

## Conflict Between Current Knowledge About Posttraumatic Stress Disorder and Its Original Conceptual Basis

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*Objective:* The authors' goal was to explore the historical, political, and social forces that have played a major role in the acceptance of the idea of trauma as a cause of the specific symptoms of posttraumatic stress disorder (PTSD) and to discuss the impact that current research findings have had on some of the initial conceptualizations of the disorder. *Method:* The conceptual origins of PTSD are described, and the literature on the prevalence, longitudinal course, phenomenology, and neurobiology of PTSD is reviewed. *Results:* Paradoxically, there are a series of findings that support the idea that PTSD is a distinct diagnostic entity, but these are different from those originally developed from psychosocial theory and stress research. *Conclusions:* PTSD has been a controversial diagnosis and is again at a vulnerable point. It is imperative that the field address how current findings challenge the original conceptualizations of this disorder so that the next generation of conceptual issues can be formulated.

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The empirical findings that have emerged since the original definition of posttraumatic stress disorder (PTSD) in 1980 have contradicted the original theoretical proposition that the response to trauma as described by the diagnosis of PTSD is essentially a normative one (1-5). This has led to an unusual tension between those with a strong allegiance to the social achievements that have been met by the existence of the disorder and the clinical researchers. In particular, as clinical researchers continue to make observations such as the relative rareness of the disorder following exposure to trauma (6-16), the existence of risk factors other than the trauma as predictors of PTSD (6-9, 12, 15-25), the atypical rather than normative nature of the biological stress response in PTSD (26, 27), and the prevalence of pre- and posttraumatic comorbidity (6, 7, 14, 15, 28, 29), there arises a conflict between those

who wish to normalize the status of victims and those who wish to define and characterize PTSD as a psychiatric illness (30). The future of the traumatic stress field depends upon an acknowledgment of the competing agendas and paradigms that have emerged in the last 15 years since the inception of the diagnosis, a clarification of theoretical inconsistencies that have arisen, and a reformulation of the next generation of conceptual issues. Because PTSD is a prototypic illness in that it allows a characterization of the effects of environmental factors in psychiatric illness, clarification and resolution of divergent philosophical conceptions are imperative to a broader understanding of the role of stress in mental illness.

This article will review some of the conceptual origins of PTSD. We will address primarily the forces that provided the major impetus for establishing a diagnosis that defined a normative process of coping and adaptation in response to exposure to trauma. After a review of the relevant research findings that have emerged in the last decade regarding the prevalence, longitudinal course, and neurobiology of PTSD, it will be suggested that although current data have supported PTSD as a distinct diagnostic entity, some of the specific features of the disorder are different from those originally developed. The implications of these differences will be explored.

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## FACTORS THAT GAVE RISE TO THE DEVELOPMENT OF PTSD

*Conceptual Origins*

The diagnosis of PTSD was established to fill a gap in the prevailing mental health field by acknowledging that extremely traumatic events could produce chronic clinical disorder in normal individuals (1-5). Although the idea that stress could contribute to psychiatric symptoms had been accounted for in previous diagnostic systems, those models primarily viewed enduring symptoms as being caused by premorbid vulnerability (1-5, 27, 31). In DSM-I and DSM-II, for example, the categories of gross stress reaction and transient situational disturbance, respectively, were used to describe acute symptomatic distress following adversity, whereas more prolonged disorders were conceptualized as being anxiety or depressive neuroses. Regardless of whether these conditions were considered as resulting from developmental fixation or genetic predisposition, the role of environmental stress was at best considered a nonspecific trigger that might serve to release, exacerbate, or prolong a predictable diathesis to psychiatric symptoms. Thus, the primary philosophical shift involved in including PTSD in DSM-III was to create a diagnostic category that resolved a previous quandary of how to classify a chronic condition in normal people who developed long-term symptoms following an extremely traumatic event (1, 2). This formulation postulated a general concept of a "posttraumatic stress disorder" and implied that PTSD involved a natural process of adaptation to extraordinarily adverse situations and that the pattern of symptoms did not depend on a constitutional vulnerability.

