CHAPTER 1

PTSD and Complex PTSD
Symptoms, Syndromes, and Diagnoses

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Two decades have now passed since the medical–psychiatric term "post-traumatic stress disorder" (PTSD) was introduced into the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) of the American Psychiatric Association (1980). Since then, the proliferation of professional journals dedicated to trauma studies, reference books, and professional societies and the perfusion of interest in PTSD into mainstream areas of medicine, law, the social sciences, academic curricula, and social policy-making has become worldwide in scope (Wilson & Raphael, 1993; Wilson & Keane, 1997; Wilson, Friedman, & Lindy, 2001; Williams & Somers, 2002).

The United States, Australia, and Croatia, for example, have national centers for the study and treatment of PTSD, especially victims of war. Private hospitals have specialized inpatient units for the treatment of PTSD. International centers treat asylum seekers, refugees, and war and torture victims suffering from PTSD and its associated comorbidities (Wilson & Drozdek, in press). Access to PTSD informational materials is readily available on Internet websites (e.g., *ncptsd.org*). National media interest in PTSD intensified after the terrorist attacks on the World Trade Center towers on September 11, 2001. Prestigious newspapers, such as *The New York Times*, ran full-page articles on PTSD, its history, diagnosis, and treatment. Traumatic events of daily life or those of catastrophic proportions remind us that they are as much a part of history as the greatest scientific and cultural achievements of humankind.

Viewed historically, research conducted during the last half of the 20th century and the recognition of PTSD as an "official" psychiatric diagnosis gave birth to more vigorous and widespread scientific pursuits of knowledge about the disorder (Wilson et al., 2001). Although all of the stones of discov-

ery have not yet been overturned, scientific inquiry progresses toward understanding the conditions and consequences that produce the human suffering, injuries, and physical and mental scars that are manifestations of traumatization.

The purpose of this chapter is to describe PTSD and complex forms of PTSD in terms of stress response syndromes, symptoms, and diagnoses. I focus on explaining PTSD in its interrelated organismic aspects and how the consequences of trauma affect the mind, body, and spirit. I discuss PTSD as a syndrome of dynamically related psychobiological processes that include the brain, the nervous and hormonal systems, psychological systems of memory, cognition, emotion, motivation, perception, and behavioral expressions of the organismic changes caused by trauma. I discuss PTSD within a holistic framework of five synergistically related symptom clusters that were not present before the traumatic event. Finally, I present a set of recommendations regarding diagnoses of PTSD and other disorders.

THE PSYCHOBIOLOGICAL DIMENSIONS OF POSTTRAUMATIC STRESS DISORDERS

Figure 1.1 presents a simplified conceptual model of PTSD as a prolonged stress response syndrome. The figure diagrams the development and function of six categories of psychological processes that constitute the structure and mechanisms of PTSD as a stress disorder. These categories represent the epigenesis of posttraumatic stress syndromes.

Traumatic Experience

Traumatic events are defined by the existence of stressors that have differential effects on organismic functioning. Traumatic stressors exist on a continuum. As defined by the current criterion A1 for PTSD in DSM-IV-TR (American Psychiatric Association, 2000), "the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of the self or others" (p. 467; see Table 1.1). In response to these stressors, the person's reaction involves fear and horror (emotions), helplessness (a learned behavior), or denial (cognitive alterations and ego defenses). These psychological reactions to trauma constitute criterion A2 for PTSD (see Table 1.1).

Primary Psychobiological Substrates

There are two primary interrelated substrates of PTSD as a prolonged stress response system: biological and psychological. The *biological* process refers to the neurophysiological substrates that are innate, preprogrammed capacities of the organism. The *psychological* processes involve perception, memory,

