide complete and meaningful memories in every life experience. Brewin (2001) hypothesizes that traumatic stressors shift the ordinary balance between VAM and SAM such that SAM dominates and VAM becomes impoverished. This is consistent with research findings indicating that if VAM is interrupted and SAM is relatively enhanced in an experiment, healthy young adults report more "intrusive" (unexpected, spontaneous) memories of experimental activities than when VAM is intact or SAM is interrupted (Holmes, Brewin, & Hennessy, 2004). VAM and SAM also are consistent with the cognitive processes that are associated with brain activation patterns that have been identified with, respectively, healthy trauma survivors or nontraumatized adults (VAM) and adults with PTSD (SAM) (Brewin, 2001; see also Chapter 5).

Dalgleish (2004) incorporates elements from each of these information processing theories of the alterations in PTSD in the schematic, propositional, analogue, associative representational system (SPAARS) theoretical model. PTSD is theorized to be the result of alterations in verbal information processing (similar to Brewin's VAM), including schemas (S), propositions (i.e., basic beliefs or appraisals, P), and analogue information processing (i.e., nonverbal sensory-perceptual knowledge, A, which is similar to Brewin's SAM). When these alterations in information processing are sufficient to change the person's basic associative representational systems, PTSD is theorized to result (Dalgleish, 2004). Although there are numerous research studies

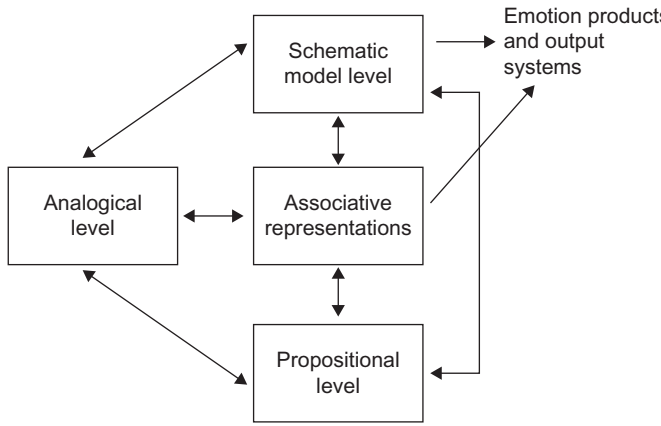

Figure 3.3 SPAARS model of altered information processing in PTSD.
From Power and Dalgleish (1997). Copyright 1997 by Psychology Press. Reproduced with permission.

that indirectly support the SPAARS and related theoretical models of altered information processing in PTSD (Dalgleish, 2004), definitive studies showing whether exposure to traumatic stressors is preceded or followed by these cognitive alterations, and when and for whom such alterations lead to PTSD, are needed before altered information processing can be considered to be established as a causal risk factor (versus a by-product) of PTSD (or correlated but separate phenomenon) (Vogt et al., 2007) (Figure 3.3).

Limitations in cognitive capacities have been consistently found to be a general risk factor for PTSD. The most consistent finding is that individuals with lower levels of childhood verbal IQ (Betts, Williams, Najman, Bor, & Alati, 2012; Koenen et al., 2007—although possibly only females) or adult verbal IQ (Orr et al., 2012; Parslow & Jorm, 2007) and cognitive processing and memory (Parslow & Jorm, 2007) abilities are vulnerable to developing PTSD. Research on anxiety disorders has identified a specific cognitive mechanism that may be a causal risk factor for PTSD: "attention bias to threat" refers to a tendency to pay more attention to either recognizing or avoiding awareness of potential threats (Pine, 2007). These two reactions could help to explain the PTSD symptoms of hypervigilance (the bias toward paying attention to threats) and avoidance (the bias to shifting attention away from threats). A study with adults who had experienced traumatic stressors showed that those with PTSD were more prone to shift their attention toward or away from stimuli associated with threats. Of particular importance, those with PTSD had a unique pattern of brain activity. They had generally higher levels of activation of the "executive" area of the brain (the dorsolateral prefrontal cortex), which suggests that they were making a conscious effort to focus their attention (similar to what would be expected if an individual is hypervigilant). In addition, when they avoided paying attention to threats, the adults with PTSD had higher levels of activation in the "emotion processing" areas of the brain, the ventromedial prefrontal cortex and the anterior cingulate (Fani et al., 2012).