*Historical Context*

The idea that stress triggers psychiatric illness in normal individuals had even earlier origins than the formal nosologic classification systems. For example, before the development of his libidinal theory of neurosis, Freud's initial theory was that hysteria had traumatic origins (32). Freud subsequently rejected this idea in favor of more developmental models (33) but, nonetheless, continued to assert that the phenomenology of responses to actual trauma (traumatic neurosis) could be differentiated from those that were the product of developmental fixation (34). Similarly, Janet's observation of dissociative responses following exposure to trauma (35), Kardiner's description of the physioneurosis in traumatic war neuroses (36), studies of concentration camp survivors that emerged in the aftermath of World War II (37-42), and studies of the psychological consequences following burn injury (43, 44) were also perspectives that validated the idea that exposure to trauma may have mental health consequences.

The issue that was raised by the proponents of PTSD was not whether extreme stress or trauma could cause psychological damage—it would have been difficult to

argue that trauma could not be a major factor in precipitating psychological symptoms. Rather, the question was how should people who succumb to the effects of trauma be viewed, and treated, by the mental health field. Thus, the formulation of PTSD as a normative or adaptive response to trauma in DSM-III addressed a social and political issue as well as a mental health one. Although DSM-III focused on differentiating stressful from traumatic events and articulating the nature of the actual syndrome of PTSD, the very existence of this disorder raised the issue of how to best conceptualize the phenomenon of individuals who decompensate following exposure to trauma (1-5). For example, were trauma survivors to be viewed as psychologically damaged by the experiences that befell them or was it more appropriate to validate the experience of trauma from a humanistic and existential perspective by viewing their responses as an adaptation to frightening environmental events? These formulations had substantial implications for the treatment of trauma survivors (A.C. McFarlane and R. Yehuda, unpublished paper).

The question of how to view the trauma survivor was fueled by other social and moral agendas. For example, the human rights issues that emerged in the investigation of the effects of torture and political repression, civil rights issues, and the rise of feminism all resulted in an increasing urgency to address the plight of the traumatized individual (5). These social and political developments gave rise to a humanitarian concern that was championed by some proponents in the mental health field (45).

## NORMATIVE STRESS RESPONSE: IMPLICATIONS FOR THE ESTABLISHMENT OF PTSD

The earlier discussion highlights the historical, social, and political impetus for establishing a diagnosis that normalized an individual's symptoms following adversity. However, it was equally important to support the rationale for PTSD with scientific evidence about the direct effects of trauma. In the absence of empirical data about the effects of trauma, support for the original conceptualizations of PTSD was derived largely from other areas of theory and research.

A major intellectual cornerstone for early conceptions of PTSD was the field of biological studies of stress, which essentially justified a normal continuum of responses to adversity (46-48). Indeed, during the formative years of empirical biological studies of PTSD, most theoreticians hypothesized that neurobiological alterations in this disorder would be similar to those observed in studies exploring the neurobiology of stress (49-51). In particular, Selye's findings that any adversity could provoke a biological stress response (47) provided a scientific validity of the conception of PTSD that was derived from scientific observations and not from the need to advocate on behalf of victims. Furthermore, the Selye formulation suited the political and social agenda because it shifted the emphasis away from

the victim's vulnerability as the etiologic factor and focused on the responsibility of the perpetrator. The concept of an a priori biological stress response was an appropriate counterargument to critics who attacked the diagnosis of PTSD as having a political and philosophical origin, and it provided a post hoc scientific hypothesis that a biological response to trauma reflects a natural physiologic process.

A second body of literature that was compatible with Selye's ideas about stress was the life events literature (52-57). This literature provided indirect support for the notion of PTSD as a normative stress response by demonstrating a temporal relationship between adverse life events and the development of psychiatric and physical symptoms. Similarly, the crisis intervention and bereavement fields provided clinical support for the observation that transient traumatic events could produce symptoms that were amenable to intervention (2, 58-61). These fields were important to the mental health conceptions of PTSD because they provided a therapeutic model of how to address the "event" in treatment (62-67). Ultimately, the focus on the traumatic event brought to bear the need to carefully define the universality and commonalities inherent in confrontation with death and helplessness and underscored the need to examine typical reactions of trauma survivors. The crisis intervention literature formed the conceptual basis for viewing chronic PTSD as a prolongation of the normal response to stress, as well as for the use of preventive debriefing treatments that are currently used following exposure to trauma (67).