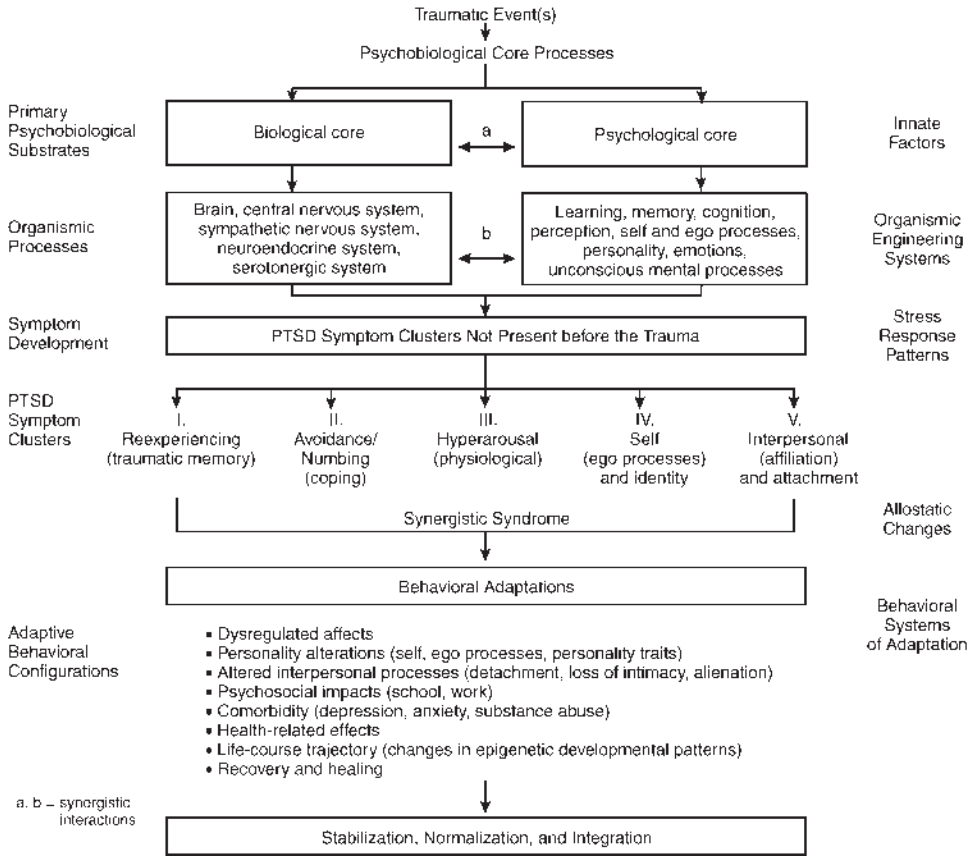

FIGURE 1.1. Psychobiological dimensions of PTSD.

cognition, learning, personality processes, and the self-structure. The two primary substrates are the organismic "soil" from which PTSD develops and forms adaptive patterns of behavior—the epigenesis of traumatic stress development.

Organismic Processes

The organismic processes in PTSD are manifestations of synergistic interactions between the biological and psychological systems in terms of adaptive responses to traumatic experiences. As organismic processes (see Figure 1.1), they reflect integrated holistic system dynamics; that is, the symptoms influence all aspects of psychological functioning. Assessing how PTSD symptom clusters "trigger" each other is important to accurate clinical assessment procedures.

TABLE 1.1. DSM-IV-TR Diagnostic Criteria for PTSD

A. The person has been exposed to a traumatic event in which both of the following were present:
   (1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
   (2) the person's response involved intense fear, helplessness, or horror. **Note:** In children, this may be expressed instead by disorganized or agitated behavior.

B. The traumatic event is persistently reexperienced in one (or more) of the following ways:
   (1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. **Note:** In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.
   (2) recurrent distressing dreams of the event. **Note:** In children, there may be frightening dreams without recognizable content.
   (3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). **Note:** In young children, trauma-specific reenactment may occur.
   (4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
   (5) physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
   (1) efforts to avoid thoughts, feelings, or conversations associated with the trauma
   (2) efforts to avoid activities, places, or people that arouse recollections of the trauma
   (3) inability to recall an important aspect of the trauma
   (4) markedly diminished interest or participation in significant activities
   (5) feeling of detachment or estrangement from others
   (6) restricted range of affect (e.g., unable to have loving feelings)
   (7) sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:
   (1) difficulty falling or staying asleep
   (2) irritability or outbursts of anger
   (3) difficulty concentrating
   (4) hypervigilance
   (5) exaggerated startle response

E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:
   Acute: if duration of symptoms is less than 3 months
   Chronic: if duration of symptoms is 3 months or more

Specify if:
   With Delayed Onset: if onset of symptoms is at least 6 months after the stressor

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Symptom Development: Five Stress-Related Clusters

PTSD symptoms develop in the aftermath of trauma (Green & Schnurr, 2000). New psychological, physiological, and behavioral patterns of reactivity develop that were not present before the trauma. The development of new symptoms may aggravate or add to preexisting psychological or psychiatric disorders. Clinically, PTSD can present with other Axis I and Axis II psychiatric disorders in many different combinations.

PTSD Symptom Clusters

The domain of PTSD symptom clusters is defined by five interrelated sets of symptoms: (1) the reexperiencing of trauma; (2) avoidance, numbing, and coping patterns; (3) hyperarousal; (4) self and ego processes; and (5) interpersonal affiliative patterns of attachment, bonding, intimacy, and love (Wilson et al., 2001). These five symptom clusters emerge from the primary substrates and are manifestations of changes in the organism's baseline functioning. In an overly simplified sense, posttraumatic adaptations constitute a new "set point" of behavioral functioning.

Adaptive Behavioral Configurations

Once PTSD symptom clusters develop from the primary psychobiological substrates, they comprise different and complex configurations of behavioral adaptation. These behavioral configurations include (1) dysregulated affects, (2) personality alterations, (3) altered interpersonal processes, and (4) a broad range of psychosocial consequences, including current health, academic, and occupational functioning (Schnurr & Green, 2004).