Another study similarly found that adults with PTSD were found to more often direct their attention toward or away from laboratory stimuli that were associated with threat and comparable adults who had experienced traumatic events but did not develop PTSD or who had never been exposed to traumatic events (Iacoviello et al., 2014). However, military personnel who either were or were not deployed to hazardous assignments did not differ before deployment but diverged during the deployment period, with the hazardous duty group showing greater attentional bias toward and away from threat than nondeployed personnel—and the greater the extent of attentional bias, the greater the likelihood of developing PTSD and depression symptoms. Further analyses of the civilian adult sample by Fani et al. (2013) revealed that individuals who showed a bias in attention toward or away from threat also had a genetic variation that affects the body's stress reactivity (the *FKBP5* gene that regulates brain receptors activated by cortisol)—and the shape and activity of the brain area that is crucial to forming and retrieving memories—the hippocampus—also was different for those individuals compared to similar adults. Thus, attention bias related to threat may be a by-product of a genetic difference that either results from or is a pretrauma causal risk factor for PTSD. Either way, altering this attention bias could be an important aspect of preventing or treating PTSD. For example, a study showed that cognitive training for military veterans with PTSD to shift their attention toward neutral (rather than toward or away from threat-related) stimuli substantially enhanced the benefits of other evidence-based psychotherapies and pharmacotherapy (Kuckertz et al., 2014).

Potential risk factors for exposure to traumatic stressors

There have been fewer studies on the factors that place a person at risk for exposure to traumatic stressors than on risk and vulnerability factors for PTSD itself. Two consistent predictors or correlates of exposure to traumatic violence (primarily violence in the community among children (Finkelhor, Ormrod, Turner, & Hamby, 2005) or adults (Briggs-Phillips & Hobfoll, 2006; Schumm, Rayburn et al., 2005) or due to war or terrorism as a noncombatant (Ford, 2008), and secondarily to traumatic family violence (Dutton, 2008)), are living in an unsafe community (or family) and having impoverished socioeconomic resources (such as due to economic poverty, unemployment, or homelessness; Kim et al., 2010).

Another potential risk factor for exposure to traumatic stressors is externalizing behavior, which refers to behaviors that "act out" (or "externalize") emotions such as frustration or anger. Externalizing behavior disorders include childhood attention deficit hyperactivity disorder (ADHD), oppositional-defiant disorder (ODD), and conduct disorder (CD), as well as adult antisocial personality disorder (ASPD). It has been found that children diagnosed with ADHD are not more likely than other children to have histories of exposure to traumatic stressors, but those diagnosed with ODD appear to have a substantially increased likelihood of having been exposed to traumatic violence and victimization (Ford & Connor, 2009).

One study found that adults who were diagnosed with CD as children or adolescents have a greater risk of subsequent exposure to traumatic stressors and of developing PTSD than other adults (Koenen et al., 2005). Another study showed that young adults (ages 20–23 years old) who were identified by their first-grade teachers as exhibiting problematic aggressive or disruptive behavior were 2.6 times more likely to subsequently experience traumatic physical violence or threats of violence than other youths. However, aggressive/disruptive behavior problems in first grade were not associated with an increased risk of PTSD (Storr, Ialongo, Anthony, & Breslau, 2007). PTSD was more likely among young adults who as first graders reported high levels of symptoms of depression or anxiety. A longitudinal study of children who were assessed for ADHD and then reassessed 10 years later found that both childhood ADHD and maternal smoking during pregnancy (which is associated with physical health and behavioral problems in offspring; Huijbregts, Seguin, Zoccolillo, Boivin, & Tremblay, 2008; Li, Langholz, Salam, & Gilliland, 2005; Riedel et al., 2014) were independent risk factors that increased the likelihood of PTSD in late adolescence or early adulthood, by a factor of 225% and 350%, respectively (Biederman et al., 2014). Thus, behavior problems seem to put children at risk for victimization by traumatic violence, but it is not clear whether such acting out or an underlying sense of dysphoria and anxiety that puts children at risk for PTSD. Replication of these studies also is needed to definitively determine the sequence of externalizing behavior disorders, exposure to traumatic stressors, and PTSD. Severe externalizing behaviors involving aggression and illegal activities are a risk factor for exposure to traumatic stressors, but childhood victimization trauma also may precede such behavior problems (Ford, Chapman, Mack, & Pearson, 2006).

