



Review

Primitive mechanisms of trauma response: An evolutionary perspective on trauma-related disorders



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ABSTRACT

The symptoms we identify and the behaviors we recognize as defenses define which symptoms we see as trauma-related. Early conceptions of trauma-related disorders focused on physical signs of distress while current ones emphasize mental symptoms, but traumatizing experiences evoke psychobiological reactions. An evolutionary perspective presumes that psychophysical reactions to traumatizing events evolved to ensure survival. This theoretical review examines several primitive mechanisms (e.g., sensitization and dissolution) associated with responses to diverse stressors, from danger to life-threat. Some rapidly acquired symptoms form without conscious awareness because severe stresses can dysregulate mental and physical components within systems ensuring survival. Varied defensive options engage specialized and enduring psychophysical reactions; this allows for more adaptive responses to diverse threats. Thus, parasympathetically mediated defense states such as freeze or collapse increase trauma-related symptom variability. Comorbidity and symptom variability confuse those expecting mental rather than psychophysical responses to trauma, and active (sympathetically mediated flight and fight) rather than immobility defenses. Healthcare implications for stress research, clinical practice and diagnostic nosology stem from the broader evolutionary view.

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1. Introduction

Nothing makes sense in biology except in the light of evolution
(Dobzhansky, 1964, p. 449)

The history of trauma-related diagnoses shows that how we classify Posttraumatic Stress Disorder (PTSD) depends on the symptoms we see and the behaviors we recognize as defenses. In the US Civil War, the most closely corresponding diagnosis was irritable heart (Da Costa, 1871), colloquially called soldier's heart (Mackenzie, 1920). During World War I, similar symptoms were diagnosed as shell shock or the effort syndrome (Lewis, 1940). With World War II came the diagnosis of traumatic neurosis (Kardiner, 1940). Though the symptoms seen were relatively stable over time, serial diagnoses emphasized differing features. The early medical diagnoses cued in on somatic complaints, such as exertion, infection, and cardiac or thyroid issues. Yet medical explanations of these signs failed to eradicate soldier's heart. As Wilson (1916, p. 120) lamented, "The theorists ... are bankrupt; the disease remains". When cardiac symptoms came to be seen as signs of anxiety after World War I, the physical symptoms associated with soldier's heart were no longer treated as medical problems (Cohn, 1919).

The term PTSD first appeared as an anxiety disorder in the third edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM) (APA, 1980). We continued to group PTSD among the anxiety disorders until the DSM-5 (APA, 2013). The diagnostic criteria for PTSD still emphasize sympathetically mediated behavioral responses to traumatic events (APA, 2000, 2013). How we see or understand trauma-related disorders has research and clinical implications because our perceptions guide diagnosis, research and treatment. For example, only individuals who complain of mental symptoms are diagnosed with PTSD, not those displaying the cardiac signs that would historically have been used to diagnose irritable heart. Some researchers contend that our culture influences trauma responses, while others stress that our biological survival informs these responses. Both may be right; but conflicting perspectives such as these could give rise to differing perceptions of trauma, some of which fail to consider exactly what occurs during a traumatizing experience. The PTSD diagnosis highlights reliably observable behaviors. It has provided a focal point for research and has increased the visibility of trauma-related issues. However, our prevailing notions about trauma responses cannot explain the variability that is seen to surround this disorder.

This variability is manifest in two forms: as comorbidity across disorders and as varied symptoms that can change over time. The PTSD diagnosis is highly comorbid with other mental health diagnoses (e.g., anxiety, bipolar disorder, depression, dissociative disorders, personality disorders, schizophrenia, and substance abuse; Courtois and Gold, 2009; Kessler et al., 2005; Moskowitz et al., 2008). Traumatic events are also surprisingly comorbid with physical illnesses (Boscarino, 2008; Felitti et al., 1998), with chronic pain (Lyon et al., 2011), and with some medically unexplained symptoms (Brown, 2007). In addition, traumatized individuals who receive a diagnosis of PTSD often display widely different symptoms, some of which seem unrelated to sympathetic activity

(Lanius et al., 2003; Orr et al., 2004). Symptoms also vary within people over time (Mason et al., 2002; Reinders et al., 2006).

The confusion around both forms of variability has historical roots. Immunology and neuroendocrinology became distinct academic disciplines because researchers discovered immune and neuroendocrine systems separately. We expect that cognitive and noncognitive threats will elicit separate central, endocrine, or immune responses. Ader (1981) coined psychoneuroimmunology (PNI) as a term in the year after publication of the DSM-III; still, the bidirectional communication among psychobiological elements that is inherent in PNI has never informed the criteria for PTSD. Comorbid physical disorders surprise us because they violate a presumed independence of distinct reactions to different threats. Yet the fact that comorbid disorders exist shows that our reactions to cognitive and noncognitive threats are not orthogonal.

Two implicit premises in the prevailing cognitive perspective impede a full understanding of trauma-related symptoms. One is that mental disorders merit mental explanations. Although mental explanations fruitfully address many affective disorders, they do not always fully resolve trauma-related disorders. Generalizing this premise to trauma-related disorders hinders our understanding of the comorbidities observed between traumatic-stress and physical diseases. The second premise is that only active defenses count as responses to trauma. Cannon (1932) contended that we respond to stress with sympathetically mediated actions (i.e., fight or flight). Clinicians and researchers followed his lead by categorizing PTSD as an anxiety disorder (APA, 1980, 2000; Gray and McNaughton, 2000). Yet parasympathetically mediated defenses generate symptoms as well; the variability accompanying these symptoms baffles us because we do not see immobility responses as defenses, if we notice them at all. These premises fail to account for the primitive mechanisms seen in trauma-related defensive responses.

In contrast, an evolutionary perspective sees both traumatizing experiences and defensive responses through the longer lens of biological survival. Humans inherited the same defensive options that animals use to survive threats such as predation. Bite wounds carry a high incidence of pain and infection. Predation and associated emergencies, such as infection, require rapid and effective reactions that take priority over ongoing behaviors. The ability to coordinate across discrete survival systems should enhance the responsiveness and effectiveness of behavioral, immune, and neuroendocrine defenses. Given a shared goal of protecting the host, it would certainly be adaptive for mammalian behavioral defenses to tap into reciprocal communication with the central nervous system (CNS) and central autonomic network (Maier, 2003). Indeed, this would support immunological memory and learning motivated by survival demands. Survival-related learning seems to exploit internal signals of threat, possibly co-opted from older immunologic responses to antigens (Ottaviani and Franceschi, 1996). Our brain coordinates the neural and physical elements of survival systems, but severe stress disrupts this coordination. Disruptions in the bidirectional dialogs between the CNS and peripheral signals may give rise to trauma-related symptoms. Persisting dysregulation of primitive mechanisms prolongs symptoms after a danger has passed. It follows that trauma-related symptoms are psychobiological, and inherently so. They stem from disruptions in primitive

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