Obstacles to the Study of Risk Factors for Posttraumatic Stress Disorder

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Invited ms for Strides

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People vary dramatically in their susceptibility for developing medical illnesses, including psychiatric ones. Yet for many years, studying risk factors for posttraumatic stress disorder (PTSD) was taboo. Psychiatric advocates for troubled Vietnam veterans argued that the war itself, not preexisting psychological problems or vulnerabilities explained why many veterans experienced delayed stress reactions after their return home. They aimed to banish discourse on vulnerability lest it provide an excuse for the federal government to skirt its responsibility to provide veterans with psychiatric treatment and disability compensation (McNally, in press). As Blank (1985) put it, discussing vulnerability for PTSD amounts to “blaming the victim” (p. 83). Of course, identifying predictors of PTSD among people exposed to trauma involves statistical discourse regarding effect sizes, not moral discourse about blame. Nevertheless, fear of blaming victims for their plight impeded the study of risk factors for PTSD for many years.

However, now that the PTSD diagnosis is secure within our diagnostic system (Shephard, 2004), traumatologists are increasingly willing to investigate why victims of trauma vary in their likelihood for developing the disorder (Yehuda, 1999). Indeed, our field must confront risk factors because
the vast majority of people exposed to PTSD-qualifying stressors do not develop the disorder (e.g., Breslau, Davis, Andreski, & Peterson, 1991).

There are two reasons why most trauma victims do not succumb to PTSD. First, victims vary in their vulnerabilities. Just as not everyone who smokes cigarettes is equally likely to develop heart disease or lung cancer, not everyone exposed to a traumatic stressor is equally likely to develop PTSD.

Second, our concept of trauma has dramatically expanded since PTSD first appeared in the third edition of the Diagnostic and statistical manual of mental disorders (DSM-III; American Psychiatric Association [APA], 1980). To qualify as trauma-exposed, people no longer need be survivors of rape, combat, natural disasters, or other catastrophic, life-threatening events. Thanks to a conceptual bracket creep in the definition of trauma (McNally, 2003a), vastly more people are eligible for the PTSD diagnosis than in the past (Breslau & Kessler, 2001). In fact, DSM-IV-TR (APA, 2000) does not even require that a person be physically present at the scene of the trauma to qualify as a trauma survivor. A person who feels helpless when learning about threats to the safety of other people now qualifies as a
trauma survivor just as much as those whose lives were in danger (McNally, 2009; McNally & Breslau, 2008). For example, Schlenger et al. (2002) reported that the terrorist attacks of September 11, 2001 triggered apparent PTSD in 4% of Americans who lived very far from the scenes of the attacks. Evidently, televised coverage of the violence caused the disorder. Although preexisting vulnerability factors affect the probability of psychiatric breakdown even in response to the incontestable trauma of combat (Helzer, 1981), vulnerability factors will dominate the etiological picture when people develop PTSD in response to noncatastrophic stressors (e.g., watching televised coverage of terrorist attacks).

**Conceptual Obstacles to Understanding Risk Factors**

According to the dose-response principle (March, 2003), the more severe the stressor, the more likely PTSD will develop. There are plenty of exceptions to this rule, and measuring severity of trauma independently of the victim’s subjective response remains challenging (For a review, see McNally, 2003b, pp. 79-89). Yet apart from these complexities, there is its ambiguous conceptual status. On the one hand, the dose-response principle implies that trauma severity is a risk factor for PTSD. On the other hand,
trauma is no mere risk factor. Trauma, or more precisely, the memory of trauma (Rubin, Berntsen, & Bohni, 2008), is integral to the concept of PTSD. It is what ties an otherwise disparate list of symptoms together (Young, 1995, p. 5). Hence, discovery of unambiguous risk factors entails the search for predictors of PTSD among people exposed to trauma. That is, given exposure to trauma, what factors heighten risk for PTSD?

Many people with PTSD have encountered traumatic stressors prior to the one triggering their disorder (e.g., Bremner, Southwick, Johnson, Yehuda, & Charney, 1993; Breslau, Chilcoat, Kessler, & Davis, 1999). These findings seem to imply that exposure to trauma is a risk factor for developing PTSD in response to a later trauma. Even when a specific stressor does not itself incite PTSD, it may sensitize victims, rendering them especially likely to develop the disorder in response to subsequent stressors.

