Who Develops Posttraumatic Stress Disorder?

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ABSTRACT—*Nearly half of U.S. adults experience at least one traumatic event in their lifetimes, yet only 10% of women and 5% of men develop posttraumatic stress disorder (PTSD). Why this is so is among the most central questions in current PTSD research. This article reviews the current status of knowledge about who develops PTSD, discussing the strengths and weaknesses of the evidence. We describe the major models used to understand responses to traumatic events, as well as future research directions. We also propose that an exclusive focus on individual differences and individual intervention overlooks opportunities to reduce the prevalence of PTSD by modifying factors at the neighborhood, community, or national level.*

KEYWORDS—*PTSD predictor; dissociation; traumatic event; prevention*

The response to traumatic stress varies widely, ranging from transient disruption of functioning to the chronic clinical condition known as posttraumatic stress disorder (PTSD). Interest in and knowledge about PTSD increased dramatically after its diagnosis was formalized in 1980, but study of the effects of extreme stress has a long history, primarily focused on the effects of war (e.g., shell shock in World War I) and of sexual assault against women. According to generally accepted criteria, diagnosis of PTSD requires exposure to a traumatic event that causes feelings of extreme fear, horror, or helplessness. Traumatic events are defined as experiences that involve death, serious injury, or threat of death. The consequences of this exposure are manifested in three symptom clusters required for diagnosis: involuntary reexperiencing of the trauma (e.g., nightmares, intrusive thoughts), avoidance of reminders and numbing of responsivity (e.g., not being able to have loving feelings), and increased arousal (e.g., difficulty sleeping or concentrating, hypervigilance, exaggerated startle response).

Because PTSD requires the presence of an external event and symptoms linked to this event, it differs from virtually all other psychiatric disorders and raises intriguing issues regarding the definition of trauma, the role of individuals' appraisal of and responses to the event, the implications of a single versus repeated or ongoing exposure, and the role of community- and societal-level changes in attempting to prevent PTSD.

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PREVALENCE

Results from a nationally representative study indicated that over the life course, 10% of women and 5% of men in the United States experience PTSD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Moreover, approximately half of adults have experienced a traumatic event. In a national survey of Vietnam veterans conducted in the late 1980s, Kulka et al. (1990) estimated that 31% of males and 26% of females in this population had PTSD from their military service. Because PTSD symptoms wax and wane, especially in response to subsequent life events (not necessarily traumatic ones), many people experience *partial PTSD*, or clinically significant symptoms of PTSD that do not meet the diagnostic criteria for the disorder. Including individuals with partial PTSD resulted in an estimate of roughly 830,000 Vietnam veterans with significant posttraumatic distress or impairment approximately 20 years after service (Weiss et al., 1992).

The disparity between the 50% prevalence of exposure to trauma and the 7% lifetime prevalence of PTSD means that individual responses to trauma vary dramatically. This variability sparks what appears to be the key question in the field: Why do some people, and not others, develop PTSD? This issue has been of particular interest in recent years, leading to a search for systematic risk factors. Central questions have focused on the correlates or predictors of who develops the disorder and the strength of these effects. Current conceptualizations of PTSD symptoms provide potential explanatory frameworks for appreciating how predictors may influence the stress response and lead to differential risk for PTSD.

MODELS OF TRAUMA RESPONSE

Models Focused on Cognitive and Emotional Processes

The two most influential cognitively oriented formulations of trauma response and recovery highlight either the importance of beliefs and linked emotions about the self and the world (McCann & Pearlman, 1990) or the network of associations linking thinking about or reminders of a traumatic event to cognitive, emotional, physiological, and behavioral responses (Foa & Rothbaum, 1989). In the former formulation, a traumatic event is conceptualized as shattering the

previously held assumption that though the world is not always safe, the lack of safety affects other people only. Thus, the trauma victim's thinking about the world must be adapted to assimilate this shattered assumption and make sense of and integrate the event. The PTSD symptoms of intrusion and avoidance arise from this process, which is generally experienced as painful because it requires remembering the trauma and the accompanying distress. Recovery gradually occurs when this iterative process can be tolerated without avoidance or being overwhelmed emotionally. Thus, factors that reduce the likelihood of effective integration and assimilation would theoretically increase the likelihood of chronic stress-related symptoms and PTSD. These factors include characteristics of the individual, his or her environment, and the event itself.

In the latter cognitive formulation, the metaphor of a memory network is invoked to describe linked information about the traumatic event and subsequent cognitive, affective, physiological, and behavioral responses. Activation of one element in the network activates other aspects—almost always including fear—and this uninterrupted repetition accounts for the continuing symptoms. Recovery occurs if the strength of the associations among network components is reduced by a combination of desensitization and substitution of more adaptive associations.

Biologically Focused Models

Research on the biology of PTSD initially focused on studying psychophysiological arousal in the presence of reminders (sounds, images, or scripts) of the traumatic event. Results indicated that individuals with PTSD demonstrated heightened arousal and prolonged duration of arousal compared with control subjects (e.g., Keane et al., 1998). Recently, researchers investigating the biological substrates of PTSD have focused on the processes and structures of the brain. Research has centered on the amygdala and hippocampus, key brain areas involved in the fear response and in the consolidation of memory (e.g., LeDoux, 2000), as well as on the hypothalamic-pituitary-adrenal (HPA) axis, the parts of the neuroendocrine system that control reactions to acute stress.