Genetic inheritance also may be a risk factor for exposure to traumatic stressors. A study with more than 400 adult twins found that genetic inheritance (comparing monozygotic and dizygotic twins; see Chapter 5) played a role in determining whether "assaultive" traumatic stressors had occurred, but not for "nonassaultive" traumatic stressors (Stein et al., 2002). "Assaultive" refers to psychological traumas involving interpersonal violence, including physical, sexual, domestic, and community violence (but not combat or war, although a relationship with combat or war trauma cannot be ruled out because it happened too rarely to be adequately tested in this study). The genetic influence on exposure to interpersonal violence was equivalent for both genders, although the findings suggested that different genetic locations were likely to be involved in the risk for females and males. The study sample, although large, was not large enough to permit a more definitive investigation of specific gene variations associated with traumatic assaults.

Vulnerability factors for PTSD

A variety of other "preevent" and "postevent" factors have been demonstrated by research studies to serve as "risk" or "vulnerability" factors for PTSD (see Figure 3.2). These characteristics of the person and their social environment prior to exposure to

traumatic stressors are associated with a higher likelihood of subsequently developing PTSD (Vogt et al., 2007). A first subgroup of vulnerability factors are characteristics of the individual, including:

- age (see Box 3.3);
- female gender (see Chapter 4);
- ethnoracial minority status (see Chapter 4).

Another subgroup of vulnerability factors for PTSD involves stressful events experienced prior to a new traumatic stressor. Although it has been speculated that facing traumatic stressors might "toughen up" or "inoculate" people against the ill effects of subsequent stressors, in fact research studies with adults and children consistently indicate that stressors (particularly if they occur persistently in childhood or if they are traumatic at any point in the lifespan; Box 3.6) deplete rather than increase people's psychological and biological coping abilities and resources. Therefore, the following pretraumatic stressor events increase vulnerability to PTSD:

- prior exposure to other potentially traumatic adversities in childhood such as family mental illness, loss, family violence, poverty, or abuse (Boxes 3.4 and 3.5);
- prior exposure to other traumatic stressors (see Boxes 3.4 and 3.5).

Box 3.6 Social Support: Protective Against PTSD? Or a Potential Problem?

Coping with traumatically stressful events is seldom a solitary task and typically requires considerable amounts of assistance and support from other people. "Social support" refers to those social interactions that provide people with *actual assistance* and *embed them* into a web of social relationships *perceived to be* loving, caring, and readily available in times of need. There are a variety of ways in which social support may benefit psychological well-being and physical health. For example, people who care for us may prevent the occurrence of stress or reduce its severity. Social support networks can facilitate more accurate appraisal of ambiguous stressful encounters and assist in the reappraisal of ongoing stressors. Social support most routinely serves directly to meet demands caused by consequences of the stressor, such as when people provide material aid and other necessities to disaster survivors. Physical presence of other people may hasten a return to physiological equilibrium (e.g., closely embracing a victim of terror). Concerned helpers may also suggest coping options, such as persuading a victim of crime to seek professional help. These expressions of support may help to sustain self-efficacy, self-esteem, optimism, and other psychological resources severely threatened by exposure to stress. Helpful relationships may validate emotional reactions and support cognitive processing of stressful experiences. Family and friends facilitate recovery of emotional equilibrium through continuous expressions of caring, as well as by simply creating opportunities for diversions (Kaniasty & Norris, 2008).

The strongest and most consistent protective factor that reduces the risk of developing PTSD following exposure to a variety of traumatic stressors is social support (Brewin et al., 2000; Vogt et al., 2007). Social support is a source of both emotional (e.g., companionship, moral support) and instrumental (e.g., access to money, help in rebuilding a home or finding a job) resources (Hobfoll, 2001; see Box 2.2). After a traumatic experience, social support can help restore a person's belief that life can be predictable, controllable, and safe after exposure to traumatic stressors has shaken or shattered these sustaining beliefs (Foa et al., 1999).

Social support can be provided by family, friends, or even by strangers in the form of sympathetic media depictions of and demonstrations of support for persons who are directly and indirectly affected by traumatic stressors. The opposite of social support, "social constraints," on the other hand, are reductions or limitations in the amount or quality of social support available to persons affected by traumatic stressors (Hobfoll, 2001). Social constraints can interfere with resistance to or recovery from exposure to traumatic stressors by increasing emotional distress, emotional or physical injuries, and damaging people's sense of self-efficacy and trust, as well as by limiting people's abilities to understand and resolve their distress or injuries. Koenen et al. (2007) showed that children who grow up in poverty, when followed prospectively with assessments over the next 3 decades, were at high risk for developing PTSD.