PTSD as a dynamic stress-response syndrome varies in severity and intensity and can develop at any point in the lifespan (e.g., childhood, adolescence, adulthood, old age; Pynoos & Nader, 1993). There are acute, chronic, and delayed-onset patterns of the disorder that may be episodic in manifestation. PTSD can be successfully treated and resolved, but it may reoccur on reactivation by new stressors, life crises, and trauma-specific stimuli or cues (TSCs) that reawaken the disorder.

PTSD AS A PSYCHOBIOLOGICAL SYNDROME

It is useful to understand PTSD as a normal, organismically based response pattern to extremely stressful life events. PTSD is a *psychobiological syndrome* that comprises an interrelated set of symptoms that cohere to form a prolonged stress reaction to trauma. As a syndrome, PTSD is synergistic in nature; the symptom clusters dynamically influence each other in behavioral manifestations.

Trauma affects all dimensions of behavioral functioning and psychological responses to physical and psychological injuries. The whole person is wounded by trauma; individuals have posttraumatic physiological reactions, emotions, perceptions, and cognitive attributions about the trauma experience that caused their injuries. Traumatic impact, such as emotional horror at witnessing a brutal unexpected death, for example, is not only emotionally overwhelming, distressing, and difficult to cope with but also triggers the release of neurohormones and activates "fight-or-flight" readiness. The traumatic event may also challenge belief systems concerned with meaning, faith, and expectancies about humanity and life itself. The effects of trauma can produce changes in worldview, beliefs about human nature, patterns of intimacy, interpersonal relationships, and conceptions of oneself and personal identity. Trauma does not occur in a vacuum or an isolated state. Its effects are multidimensional in terms of posttraumatic psychological functioning, influencing motivation, goal striving, and levels of consciousness about the self in the world.

Trauma's impact on the individual is holistic in nature, and effects on one part of the whole generate reciprocal effects on the other parts—a complex mind–body or psychobiological phenomenon. It is important to note that semantically the word "trauma" derives from Greek and Latin roots (cf. *traumatos*), in which "trauma" means injury to the body and results in a state of being wounded. Physical trauma causes injury to bodily integrity and normal biological functioning. Psychological trauma causes injury to the mind and its inherent processes and functions, including the ego, identity, and self-structure. Psychological trauma is caused by an *external* event that affects *internal* psychological phenomena at multiple levels of functioning and in conscious and unconscious modalities of awareness and behavior (Wilson et al., 2001).

Psychological and physical trauma perfuse the intricacies of psychological functioning like a stream of water cascading down a long hillside, with its shallow rocky areas, dropoff cliffs, collecting pools, and changes in the volume of flow and pressure. If one builds a dam to contain the stream, the water deepens and grows in pressure and potential to generate energy. If the dam gives way and collapses, the water may destroy everything in its path. Similarly, the "energy" of psychological trauma may dam up and burst forth in accumulated power to create deleterious effects to the well-being of the person and his or her relationships.

THE BRAIN'S COMMAND AND CONTROL CENTER OF STRESS RESPONSE

PTSD, as a psychobiological syndrome, is only one manifestation of the human stress-response system. Under conditions of stress, the organism energizes itself to cope with the need or perception to react in a situation of threat or

danger that poses a challenge to well-being, coping resources, and adaptation. The evocation of the stress-response system is psychobiological in nature: (1) the person appraises, perceives, or is physically affected by a trauma; and (2) the biological response is instinctively activated to function congruently with stressor demands. The brain, as the central processing unit (CPU), mobilizes the sympathetic nervous system (SNS) to do its job of "switching on" the "fight–flight" response. The body, under brain controls, responds to action in an automatic way, commanding the cardiovascular, hormonal, muscular, nervous, and other systems to confront the trauma situation. The brain's control center activates one of the many "computer programs" hardwired into the central nervous system to release neurotransmitters that carry chemical messages to activate and energize the body's adrenergic and noradrenergic response systems, which regulate heart rate, blood vessels and nerve conduction. The adrenergic systems are part of the hypothalamic–pituitary–adrenocortical (HPA) axis, extending in a reciprocal fashion from the brain to the adrenal glands located on the kidneys, which release a neurohormone, cortisol, in response to stress. In a systematic fashion, activation of the brain and SNS by a traumatic event switches on the control apparatus in the brain, increasing arousal and readiness to respond by neurons carrying informational messages to each other. These neuronal relay systems are like neighbors telephoning one another to warn of a dangerous situation. Upon perception of threat or danger, the brain releases corticotropin-releasing factor, known as CRF, which sends chemical messages to other areas of the brain, which are awakened and "called to duty" to deal with stress and trauma.

In the HPA axis of the body, the brain releases adrenocorticotropic hormone (ACTH; *adreno* = adrenal gland; *cortico* = cortical or brain; *tropic* = zone or area), which circulates through the blood to the kidney, triggering the release of glucocorticoids (*gluco* = glucose or sugar), which in turn increase metabolic energy for muscular and nervous system response to stress and trauma. When this occurs, the system is "fired up," "primed," and "energized," with the "engine," performing at a metaphorical high speed of "revolutions per minute" (RPMs). Once activated, the stress-response systems execute their preprogrammed functions under the central control of the brain. The neurohormonal programs activate their programmed sequence until switched off and the program terminates. However, one of the unique aspects of PTSD is that the stress-response program may not switch off but continue to operate as if the threat were actively persistent and still present as an anxiety- or fear-provoking danger.