Examination of parts of the brain involved in the fear response has been extensive because traumatic events usually generate fear, and because fear initiates the "flight or fight" physiological arousal associated with the hyperarousal symptoms of PTSD. Fear has also been implicated in the mechanisms establishing and maintaining traumatic memories. Research in animals has generally examined brain circuitry; research in humans has included neuroimaging studies of brain structures (Schuff et al., 1997) and processes (Rauch et al., 1996). New findings from animal studies have established direct neural pathways from sensory input to areas of the amygdala. In light of the known reciprocal neuronal connections between the hippocampus and amygdala, these findings suggest a powerful explanation for the automaticity of the fear response and the manner in which emotional memories occur and are transmitted to the hippocampus.

Careful study of individuals with PTSD indicates that they are characterized by an oversensitivity of the HPA axis. The HPA axis is involved in generating, maintaining, and shutting down increases in stress-related hormones in the face of danger, a central aspect of traumatic events. Evidence suggests that individuals with PTSD exhibit dysregulation in the activity of cortisol, a hormone regulated by the HPA axis. The destructive effects of the excessive production of cortisol are believed to be responsible for the atrophy of the hippocampus frequently found among individuals with chronic PTSD. The dysregulation in the HPA axis involves the feedback loop that puts the brakes on the arousal generated by the perception of fear (Yehuda, 1997). These findings have generated research aimed at exploring the use of medications such as beta-blockers to dampen initial arousal. With initial arousal dampened, the consolidation of emotional memories may be attenuated. The hope, therefore, is that the reduction of physiological arousal immediately after the traumatic event will interfere with the processes that lead to the development of PTSD.

PREDICTORS OF PTSD

Two major meta-analyses (statistical analyses combining the results of many studies) of the predictors of PTSD have recently been published (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003). These studies examined four categories of predictors: (a) historical or static characteristics such as family psychiatric history, intelligence, childhood trauma, and other previous trauma; (b) trauma severity; (c) psychological processes during and immediately after the trauma; and (d) social support and life stress after the traumatic event. Both meta-analyses showed that there were significant predictors of PTSD in all four categories, but that the strength of prediction varied across the categories. Those factors closer in time to the traumatic event (i.e., proximal factors) showed a stronger relationship to PTSD ($r \approx .40$) than did characteristics of the individual or his or her history that were more distant in time (i.e., distal factors; $r \approx .20$). The strongest predictor (included only in Ozer et al.) was peritraumatic dissociation. Peritraumatic dissociation refers to unusual experiences during and immediately after the traumatic event, such as a sense that things are not real, the experience of time stretching out, and an altered sense of self. Feeling that one is watching oneself in a movie or play as the event unfolds is a common description of the experience of dissociation. The strength of the relationship between such dissociation and likelihood of developing PTSD was in the moderate-to-large range.

Several important points regarding the predictors of PTSD should be highlighted. First, because largest correlations were about .40, peritraumatic dissociation and other predictors are neither necessary nor sufficient for developing PTSD. Second, the explanation for why peritraumatic dissociation is a predictor requires considering a host of differences in both the people exposed and the nature of the exposure. It may be that the severity of the traumatic event influences the likelihood of peritraumatic dissociation, either through the level of psychophysiological arousal the individual endures during the event or through more complicated relationships involving the effects of the individual's temperament, prior experience, prior psychological functioning, and other genetic or environmental factors that affect his or her capacity to regulate the emotional response. Third, level of social support following the trauma was also a strong predictor, with more social support associated with lower likelihood of later PTSD symptoms. An individual's level of social support likely relates to his or her history and functioning prior to the trauma, factors that this literature has generally not investigated and that meta-analytic approaches cannot easily summarize.

PROBLEMS AND POTENTIAL SOLUTIONS

The main limitation of the research on predictors of PTSD is the heavy reliance on self-report measures and retrospective designs. This natu-

ralistic, retrospective approach makes sense considering the general unpredictability of exposure to trauma and the obvious ethical problems of exposing research participants to extreme stress in experimental or quasi-experimental designs. Prospective studies initiated prior to the occurrence of a major disaster or trauma, however, help address this limitation. For example, recent prospective research has assessed the psychological aftermath of the September 11 terrorist attacks in the United States (Silver, Holman, McIntosh, Poulin, & Gil-Rivas, 2002). Longitudinal research with individuals in high-risk jobs, such as jobs in the military, emergency services, and police force, also provides opportunities for prospective studies of possible predictors of PTSD.