Financial resources are a form of both instrumental support and social belongingness that can influence recovery because they make it possible for persons affected by a traumatic stressor to obtain more social support, such as by traveling to visit with family or friends, rebuilding a community destroyed in a disaster, resuming an interrupted education, or getting help from a medical or mental health professional. Financial resources can also result in reduced stress by making it possible for victims or family members to rest and recover without suffering problems that undermine their instrumental and emotional social support and social embeddedness, such as job loss, being unable to meet living expenses, or having to forego costly but needed health care.

In some cases, social "support" actually may be unhelpful and burdensome for survivors of traumatic stressors, depending on who provides the support and when and how it is provided. Studies of children and adults who were exposed to a range of potentially traumatic stressors including war, community violence, disasters, and law enforcement stressors showed that social support was *not* protective against adverse outcomes such as depression or PTSD for some individuals or when perceived to be at a high level or by nonfamily members (Kaniasty & Norris, 2008). Social support also has been found to take different forms and to have different degrees of protective value, depending on gender (Tolin & Foa, 2006) and ethncocultural background (Pole, Gone, & Kulkarni, 2008), when individuals or entire communities face traumatic stressors.

Moreover, social interactions that are objectively or subjectively "unhelpful" (especially if they are rejecting or conflictual in nature) have been found to

(*Continued*)

Box 3.6 Continued

increase vulnerability to PTSD in the wake of exposure to traumatic stressors—and also depression and complicated bereavement ("traumatic grief") in the wake of deaths or losses of important relationships (Maercker & Muller, 2004; Ullman & Filipas, 2001). Thus, it is important to build or find genuinely helpful sources of social support in order to protect people from developing or being unable to recover from PTSD, rather than simply telling people that they "need to" ask for help in order to cope with the effects of a traumatic stressor and the other stressors that often follow in its aftermath.

Conflict within a traumatized individual's social network may diminish the positive effects of social support on well-being, and these effects may be felt particularly acutely within vulnerable populations of persons who face complex stressors and have limited resources. For example, a study with homeless women who were parenting children examined the impact of conflict and social support on parenting behaviors over time in a sample of mothers who are homeless and were involved in a study of case management interventions of varying intensity (Marra et al., 2009). Homeless women who reported high levels of helpful emotional and instrumental social support self-reported greater improvements in parenting consistency over time than those who reported lower levels of support. However, when they experienced conflict in support networks this was a risk factor for harsh parenting practices if they also had lower levels of instrumental social support. Thus, social support may enhance homeless mothers' ability to provide consistent parenting, and this may in turn may reduce the adverse intergenerational effect on their children if they (the mothers) are coping with PTSD (see Chapter 2). However, when conflict occurs in the support network of highly stressed and vulnerable individuals, such as homeless mothers, and they also have little access to practical resources and help (instrumental social support), this combination of stressful relationships and limited resources may undermine their ability to provide consistent positive parenting and increase their risk of their parenting in a harsh manner.

Furthermore, social support often begets more social support, when it is provided in a timely and genuinely helpful manner in the wake of traumatic stressors. Kaniasty and Norris (2008) have developed a *social support deterioration deterrence model* based on research showing that it was not only a limitation or absence of social support that failed to protect people exposed to traumatic stressors from developing PTSD or other psychiatric or psychosocial problems but the *progressive diminishment* (or "deterioration") of social support over time following a traumatic stressor. They found that hurricane survivors who received early helpful social support were not only more likely to be relatively resistant to adverse psychological outcomes but also to maintain a positive perception of the social support available to them.

In addition, the conditions of the most basic support system—the family—can lead to increased vulnerability to PTSD. When family relationships are unpredictable, uncontrollable, and negative in their emotional tone, traumatic stressors are more likely to lead to PTSD (Koenen et al., 2007; Smith & Fischer, 2008). Family conflict or disorganization alone cannot cause PTSD, but it can increase vulnerability to PTSD. It also can lead to exposure to traumatic stressors, including childhood maltreatment and domestic violence. Poor psychological stability of family members, and of their relationships, therefore are vulnerability factors for the development of PTSD, particularly in childhood, specifically:

- family history of psychiatric illness (Inslicht et al., 2010);
- family instability (Copeland et al., 2007).