The activation of the adaptive, psychobiological stress-response system is genetically designed to meet the external demands of trauma or those posed by situations of extreme stress. Under normal conditions, the hyperarousal state activated by threat or danger will decrease and return to its baseline function. The system responds by generating more "fuel" and "power" to cope until the demands have been met and they are no longer required. At this point, the system can return to homeostatic functioning.

HOMEOSTASIS AND ALLOSTASIS: POSTTRAUMATIC CHANGES IN BASELINE FUNCTIONS

Homeostasis describes the return to baseline or normal functioning. As traditionally conceptualized, homeostasis means equilibrium, balance, or average regulatory baseline function in biological systems (Miller, 1978). However, recent studies of PTSD have shown that following traumatic stress experiences, a homeostatic condition may not be reestablished, especially with traumas that are prolonged or repetitive in nature. The different types of events that define traumatic situations may cause *prolonged hyperarousal states* in which the organism does not return to homeostasis. Rather, it continues to function *as if* the trauma were continuous and ongoing in daily life. It is as if the "computer controlled" software in the brain's CPU fails, rendering the "on" switch locked into position so that the SNS is in continuous high gear, increasing the neurohormonal responses of the engineering system that governs adrenaline, noradrenaline, glucocorticoids, and other stress hormones (e.g., serotonin, acetylcholine, etc.) to the status of an emergency, even *after* the crisis has terminated. "Allostasis" refers to the posttraumatic stress-response pattern to seek stability in functioning following a change in the homeostatic baseline.

ALLOSTASIS: STABILITY AFTER STRESS-INDUCED ORGANISMIC CHANGES IN FUNCTIONING

> At every level of living systems numerous variables are kept in steady state, within a range of stability . . . when these fail, the structure and process of the system alter markedly.
>
> —J. G. Miller (1978, p. 38)

In recent research studies, Bruce McEwen (1998) has described the nature of allostasis and allostatic load mechanisms. He notes: "the core of the body's response to challenge—is twofold, turning on an allostatic response that initiates a complex adaptive pathway, and then shutting off this response when the threat is past. . . . However, if the inactivation is inefficient, there is overexposure to other hormones. Over weeks, months, or years, exposure to increased secretions of stress hormones can result in allostatic load and its pathophysiologic consequences" (pp. 171–172). It is possible to think of *one* aspect of PTSD as an organismic syndrome of *dysregulated affective responding*. The failure of the system to shut down and adapt is also known as a process of *allostasis*, wherein the organism attempts to recalibrate its "set point" of functioning. This "set point" is not homeostatic but a recalibrated and changed baseline level of functioning. In simple terms, the organism's "engine" has a higher level of nervous system arousal after extreme stress; the system is still on "red alert" status despite a change in the external environment. *Allostasis is an attempt at stability through the changes induced by the trauma experience*. The state of traumatization reflects the state of ongoing injury or

changes in system function until stabilization is achieved and normal functioning returns to baseline.

PTSD as a psychobiological state is a *dysregulated system* whose manifestations are biobehavioral, internal, and external in nature. Posttraumatic literally means "after injury," and in PTSD the prolonged stress-response patterns constitute a dynamic syndrome of symptoms and behavioral dispositions. It may include changes in personality (e.g., self, personal identity) and cognitive processing, memory, perception, motivation, and interpersonal relations. PTSD as a prolonged stress-response pattern disrupts *optimal functioning*. However, not all persons develop a full-blown diagnosable disorder; some will manifest acute reactions, acute stress disorders (ASD), and relatively high levels of stress reactions that do not meet the criteria for being considered pathological (i.e., partial PTSD).

It is important to understand that PTSD is not a unidimensional phenomenon. The psychological effects of trauma are expressed on all levels of organismic functioning: physical; psychological; social; spiritual; interpersonal; and systems of belief, ideology, values, and meaning. As a psychiatric disorder, PTSD is currently defined in diagnostic manuals as a limited set of clinical symptoms with specific sets of algorithmic criteria. To be considered an illness or psychiatric disorder, PTSD must impair areas of psychological functioning. More recently, complex PTSD has referred to a more inclusive set of symptoms, reactions, and behaviors that embrace trauma's impact to the self-structure, ego identity, and patterns of affiliation, intimacy, and attachment (Wilson et al., 2001).