Although these areas of theory and research likely influenced original conceptions of PTSD, the relevance of these notions to current knowledge of PTSD requires reevaluation. For example, as discussed later in this article, empirical data on the biology of PTSD suggest a formulation that differs from the one that might have been predicted by the Selye model (26). Furthermore, studies of the prevalence, course, and comorbidity of PTSD have raised issues regarding the role of the stressor as the true etiologic factor in the development of this disorder. In the next section, findings from empirical studies of PTSD will be reviewed to illustrate that contrary to what might have been predicted at the time that the diagnosis of PTSD was established, many recent findings are inconsistent with the notion that traumatic events are the primary cause of symptoms and challenge the idea of PTSD as a typical stress response. Paradoxically, other data, such as the emerging evidence of a distinct and biological basis of PTSD, provide strong support for the validity of this diagnosis as well as insights into its nature.

#### EMPIRICAL OBSERVATIONS AND THEIR RELATIONSHIP TO PTSD SOCIAL AND CLINICAL THEORY

The conceptual underpinnings of PTSD have been addressed through a wide variety of research methodologies. First, prevalence studies have examined whether

PTSD is the expected response following exposure to trauma. Second, the role of vulnerability factors as predictors of PTSD has raised questions regarding whether PTSD is a distinct disorder that arises from exposure to trauma or, rather, some other factor or combination of factors. Third, studies on the longitudinal course of the disorder have provided information about the process that separates the chronic and disabling form of the disorder from the normal process of adaptation and restitution. Studies of comorbidity have challenged the idea that PTSD can be clearly differentiated from other psychiatric disorders. Finally, the biological studies of PTSD have allowed an independent test of the distinctness of the disorder and its similarity to neurobiological alterations observed following stress and trauma.

#### *Epidemiological Studies of the Prevalence of PTSD*

Epidemiological studies of "high-risk" individuals (e.g., defined as those surviving a traumatic event, such as combat veterans or rape victims) have generally suggested that the occurrence of PTSD following a traumatic event is the exception rather than the rule (6-16, 68, 69). In DSM-IV, estimates of the prevalence of PTSD among those exposed to a criterion A stressor range from 3% to 58%.

One of the classic epidemiological studies found a 15% prevalence of current PTSD and a 30% prevalence of lifetime PTSD among Vietnam veterans (15). The prevalence of PTSD in Desert Storm veterans was found to be substantially lower. Only 9% of Desert Storm soldiers could be diagnosed with PTSD 6 months following their return from the Persian Gulf (16). Studies of civilians have also shown that PTSD is relatively rare compared to the prevalence of trauma. A study of a random sample of 1,007 young adults from a large health maintenance organization in Detroit found that of the 39% of individuals exposed to trauma, only 23.6% had developed PTSD at some time in their life (12).

Studies of the prevalence of PTSD in civilian populations exposed to specific traumatic events yield similar statistics. The study of the Mount Saint Helens volcanic eruption demonstrated that only 3.6% of those exposed to this natural disaster had PTSD and that most of the symptoms had resolved in the 2 years following the traumatic event (8). Similarly, in a representative sample of 469 volunteer firefighters exposed to severe bushfires, a PTSD prevalence rate of 16% was observed immediately following exposure, and less than half of those cases had gone into remission at 42 months (29).

In evaluating these results it is important to consider the nature and severity of the traumatic event, since a number of investigators have documented a relationship between severity of the trauma and the development of chronic PTSD (70, 71). Indeed, it is likely that some types of events are more traumatic than others and produce different rates of PTSD. In contrast to the low prevalence rates reported for natural disasters, for

example, the lifetime prevalence rates of PTSD among crime victims has been estimated to vary from 19% to 75% (72). Also of note is the relatively high prevalence rate of persistent and chronic PTSD (47%–50%) among prisoners of war and concentration camp survivors (73–75). However, even among those who are exposed to very severe and prolonged trauma, there is usually a substantial number of individuals who do not develop PTSD or other psychiatric illnesses (76). Moreover, in documented epidemiological studies it is difficult to find even transitory symptoms in more than 50% of the population, and in the majority the symptoms will have usually resolved within 2–3 years (77). Thus, the available epidemiological data show that PTSD, and certainly chronic PTSD, is more unusual than usual following exposure to a variety of traumatic events.