One can understand the appeal of this hypothesis to anyone who worries that identifying risk factors blames victims for their plight. Although it recognizes individual differences in vulnerability to PTSD, the hypothesis locates the ultimate source of this vulnerability in earlier stressors, not within the person’s cognitive or genetic make-
up. Hence, trauma serves two functions. It functions as a risk factor for developing PTSD in response to later trauma, and as the direct cause of PTSD when the disorder does emerge.

Unfortunately, researchers testing this hypothesis failed to assess how subjects responded to the original trauma, as Breslau, Peterson, and Schultz (2008) observed. Revisiting their previous prospective, longitudinal data of civilian PTSD, Breslau et al. found that previous exposure to trauma did not increase risk for PTSD in response to later trauma unless subjects had developed PTSD in response to the original trauma. Hence, prior trauma per se is not a risk factor for PTSD in response to later trauma. Rather, PTSD in response to prior trauma predicts PTSD in response to later trauma. In a subsequent investigation, Breslau and Peterson (in press) found that this effect held regardless of stressor type. For example, exposure to assaultive violence early in life in the absence of PTSD did not confer risk for PTSD in response to later trauma.

Breslau et al.’s (2008) findings dovetail with Koenen et al.’s (2008) prospective, longitudinal study of subjects in Dunedin, New Zealand. Koenen’s research group found that trauma victims in this civilian cohort almost never succumbed
to PTSD unless they had developed another psychiatric disorder earlier in life. Hence, with only very few exceptions, PTSD never occurred in people who had not already exhibited vulnerability for mental disorders.

Much research on risk factors for PTSD among people exposed to trauma concerns individual difference variables such as intelligence and neuroticism. Relative to victims with higher intelligence, those with lower intelligence are more likely to develop PTSD (e.g., McNally & Shin, 1995; Macklin et al., 1998). Relative to victims with lower neuroticism, those with higher neuroticism are more likely to develop PTSD (e.g., Breslau et al., 1991; McFarlane, 1989).

Research on these individual difference variables provides clues to vulnerability. Yet constructs emerging from psychometric studies do not directly illuminate the mechanisms operative within a person (McNally, in press). They are latent variables that psychologists postulate to account for differences among people. To be sure, these traits must somehow have their source in human psychobiology, but none is a mechanism that can explain intraindividual development, cognition, emotion, or behavior (Bechtel, 2008), and thus risk for trauma.
Consider a measure of intelligence or psychometric $g$. It reflects a source of interindividual differences, but it does not correspond to any intraindividual cognitive mechanism within the brain of a single person (Borsboom & Dolan, 2006). The same holds for other abstract constructs, such as neuroticism. It alludes to a broad range of correlated dispositions devoid of any contextual reference (Kagan, 1996). Among its facets are self-consciousness and angry hostility. Yet knowing that someone is high on neuroticism does not tell us whether we should expect a shy and self-conscious person or someone who is angry and hostile. Indeed, it is little wonder that researchers have been unable to identify any reliable biological correlates for such an abstract construct as neuroticism, its high heritability notwithstanding (Claridge & Davis, 2001).

Other scholars question the explanatory potential of neuroticism (Ormel, Rosmalen, & Farmer, 2004). To be sure, measures of this broad construct predict diverse negative outcomes, including life stress, depression, unexplained medical symptoms, and substance abuse. Yet because neuroticism reflects a person’s average level of emotional distress over time, the connection between neuroticism and its predicted outcomes borders on the tautological. People
Risk Factors for PTSD

who experience anxiety, depression, and anger at one point in time are those mostly likely to experience these emotions in the future. To assert that neuroticism is a shorthand marker predicting distress tells us nothing about its psychobiological substrate. Hence, it does not disclose why people high on neuroticism are vulnerable to PTSD.

Conclusion

Identifying risk factors for PTSD among people exposed to trauma is just as important as identifying risk factors for depression, heart disease, and cancer. The alternative is ignorance, and ignorance provides a poor basis for prevention of any disorder, including PTSD. Fortunately, the study of risk for PTSD is no longer de facto forbidden on ideological or political grounds. Nevertheless, some traumatologists still accuse risk factor researchers of blaming victims. For example, the clinical psychologist, Karestan Koenen, receives these accusations for her work on genetic variables associated with risk for PTSD (Karestan, 2009).
References


Breslau, N., Chilcoat, H. D., Kessler, R. C., & Davis, G. C.