THE CORE TRIAD OF PTSD SYMPTOM CLUSTERS

What are the core characteristics of PTSD as a stress disorder that distinguish its uniqueness? First, PTSD is a normal, biologically hardwired pattern of reactivity to extremely stressful situations. The stress patterns have subtypes of symptom constellations, depending on the nature of the trauma and the nature of the person. The events that are scientifically defined as traumatic are anchored at the extreme end of the stressor continuum. Generally, they are events or experiences that involve threat or danger to physical integrity and psychological well-being. Since the initial definition of PTSD in DSM-III of the American Psychiatric Association (1980), revisions have been made to the stressor criterion (A1), underscoring the need to provide conceptual clarity about the causality of the disorder. The growth of empirical research has established that the lifetime exposure to events associated with the development of PTSD are not uncommon, with 60.7% of men and 51.2% of women experiencing situations that could cause either acute or prolonged stress reactions (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995).

The lifetime prevalence of rates of PTSD varies greatly, from 1% to 30% or more for combat veterans and other trauma populations with high levels of

trauma exposure. Across several studies, the highest lifetime prevalence for PTSD is associated with the sudden loss of a loved one; interpersonal assaultive violence; sexual assault (Breslau, 1998); and exposure to massive, catastrophic trauma, such as combat, torture, the atomic bomb at Hiroshima in 1945, the Nazi concentration camps, the terrorist attacks on the World Trade Center in 2001, and the 1993 bombing of the Murrah Federal Building in Oklahoma City (North & Pfefferbaum, 2002; Friedman, 2000). Once exposed to major trauma and the onset of PTSD, the individual is at a substantial risk to develop other psychiatric disorders, especially major depression, generalized anxiety disorder, and substance abuse (Breslau, 1998; McFarlane, 2001). As a conservative rule of thumb index, about 10% of women and 5% of men will develop PTSD during their lifetimes (Kessler et al., 1995). For severely traumatized persons, especially those who are not treated and/or who develop additional psychiatric problems (i.e., comorbidity) such as major depression or alcoholism, the symptoms and deleterious effects of PTSD can last a lifetime (Friedman, 2000). Moreover, until the condition stabilizes and the normal processes of stress recovery are set into motion, PTSD has the power to impair functioning, disrupt lives, and alter an individual's sense of identity, self-worth, and systems of faith, meaning, and purpose in living. What makes PTSD unique as a psychiatric disorder is that the syndrome is caused by an identifiable external force that may be an act of God, a random act of nature, or the willful malevolence of human design.

The nature and process of adaptation to trauma varies according to age, ego strength, and prior traumatization (Friedman, 2000). Posttraumatic adaptive behaviors have many configurations as psychobiological syndromes. The constellation of symptoms in PTSD is determined by several factors: (1) the pretraumatic mental health and personality characteristics of the person; (2) the nature, duration, and severity of the traumatic event; (3) the posttraumatic recovery environment and access to support, treatment, and resources to restore normal functioning; (4) the level of injury sustained to the body and the psyche, especially internal, core aspects of personality, identity, and ego processes; and (5) critical stages of epigenetic lifespan development (Erikson, 1968; Pynoos & Nader, 1993; Green, Wilson, & Lindy, 1985; Wilson et al., 2001; Wilson, 2002b; Wilson, 2003b; Breslau, 1998). These factors determine the configuration of PTSD symptoms that have subtypes and specific typologies in which the dynamics show different forms of modal expressions in behavior. For example, some persons will reexperience the trauma more frequently and intensely in flashbacks, intrusive recollections, and dreams than others. Some individuals will manifest profound numbing and extreme avoidance tendencies, and for others, their self-structure will be fragmented and dissolved by assaults on their personhood (Kalsched, 1996). Thus, depending on the depth and thoroughness of psychological assessment, PTSD symptoms will show *variable profile configurations*, with different constellations of symptoms being more pronounced than others. These variations in profile configurations are expressions of the multidimensional psychobiological nature of PTSD at any moment in time.

PTSD IS A MULTIDIMENSIONAL EPISODIC STRESS SYNDROME

Clinical assessment procedures consider the predictable fluctuations and cycles of symptom presentations (Wang, Wilson, & Mason, 1996). PTSD is not a static, unidimensional entity but a fluctuating, episodic multidimensional stress-response pattern that affects integrative psychological functioning on many levels: (1) memory, cognition, and information processing; (2) perception; (3) affect regulation; (4) motivational striving; (5) coping and ego-defensive functioning; (6) ego processes and personal identity; (7) stress tolerance capacities; (8) interpersonal relations and capacity for attachment and intimacy; and (9) life-course trajectory in the epigenesis of ego and personality development (Wilson et al., 2001). The multileveled impact of PTSD on organismic functioning produces syndrome constellations that are built on the triad of core PTSD symptoms: (1) reexperiencing, reliving, or reenacting traumatic memories; (2) avoidance tendencies, psychic numbing, and coping behaviors; and (3) psychobiological changes and physiological reactivity (hyperarousal) that was not present before the traumatic experience.