### *Vulnerability Factors*

The observation that trauma is not a sufficient determinant of PTSD raises the possibility that there may be risk factors that account for a given individual's vulnerability to developing this disorder. A variety of possible candidates have been investigated including genetic risk factors (17, 78), family history (12, 24), the individual's personality (16, 79), past history of trauma (21, 22), past history of behavioral or psychological problems (6), nature of parental relationships (23), and other life events at the time of the trauma (17), as well as post-traumatic factors such as social support (80) and exposure to subsequent reactivating stressors (73, 81). There has been an increasing exploration of these issues in recent years that has suggested that some vulnerability factors exert their effects at relatively low thresholds (25), whereas others come into play at a relatively high level (82) of exposure.

To date, it is unclear whether the risk factors for PTSD suggest a specific predisposition to PTSD or reflect a general predisposition to mental illness that is triggered by adversity. Nonetheless, the issue that is raised by the demonstrated role of vulnerability factors is that decompensation following trauma is neither a random process nor an outcome entirely predictable by the nature of the traumatic event. This observation appears to call into question the most fundamental assumption of PTSD as potentially occurring in any individual as a result of exposure to a traumatic event.

### *Longitudinal Course*

The role of longitudinal studies is to describe the time course of symptom development and to determine whether there are a range of symptom patterns among victims who have undergone similar traumas (83). These studies provide information about the normative process of response to stress, the factors that might modify this process, and patterns of dysregulation. In particular, prospective longitudinal studies of non-treatment-seeking individuals have allowed a more systematic examination of Horowitz's influential formulation that following a tra-

umatic event, there is a process of oscillation between the states of intrusion and avoidance that is part of the normal process of integrating an experience of extraordinary magnitude (2). Horowitz's model, which has been considered to be one of the major ideological bases for PTSD (84), implied that the symptoms of PTSD are a continuation of the normal acute traumatic phenomena or, rather, the failure of restitution of this process. Thus, an implicit prediction of the model was that the severity and chronicity of symptoms would be proportional to the magnitude of the trauma. Although many studies have supported the view that the intensity of the trauma has a bearing on severity and chronicity of PTSD symptoms (2, 10, 70, 85), other studies have highlighted the complexity of this relationship and its lack of predictive power (7, 17, 21, 76, 83, 85). Furthermore, recent prospective epidemiological studies have given rise to the suggestion that the acute stress response may be quite different in individuals who develop PTSD and in those who do not.

For example, in a study of the survivors of a terrorist attack on a bus, Shalev (86) failed to demonstrate that the early intensity of the intrusive affects and cognitions related to longer-term outcome. A second study, of train drivers involved in fatal accidents, similarly demonstrated that the pattern of hyperarousal did not emerge simultaneously with the intrusions and that the avoidance phenomena developed after an initial delay (87). A third study demonstrated that the symptom profile that emerges within 2 weeks following a traumatic event may be quite different from that observed at 3-month follow-up (88). These observations imply that the intrusive phenomena in the immediate aftermath of a trauma may be different from those that occur 3 to 4 months later, or those that occur more chronically (29). Thus, the passage of time may be required before a demarcation emerges between the normal response and disorder.

The nature of acute traumatic reactions may be modified by a range of factors other than the severity of trauma, such as the history of prior trauma. Resnick and colleagues demonstrated that women with a prior rape history were three times more likely to develop PTSD than were women raped for the first time (25, 89). In that study, a prior rape history was associated with significantly lower cortisol levels in the hours after the trauma of rape. In contrast, the higher cortisol levels in women without an assault history were positively correlated with the severity of rape (89). These results suggest that acute responses to stress are not homogeneous and depend on variables other than those associated with the focal trauma. Furthermore, distinct biological responses to acute trauma that are a function of prior experience may affect the long-term course of PTSD.