Finally, the nature of the person's immediate ("peritraumatic") reactions to traumatic stressors (in the first hours and several days) have consistently been found to be predictive of—but not to guarantee or absolutely "cause"—the subsequent risk of PTSD (see Chapter 2). In addition to the individual's own peritraumatic distress (or dissociation, although dissociation has been less well established as a vulnerability factor than extreme anxiety and intrusive reexperiencing; Brewin et al., 2002; Bryant, 2007), for children the severity of their parent(s)' peritraumatic distress is associated with increased risk of PTSD (Ford, 2008). Severe physical injuries also tend to be associated with a greater risk of subsequent PTSD, although this is not always the case (Daviss et al., 2000). Similarly, directly experiencing a traumatic accident, illness, disaster, assault, or loss tends to be more strongly associated with the development of PTSD than witnessing the same or similar traumatic stressor, although witnesses may suffer from other problems such as bystander guilt. Finally, people who are unable psychologically or practically to talk with supportive others about their traumatic experiences are at increased risk of developing PTSD (Schnurr, Lunney, & Sengupta, 2004).

The peritraumatic vulnerability factors for PTSD thus include:

- peritraumatic parental distress (the parents' own personal (vicarious) distress when their child is exposed to traumatic event(s)) (Lambert, Holzer, & Hasbun, 2014);
- peritraumatic personal distress (Sugar & Ford, 2012);
- peritraumatic dissociation (Sugar & Ford, 2012);
- physical injury and associated pain (Norman, Stein, Dimsdale, & Hoyt, 2008);
- direct (versus witnessed) trauma;
- reluctance/inability to disclose traumatic experiences in the aftermath.

Except for female gender, each of these pretrauma or peritraumatic (during or soon after the trauma) factors has been found to have a relatively small—but nevertheless statistically significant—and positive relationship with the risk of developing PTSD (Brewin et al., 2000; Ozer et al., 2003). Some studies have failed to find a relationship between each preevent factor and the risk of subsequent PTSD (and no study has found more than a moderate correlation between any preevent vulnerability factor and developing PTSD), indicating that *none* of these characteristics *inevitably* destines a person to suffer PTSD if exposed to a traumatic stressor.

Outcome risk factors

When PTSD persists for several months, it often becomes a chronic condition that can last for years, or even decades. Treatment of chronic PTSD is much more difficult and generally less successful than when PTSD is treated early (see Chapters 7–9; Galatzer-Levy et al., 2013). Factors that have been found to be associated with an increased risk of persistent PTSD include several of the potential causal and vulnerability risk factors: severity of both exposure to traumatic stressors and acute PTSD symptoms (Schnurr et al., 2004), psychiatric disorders and problems such as addictions, depression, and anger (Koenen, Stellman, Stellman, & Sommer, 2003; Schindel-Allon, Aderka, Shahar, Stein, & Gilboa-Schechtman, 2010), ethnocultural minority status (Boscarino and Adams, 2009; Schnurr et al., 2004), and gender (Kessler et al., 1995). Two key protective factors also have been found to be associated with reduced risk of persistent PTSD: social support (Koenen et al., 2003; Schnurr et al., 2004) and self-efficacy (Boscarino & Adams, 2009). However, none of these studies assessed a comprehensive set of risk factors, and therefore it is not possible to determine which of these factors are necessary and sufficient to accurately identify who is at risk for chronic PTSD.

Rona et al. (2012) conducted the first comprehensive assessment of risk factors for persistent PTSD, comparing military personnel who reported severe PTSD symptoms following a hazardous deployment and also at a reassessment 3 years later ("persistent PTSD") to those who initially reported severe symptoms but had only mild symptoms 3 years later ("remitted PTSD"). Older personnel and those who had experienced multiple hazardous events during deployment were more likely to have persistent PTSD than to be remitted. However, the strongest risk factors included an 11 times greater risk of persistent PTSD if they felt unsupported on return from deployment, 5 times greater risk if they were separated from the comrades with whom they trained when they were deployed, and 3 times greater risk if they had been discharged from military service, had college level education, or were in poor physical health. The finding that older (typically midlife) individuals were at greater risk than younger adults is consistent with evidence that this period of transition in adulthood is a time of vulnerability to PTSD (see Box 3.3). The finding concerning education is the opposite of results from studies on risk factors for PTSD itself (where education generally is protective against PTSD), suggesting that, like age, education may increase *or* reduce the risk of PTSD, depending on the specific circumstances and whether new or persistent PTSD is the focus. The finding that poor physical health was associated with persistent PTSD parallels epidemiological evidence that PTSD often is accompanied by physical health problems (see Chapter 3): thus, not only do poor health and PTSD often go together because PTSD can compromise a person's physical health, but poor health also may contribute to the persistence of PTSD.