THE PSYCHOLOGICAL ARCHITECTURE OF PTSD

The core triad of PTSD symptoms are like a three-pronged support base for the larger syndrome, which sits atop its foundation. The "psychological architecture" of PTSD includes the tripod foundation and horizontal or vertical extensions that, figuratively, have different shapes to accommodate the functions of the self. The architectural principle of "form follows function" applies to understanding the structure of PTSD. *As a stress syndrome, PTSD is a psychobiologically driven organismic function of adaptation to abnormal, excessive, or extreme stressor events that tax individual coping resources*. The function of the stress-response syndrome is to meet environmental and intrapsychic challenges presented by the trauma (Kalsched, 1996). As a prolonged stress pattern, PTSD is a behavioral organization (i.e., structural form) adapted to meet allostatic loads imposed on the system. To be considered a psychiatric disorder, the symptoms must cause clinically significant impairments in functioning (i.e., DSM-IV-TR, PTSD Criterion F; see Table 1.1). The behavioral organization of PTSD constitutes its psychological structure, which can assume many complex forms as adaptive processes.

The psychological architecture of PTSD can be further analyzed to understand the relationship of structure to function. The triad of PTSD symptoms reflects the basic psychological processes of (1) memory, information, and cognitive processing; (2) coping and ego-defensive mechanisms; and (3) the mechanisms of the central nervous system associated with the capacity of either *external* or *internal* stimuli (e.g., memories of the traumatic event) to reevoke and switch on the preprogrammed neurophysiological stress re-

sponse. The symptoms are synergistic processes that influence each other tridirectionally in terms of manifestations. For example, a reminder of a traumatic stressor will activate the adrenergic response system, and heart rate will increase. The person will experience increased physiological arousal and a readiness to respond to a situation or an evoking stimulus. Similarly, a sudden increase in heart rate, anxiety, or physical arousal may automatically be associated with thoughts or memories about the trauma, which, in turn, rekindle affects. In either case, external or internal activators of the trauma experience may then lead to avoidance behaviors, such as removing painful memories, avoiding things that are associated with the event, or using alcohol to alleviate emotional pain, tension, and psychic distress. In this manner, we can see that the symptom triad of (1) *reexperiencing* (traumatic memory), (2) increased *physiological reactivity*, and (3) *avoidance* behaviors are dynamically interrelated. However, before further examining their synergistic nature, I discuss each of the core triad symptom clusters separately, as well as two additional ones *not* officially listed in the current DSM-IV-TR (American Psychiatric Association, 2000) diagnostic manual. The two additional clusters include traumatic damage to the self-structure (e.g., ego identity, self-esteem) and interpersonal affiliative patterns (e.g., bonding, attachment, intimacy, and love).

DIAGNOSTIC MANUALS (DSM-IV AND ICD-10): CRITERIA, SYMPTOMS, AND ASSOCIATED FEATURES

Since the advent of PTSD as a diagnostic illness, first classified in DSM-III (American Psychiatric Association, 1980), the manual for diagnosis has undergone revisions to make the criteria congruent with clinical wisdom and scientific findings from research studies (American Psychiatric Association, 1980, 1987, 1994, 2000). Psychotherapists, clinicians, counselors, attorneys, academics, and administrative personnel routinely rely on these criteria in their work, and they utilize the official diagnostic criteria listed in either the DSM-IV or the *International Classification of Diseases* (ICD-10; World Health Organization, 1992). The criteria are set forth as a decision-making algorithm of the *minimal* number of symptoms that must be present to properly establish a positive diagnosis and to differentiate PTSD from other Axis I (major clinical disorders) or Axis II (personality disorders) psychiatric conditions. The diagnostic manuals also include sections on "associated features," which are narrative descriptions of other symptoms or behaviors that appear with the stress disorder but that may not be sufficient or necessary in themselves to constitute a prime characteristic of the syndrome for diagnostic purposes. The diagnostic criteria for each of the core triad of PTSD symptoms also share some of the same symptoms and features of other disorders, for example, depressive disorders, anxiety disorders, and specific personality disorders, such as borderline personality disorder and paranoid personality disorder.

To ensure some measure of conceptual clarity and clinical usefulness, I present a review of the current DSM-IV (American Psychiatric Association, 1994, 2000) diagnostic criteria for PTSD and then a much expanded set of criteria based on an allostatic model of PTSD by Wilson and colleagues (2001). To aid the discussion, refer to the DSM-IV-TR (American Psychiatric Association, 2000) diagnostic criteria for PTSD presented in Table 1.1.

TRAUMATIC MEMORY: REEXPERIENCING, RELIVING, AND REENACTING THE TRAUMA EXPERIENCE

How do persons reexperience traumatic events? The hallmark feature of PTSD is traumatic memory and forms of reliving, reexperiencing, or reenacting aspects of the original trauma. The memory of the trauma experience then becomes encoded in the brain and body in a variety of different ways. As a part of the syndrome, the individual reexperiences or reenacts the trauma in psychological systems: memory, perception, affect, cognition, motivation, and interpersonal social relations.