The importance of these recent prospective studies is that they call into question the idea that PTSD is a continuation of the normal stress response. The very fact of heterogeneity of acute stress responses is incompatible with Horowitz's model of symptom formation in PTSD because it implies that certain acute responses to trauma may be adaptive, whereas others may be mal-

adaptive and result in disorder. Studies of the spectrum of acute stress responses and their long-term consequences will be critical for future conceptualizations of PTSD. For now, however, emerging data appear to challenge earlier ideas of the homogeneity and universality of the early response to trauma and raise the possibility that the emergence of chronic symptoms may be predicted by discrete biological and psychological features of the acute response to trauma.

### *Comorbidity*

The presence of psychiatric comorbidity is another issue that poses a dilemma for the initial conceptualization of PTSD as a normative stress response. The prevalence of comorbid psychiatric conditions has been investigated in a number of traumatized groups with PTSD, and these studies have found that anywhere from 50% to 90% of individuals with chronic PTSD also meet diagnostic criteria for another psychiatric disorder, including substance abuse (14, 15). Recent studies of community samples have also demonstrated high rates of comorbidity in both disaster victims and subjects selected in the North Carolina cohort of the Epidemiologic Catchment Area study (28, 68). In general, psychiatric comorbidity appears to develop over time in traumatized individuals with PTSD. In the study by North et al. of the victims of a mass shooting, for example, rates of comorbidity 1 month after the trauma were much lower than those in other populations studied (90). Thus, there may be a cascade in the months following the onset of PTSD that suggests the unfolding of a secondary psychopathological process. Regardless, the findings suggest that it is the exception rather than the rule for individuals to meet diagnostic criteria for PTSD in the absence of meeting criteria for another psychiatric disorder (91).

An important consideration in unpacking the question of psychiatric comorbidity is that these conditions may be a result of the overlap of symptoms between diagnoses as they are formulated in current nosologic conceptions and may not necessarily represent distinct disorders (91). Even so, the existence of these symptoms raises the possibility that they might be considered secondary to these comorbid disorders rather than being secondary to PTSD. A particular irony of the findings about comorbidity is that most patients would satisfy the diagnostic criteria for either depressive neurosis or anxiety neurosis, which would have been the diagnoses previously used to describe these patients. The formulation of DSM-I argued that these were sufficient to explain the long-term effects of traumatic stress, and there is some substance to this as a parsimonious argument.

The presence of psychiatric comorbidity is a complex issue that overlaps with some of the conundrums raised by the complex longitudinal course of PTSD and existence of vulnerability factors. The relative rareness of "pure PTSD" (i.e., a frequently used term that denotes a syndrome uncomplicated by the presence of symptoms of other psychiatric disorders), compared to the

presence of more complex forms, suggests that traumatic stress may precipitate a whole host of symptoms and conditions. As such, the emergence of PTSD following exposure to a trauma may represent the manifestation of an underlying diathesis rather than a normative adaptation to environmental challenge.

### *Emerging Neurobiology of PTSD*

Although a comprehensive review of the neurobiology of PTSD is beyond the scope of this paper, there are essential implications of neurobiological studies to the philosophical conceptions of PTSD. The most important conclusion to date from these studies has been that there is a distinct set of biological alterations that serves to characterize the state of prolonged or persistent symptoms in response to a traumatic event (92, 93). However, the nature of these disturbances suggests that the biological correlates of long-term psychological responses to trauma are far more complex than what was originally anticipated.

Initially, biological studies of PTSD were almost exclusively driven by the hypothesis that the alterations in symptomatic individuals would be analogous to those observed in animal and human studies of stress (48, 49, 94, 95). A series of biological findings have now emerged that show changes in stress-responsive systems that are quite different from what would be predicted on the basis of the stress literature (26, 27, 94, 96-104). Furthermore, the alterations in individuals with PTSD have been found to be distinct from those of similarly exposed individuals without PTSD (98, 99, 105-108). Moreover, the abnormalities observed in PTSD have been found to be different from those in other psychiatric disorders that have been associated with stress, such as mood and other anxiety disorders (98-104, 109-111). Together, these points suggest that the biology of PTSD is not simply a reflection of the normative biology of stress, as has been consistently hypothesized.