Perhaps most importantly, the results of the Rona et al. (2012) study highlight the important protective role that social support plays in increasing the likelihood not only that PTSD will not occur but also that, in the event it does occur, it does not become a lasting problem. Social support both during a period of exposure to traumatic events (in this case, hazardous military deployment) and in the aftermath

(during the postdeployment homecoming, including both continued contact with military comrades and support from family, friends, and society more generally) were strongly related to the likelihood of recovery from PTSD. Put the opposite way, when those crucial sources of support were not available, PTSD was highly likely to become chronic once it had developed. This provides a segue into the next section on protective factors that mitigate against the development (and in this case, persistence) of PTSD.

The study authors add one additional caveat that is an important reminder that risk factors increase the probability of PTSD but do not definitely destine a person to develop PTSD or to have PTSD become persistent. The actual likelihood that any individual with any one of the risk factors—even ones with high-risk levels such as poor homecoming support or deployment with an unfamiliar team—would have persistent PTSD was not much greater than chance. Rona et al. (2012) concluded that there are many factors that may contribute to the persistence of PTSD, but no single risk factor should be used to predict this for any individual. When multiple risk factors are present, the likelihood of persistent PTSD increases substantially, however, and it is those individuals who most can benefit from treatment so that PTSD does not become chronic.

Protective factors

When traumatic stressors strike, three factors have been identified by clinical observation and validated in scientific research studies as "protective factors"—that is, sources of a *reduced* risk of PTSD. Or in terms of the types of resources described in Chapter 9 and the trajectories of posttraumatic adaptation described in Chapter 2, these are personal and environmental resources that increase the likelihood of resistance to PTSD or resilience or recovery when PTSD develops.

The first protective factor is intelligence (Macklin et al., 1998; McNally & Shin, 1995), reading ability (Storr et al., 2007), or education (Schnurr et al., 2004). This does not mean that "smarter" or better-educated people are invulnerable to developing PTSD, because scientific studies of PTSD etiology (and epidemiology; see Chapter 4) demonstrate that adults and children of *all levels of intelligence, reading levels, and education* can develop PTSD. Intelligence or education are highly correlated with socioeconomic resources, again *not* because more economically affluent people are smarter or more able to succeed in school than people who have lower incomes or material resources, but because economic and material resources provide access to opportunities (such as high quality schools and the time and financial support required to access them) necessary for the kinds of learning tested by psychological measures used to assess intelligence (so-called tests of intellectual functioning or IQ tests).

Education and intelligence therefore are likely to represent the effects of a much wider range of socioeconomic resources, rather than simply a strength (or deficit) in a specific individual. Intelligence and education therefore are part of a network of socioeconomic resources that also are likely to increase a person's access to the other two protective factors against PTSD (self-efficacy and social support). In addition, there are many types or levels of intelligence and education, so it is unlikely

that simply testing above average on an IQ test or having a college education *alone* reduces the adverse impact of exposure to a traumatic stressor. For example, the ability to use and remember information presented in a verbal form (i.e., in words, or "verbal information processing") is more likely to be impaired among people with PTSD than "nonverbal information processing" abilities to use and recall information presented in forms other than words, such as by pictures (Bremner, 2008). With regard to education, Schnurr et al. (2004) found that either a high school or college education was protective against developing PTSD in a large survey of military war veterans but that only college education was associated with recovery from PTSD.