PROCESSING, STORAGE, AND RETRIEVAL OF TRAUMATIC MEMORIES

Once encoded in memory, the trauma may be behaviorally reexperienced in different ways: in frequency, in duration, in severity, in intensity, and in its influence on behaviors. *Reexperiencing phenomena involves cognitive processing, information storage, and retrieval from memory*. It can also be revivified at different levels of awareness (i.e., conscious, preconscious, and unconscious). Unconscious forms of reexperiencing are especially subtle and intricately expressed in overt reenactment manifestations in behaviors that have a psychological isomorphism with actions during the trauma (Blank, 1985b; Wilson & Zigelbaum, 1986; Wilson, 1989; Wilson et al., 2001). Unconscious reenactments of trauma parallel specific aspects of the actions that occurred during the chronology of the trauma experience (Wilson, 1989). The posttraumatic dynamics of the trauma experience can occur at different time intervals, ranging from peritraumatic reliving immediately after the onset of the trauma to days, weeks, months, or years afterward (Marmar, Weiss, & Metzler, 1997). As discovered by Freud (1910/1957), Janet (1890), Jung (1928) and other pioneering analysts, the unconscious is timeless, and therefore a traumatic memory can intrude involuntarily and unexpectedly at any point in the life cycle, especially when other life crises, traumas, or similar experiences rekindle memory and activate thoughts and affects associated with the disturbing experience—even decades after its disruptive or pathological effects have terminated. In this sense, it is meaningful to speak of traumatic experiences as

timeless, having the power to resurface in conscious actions and behavioral dispositions at any point in the life cycle (Wilson et al., 2001).

DSM-IV-TR PTSD B CRITERIA: REEXPERIENCING TRAUMA—FIVE CLUSTERS WITH 16 SYMPTOM FORMS

DSM-IV-TR (American Psychiatric Association, 2000) diagnostic criteria for the core triad of PTSD symptoms includes traumatic memory, that is, the phenomenon of reexperiencing, reliving, or reenacting the traumatic event. DSM-IV-TR lists five sets of symptom clusters for the reexperiencing category, which actually contains 16 individual forms of consciously or unconsciously reliving trauma. Specifically, the diagnostic criteria indicate that the traumatic event is "persistently reexperienced" in one or more of five symptom clusters: (1) memory, (2) dreams, (3) reliving, (4) increased psychological (emotional) distress, and (5) increased physiological arousal.

Traumatic Memory

The first PTSD reexperiencing criterion, B1, involves "recurrent and intrusive distressing recollections" of the event, which include three specific subtypes, "images, thoughts, or perceptions." The B1 reexperiencing criterion concerns all aspects of traumatic memory. Note that this includes raw images, such as a visual memory of parts of the experience, perceptual processes (i.e., visual, olfactory, sensory, tactile, or kinesthetic), and organized or disorganized forms of thought (see Table 1.1).

Traumatic Dreams

The second PTSD reexperiencing criterion, (B2), involves these same traumatic memory processes in dream and sleep cycles: "recurrent distressing dreams of the [traumatic] event" or anxious dreams in children that express themes directly or indirectly in connection with the trauma. By logical inference, dreaming is a form of thought during the sleep cycle, and so dreams also contain "images, thoughts, and perceptions."

Emotional and Behavioral Forms of Reliving Trauma: Two Prongs

The third PTSD reexperiencing criterion, B3, is the most complex of the traumatic memory clusters. Its complexity is due to the two-pronged nature of this particular criterion, which states that the person reexperiences by "acting or feeling as if" the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes,

including those that occur on awakening or when intoxicated)." Here, it is noted that the PTSD B3 criterion includes subtypes of reexperiencing the trauma that are *behavioral* (i.e., "acting as if") and *affective* ("feeling as if") with four distinct subforms involving: memory (reliving); perceptions (illusions as false sensory perceptions); hallucinations (hearing voices or seeing persons, places, or objects) that are anchored in the trauma experience; and dissociative flashback episodes. Trauma-based hallucinatory experiences are different from psychotic hallucinations precisely because the content of the hallucinations is trauma rooted and clearly identifiable as factually "real" upon careful analysis of the trauma history. Many PTSD patients have been misdiagnosed as paranoid schizophrenic, psychotic, or delusional because of their reports of trauma-based hallucinatory experiences (Blank, 1985b; Wilson, 1988).