A salient demonstration of the previously discussed conclusions consists of the findings of hypothalamic-pituitary-adrenal (HPA) axis alterations in PTSD (26, 27). These studies have suggested that rather than showing the classic profile of increased adrenocortical activity and resultant dysregulation of this system described in studies of stress and major depression, PTSD sufferers show evidence of a highly sensitized HPA axis characterized by decreased basal cortisol levels (98, 100-103) and increased negative feedback regulation (99, 104). Studies of psychophysiology (105-108), electrophysiology (109-112), and neurochemical (109, 110, 113-118) alterations in PTSD have revealed similar abnormalities of the sympathetic nervous system and other neuromodulatory systems. Many of these studies have demonstrated that PTSD patients have exaggerated and more finely tuned biological responses both to stimuli that are reminders of the traumatic event (105-108, 116) and to perturbations, such as neuroendocrine challenge (113, 114) and other labora-

tory stressors like loud tones and exercise (108, 118). In the aggregate, the biological data suggest that individuals with PTSD may show a sensitization of several biological systems. The sensitization observed in PTSD can be contrasted to the attenuated or dysregulated biological and behavioral responses characteristically associated with habituation and adaptation to chronic stress (47) and major depression (119). Observations of biological sensitization have prompted theorists to more recently consider the application of animal models of kindling (120) and time-dependent change (121) as relevant to the neurobiology of PTSD (95, 122).

A concluding statement that can be made concerning the neurobiology of PTSD, on the basis of the findings to date, is that there are biological changes following exposure to a trauma that are particularly associated with the symptoms of PTSD and that do not appear to be present as a result of exposure to trauma per se. Although, as described previously in this article, earlier formulations might have seen a resemblance between the biology of stress and PTSD as a rationale for the diagnosis, the distinctness of the biology of PTSD and the biology of stress provides an even stronger validation of PTSD as a discrete disorder. It is likely, however, that the significance of the neurobiological data could not be fully appreciated without the increasing knowledge derived from descriptive studies. That is, in the context of the prevalence and other data that challenge the normative nature of PTSD, biological findings that depart from classic stress paradigms appear quite reasonable. In tandem, then, the descriptive and biological data have moved the field toward a more sophisticated notion of PTSD and a greater understanding of the heterogeneous nature of the response to traumatic events.

## CONCLUSIONS

The contribution of PTSD to psychiatry is that it provides an observational framework for studying the effects of stress and trauma. From a social and political perspective, PTSD as a concept has done much to assist in the recognition of the rights and needs of victims who have been stigmatized, misunderstood, or ignored by the mental health field. The existence of this diagnosis has allowed the emergence of the much needed data about the effects of trauma that did not previously exist and could not have been systematically collected without this diagnosis. The original conceptual basis of PTSD provided the constructs for the development of hypotheses about the effects of trauma that the field of psychiatry has had to address. To date, empirical knowledge derived from investigation of the effects of trauma has substantiated the importance of these humanistic concerns to a large degree and has allowed a further clarification of the concept of PTSD that extends its original conceptual roots.

It is now clear that PTSD does provide a model for a process of adjustment and destabilization to trauma that has biological, psychological, and phenomenologi-

cal dimensions. The biological investigations have demonstrated that the substrates of the disorder may not, in fact, be similar to the "normative stress response" described by Selye but, rather, may be a progressive sensitization of biological systems that leave the individual hyperresponsive to a variety of stimuli. PTSD has provided a model for the further development of a series of sophisticated ideas about the phenotypic expression of vulnerability, perhaps through use of models such as kindling (120), sensitization (120, 121), and parallel distributed processing (123). The finding that PTSD is not an inevitable consequence of trauma requires theorists to be more precise in their formulation of the effects of trauma and to search for other vulnerability factors that give rise to and perpetuate the course of PTSD.

The current challenge in the field of traumatic stress studies is to address the emerging empirical basis of PTSD as central to the validity of the disorder while placing in proper historical perspective that the diagnosis came to be by acceptance of the political and social rights of traumatized groups. Now that PTSD's place in psychiatric nosology is safely established, it is the scientific process that must provide the organizing philosophy for the field.

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