The second protective factor is coping self-efficacy (Waldrep & Benight, 2008). Self-efficacy is the person's belief in her or his ability to effectively both set and achieve goals and succeed in handling stressful challenges. The latter aspect of self-efficacy is "coping self-efficacy," and it is particularly relevant to resisting or resiliently recovering from PTSD. For example, Waldrep and Benight (2008) describe how, after a traumatic incident, people have to cope with many high-stress demands related to recovery, including dealing with insurance companies, finding transportation, dealing with possible injuries, and managing the traumatic aspects of the accident itself. Several scientific studies following major natural disasters (hurricanes, wildfires, earthquakes, and floods), terrorist bombings, bereavement due to a spouse's death, and military combat have shown coping self-efficacy in the immediate aftermath of the traumatic stressor to be consistently associated with a lower risk of PTSD or other psychiatric or psychosocial problems (Waldrep & Benight, 2008). Coping self-efficacy also has been shown scientifically to be a bridge (in technical terms, a "mediator" variable) between other risk factors (such as peritraumatic distress or loss of social resources) or protective factors (such as social support and general self-efficacy or optimism) and reduced mental health problems in the wake of a variety of types of traumatic stressors (Waldrep & Benight, 2008). Thus, the deceptively simple—but actually quite complex and powerful—positive belief in one's own ability to successfully cope with and recover from the adverse effects of traumatic stressors may help create a positive cycle of hope and recovery in place of the vicious cycle in PTSD of anxiety, avoidance, and PTSD.

Several scientific studies have reported a protective relationship between self-efficacy more generally and resistance to or recovery from PTSD. A study of persons exposed to violence in the community that conducted three repeated longitudinal assessments over a 12-month period following exposure to a violent traumatic stressor reported that self-efficacy at the first time point was correlated with the extent of perceived positive social support at that time, as well as predicting (lesser) severity of PTSD at subsequent assessments (Johansen, Wahl, Eilertsen, & Weisaeth, 2007). Thus, self-efficacy and social support are likely to often be interrelated. People with more confidence in themselves are more likely to seek and receive social support, and people with strong social support networks are more likely to feel and be effective in coping with stressors—and to contribute jointly as well as separately to lowering the risk of PTSD.

The third and most consistently scientifically supported protective factor mitigating against the development or persistence of PTSD is social support (see Box 3.6).

Social support may take any or all of three different forms (Kaniasty & Norris, 2008): "*emotional support, informational support*, and *tangible support.* Each of these types of support may be linked to specific sources, such as kin relations (spouse, family, relatives), nonkin informal networks (friends, neighbors, coworkers), and people outside the immediate support circles (charitable organizations, professional service providers)." Most often, social support is measured in terms of people's *perception* or *appraisal* of the amount and quality of social support that they have available—confidence that adequate support would be available if needed or to characterize the extent to which potential sources of support are helpful and cohesive (Kaniasty & Norris, 2008). Schnurr et al. (2004) found that military veterans who had higher levels of perceived "emotional sustenance," "instrumental assistance" (such as family or friends from whom they could get practical or financial help), and "structural support" (i.e., access to tangible social and economic resources) were less likely than comparable veterans to develop PTSD. Interestingly, only emotional and tangible support were protective in the form of increasing the likelihood of recovery if PTSD did develop, suggesting that practical "instrumental" help may be less beneficial for recovery from PTSD than emotional support and socioeconomic resources.

Social support also may be measured more objectively in terms of the actual help that a person receives from others ("received" social support) or of the closeness and position of the person in relationship to a network of potential sources of social support ("social embededness"; Kaniasty & Norris, 2008). Koenen et al. (2003) found that military veterans who were more involved in their communities were less likely to have chronic PTSD years or even decades following war-zone deployment, consistent with a view that social embededness is an important protective factor against persistent PTSD. An additional resource that can serve as a protective factor is the receipt of early effective intervention to prevent or reduce the severity of ASD or PTSD (see Chapter 9).

Conclusion

Many factors in a person's psychological and biological makeup, relationships and resources, and life experiences (including nontraumatic and supportive experiences as well as traumatic stressors) influence the path or trajectory that will lead that person to encounter traumatic stressors and to resist, recover from, or persistently suffer from PTSD. Fortunately, many people never encounter a traumatic stressor in their lives, but most (children as well as adults) will experience at least one and often two or more traumatic stressors. Even then, PTSD is far from inevitable, occurring only for 10–20% of people exposed to a traumatic stressor. The person's strengths and limitations, and those of her or his family, community, and society, play a key role in determining whether PTSD will occur and, if so, whether recovery will occur. The risk, vulnerability, and protective factors identified through careful clinical observation and rigorous scientific research studies provide a basis for developing assessment measures (Chapter 6) and treatment (Chapters 7 and 8) and prevention (Chapter 9) interventions that can further reduce the risk of PTSD and enhance recovery from PTSD.

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