Furthermore, the processes by which identified predictors may shape the development of PTSD remain largely unexamined. Systematic investigation of the ways in which these factors influence responses to trauma at multiple levels (e.g., behavioral, social, biological) could potentially inform interventions to attenuate or prevent PTSD. Future research should emphasize the more proximal mechanisms or processes—in psychological or physiological terms—that account for the relationship between PTSD symptoms and the more distal, static predictors such as prior trauma and family history of psychopathology. Evaluation of theory-based interventions with valid operationalization of critical variables could then provide data with which to evaluate current theory, an important area of study given the ethical prohibitions regarding experimental research in this field. Meta-analytic examination of the PTSD literature was useful in identifying simple, linear relationships between predictors and PTSD symptoms. It is likely, however, that some predictors influence each other in more complex ways; for example, a given predictor may strengthen the effects of another predictor on the development of PTSD (moderation) or may serve as the mechanism through which another predictor increases the likelihood of developing PTSD (mediation). Moreover, the unique meaning of exposure for a single individual may provide the most parsimonious explanation for why a person develops PTSD.

INTRIGUING ISSUES AND QUESTIONS

Definition of Traumatic Event

The definition of what constitutes a traumatic event is central to the diagnosis of PTSD and to all research regarding the disorder. Defining a traumatic event, however, is not simple; indeed, the diagnostic definition has changed over the past decade. Definitional issues raise interesting challenges for PTSD research as they call into question what kinds of experiences are traumatic and for whom. If two people experience the same event (e.g., encountering body parts) but only one reacts with fear, helplessness, or horror, has only one of them experienced a traumatic event?

Because traumatic events typically involve immediate horror and threat to survival (e.g., sexual assault at knifepoint, torture, combat), very high physiological arousal usually accompanies the experience. A broadening of the types of events that some people consider to be traumatic has led to inclusion in the PTSD literature of studies of highly distressing events (e.g., receiving a diagnosis of cancer) that may or may not invoke the same arousal that acute life-threatening situations do. The presence or absence of arousal may well become a key phenomenon that has implications for symptoms of PTSD and whether or not an event is deemed traumatic. If the subjective emotional and physiological response to the event is overlooked, research may not yield consistent findings that would perhaps emerge if arousal were required to identify an event as traumatic.

Ongoing Exposures and the Prototype of PTSD Symptoms

Early theories of trauma response and PTSD were largely based on individuals who lived in generally positive environments and experienced a discrete traumatic event or series of events within a discrete period of time (e.g., sexual assault, disaster, military service), so that the traumatic event or events signified a dramatic disruption of pretrauma life. It is unclear how well this model fits the experience of individuals subjected to pervasive traumatic stress, for example, in the contexts of chronic physical or sexual abuse, deadly civilian conflicts and genocide, or severe community violence in low-income urban areas. The impairments of such individuals, including problems in interpersonal relationships and affect and impulse regulation, may be complicated and difficult to treat (Herman, 1992). The self-perceptions of people who have experienced ongoing trauma seem to be dramatically worse than those of individuals who have experienced discrete traumatic events in the context of otherwise normal development. Some researchers have suggested that a separate term, such as "complex PTSD" or "disorders of extreme stress—not otherwise specified," should be used in place of PTSD to better describe this disorder. Much prior research did not examine whether the predictors of disorder differ depending on whether trauma is experienced as a discrete event or as an ongoing condition of life. Future research that investigates this distinction may find clearer patterns of predictive relationships than have been uncovered so far.

Prevention of PTSD

What are the implications of the research on predictors of PTSD for the prevention of the disorder? Secondary-prevention efforts that seek to reduce the likelihood of PTSD among individuals who have recently been exposed to traumatic stress could utilize these findings by developing early-intervention models that target processes associated with PTSD risk in the meta-analyses reviewed here (e.g., social support, peritraumatic dissociation if the affected individuals could be seen immediately following the event). Strategies for the primary prevention of PTSD would entail reducing the incidence of traumatic events. The most frequent types of traumatic events studied in the research literature have been combat exposure, interpersonal assaults, accidents, and disasters. Although some traumatic stressors, such as earthquakes, are beyond human control, action at the individual and community levels could clearly reduce the risk of exposure to many forms of traumatic stress and also shape the impact of even uncontrollable traumatic stressors on populations. Indeed, such efforts form the backbone of diverse disciplines and public-health policy efforts in areas including building and transportation safety, community violence prevention, domestic violence prevention, and international diplomacy.

There have been numerous investigations of the prevalence of PTSD in diverse communities that have experienced armed civil conflict or war, political repression, or other chronic violence. In such settings of collective trauma, it is particularly critical to look beyond the individual when considering both the effects of trauma and strategies for intervention and prevention. For example, severe political repression affects not just individuals but also the social

institutions and norms of a nation or community (Martin-Baro, 1994). Virtually all interventions for PTSD focus on the individual with symptoms and utilize medication or psychotherapy. Although these interventions may help alleviate individual symptoms, they are obviously inadequate for addressing the harm to social institutions or promoting long-term healing and mental health if the sources of persistent trauma are not addressed. When PTSD is a consequence of collective social and political conditions, primary prevention of this disorder involves social and political changes in the community or nation, as does repair of the social fabric. Thus, perhaps more than any other psychological disorder, PTSD forces consideration of advocacy and political action as primary (universal) prevention tools.

Recommended Reading

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