The last of the PTSD B3 subtypes involves dissociative episodes, commonly known as "flashbacks," "zoning out," or "switching off." Dissociative psychological processes refer to an alteration in mental state—a change in the normal personality and behavior of the person and how he or she masters experience and the specific style and quality of his or her cognitive processes, especially the integrative functions of consciousness. In PTSD-related dissociation, a "dis-association" occurs upon reactivation of traumatic memory through associative learning processes. TSCs (Wilson et al., 2001) can evoke disturbing thoughts, feelings, and memories of the traumatic experience. Once activated, either consciously or unconsciously, the painful recollections and affects may be so anxiety producing and profoundly distressing to the person that he or she uses dissociative processes to protect him- or herself from the painful reliving of the trauma. It is clinically advantageous to view dissociative processes as self-protection mechanisms. The person alters his or her awareness of what he or she is feeling or reliving or fears will reoccur in the future. *Dissociation means that individuals' awareness of themselves or the environment (i.e., specific situations, places, persons, activities) alters and transforms into a* qualitatively *different state of being, which is discernible to others who are attuned to their personality functioning*. In depersonalization, for example, individuals alter awareness of themselves and feel as though they are observing their actions "outside their bodies," in a detached dream-like state of awareness. In such an altered state of consciousness, a PTSD (B3) dissociative flashback, the person may feel that he or she is in a surreal daze in which awareness of reality and the sequence of events that occurred while dissociated were reduced. The dissociative changes in conscious self-awareness and self-monitoring capacity are metaphorically similar to a dimmer switch on a light; the more it is turned down, the lower the available lighting by which to see one's environment. In dissociative flashback episodes in PTSD, the dimmer switch may alternate in brightness intensity, from total darkness to intolerable brightness. Moreover, dissociative episodes may occur when the person has lowered his or her cognitive controls through alcohol consumption or on awakening from sleep and transitioning from one state of mental activity (hypnogogic) to another (hypnopompic).

Increased Psychological and Physiological Distress

The last two (B4, B5) PTSD symptom categories of the traumatic memory cluster are psychobiological changes in response to "internal or external cues that symbolize or resemble an aspect of the traumatic event." Specifically, the B4 reexperiencing criterion concerns increased emotional reactivity upon reactivation of memories by associative learning—TSC or a stimulus of a more generalized nature, which triggers the painful emotions originally experienced at the time of the traumatic event (i.e., peritraumatic affectivity) or afterward (Marmar et al., 1997). Similarly, the B5 reexperiencing criterion reflects increased "physiological reactivity" (hyperarousal) in association with TSCs or other activating stimuli. The increased physiological reactivity (e.g., blood pressure, sweating, heart palpitations, hyperventilation, urinary urgency, flushing, etc.) indicates that the fight–flight stress response patterns have been switched on or amplified in intensity (Friedman, 1994). As part of allostasis following trauma, the body remains in a state of readiness (i.e., by degrees of hyperarousal potential). When an event, TSC, or situation signifies a potential threat or activates encoded memories of the traumatic experience, the command and control centers of the brain activate the engineering mechanisms of the adrenergic response system to execute their functions. Like soldiers scurrying to their battle stations, the neuronal messengers of the stress-response system respond to a potential red-alert status and prepare the organism to cope and adapt as necessary. As a posttraumatic stress response symptom, increased physiological reactivity "on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event" is a biobehavioral indicator of the specificity of PTSD as an integrated organismic state whose baseline functions were shifted by trauma and recalibrated at a new set point level. The new level is an index of allostatic load and recalibration of the organisms set point of responsiveness to the perception of threat, harm, or challenge to its integrity as a system (McEwen, 1998).

UNDERSTANDING AND ASSESSING THE 16 FORMS OF REEXPERIENCING TRAUMA

In summary, the reexperiencing cluster of PTSD diagnostic symptoms (criteria B1–B5) reflects the manner in which traumatic memory functions. Examination of each of the separate symptom clusters reveals a total of 16 different forms of reexperiencing a trauma in conscious or unconscious behavioral manifestations, altered states of awareness (dissociation), sensory and perceptual processes, and somatic symptoms of hyperarousal. These 16 symptoms are listed for purposes of clarity in understanding their structure and function in traumatic memory. They are important, too, as part of any clinical assessment process for PTSD diagnosis. Currently, DSM-IV-TR requires only one symptom from this entire cluster to establish the diagnosis of PTSD. Note: All symptoms are in direct or indirect reference to the traumatic event.

- B1. *Intrusive distressing recollections of trauma*—three symptoms
  1. Images (raw images, primary process thinking, visual or fragmented memories, "flicker-flashes," freeze-frame images)
  2. Thoughts (coherent, disorganized)
  3. Perceptions (sensory/perceptual processes, e.g., tactile, kinesthetic, visual, olfactory, auditory)
- B2. *Dreams associated with trauma*—3 symptoms
  4. Images (same as B1)
  5. Thoughts (same as B1)
  6. Perceptions (same as B1)
- B3. *Response predisposition: Acting or feeling "as if"*—4 x 2 modalities: modality I, "acting as if," or modality II, "feeling as if" = eight symptoms
  7 and 8. Reliving (revisualization or acting out prior trauma experiences)
  9 and 10. Illusions (perceptual, sensory, etc.)
  11 and 12. Trauma-rooted hallucinations (trauma-based sensory/perceptual hallucinations)
  13 and 14. Dissociative processes (depersonalization, derealization, amnesia, etc.)
- B4. *Increased psychological distress on exposure to trauma-related stimuli*—one symptom
  15. Anxiety, fear, sadness, terror, or other negative affects
- B5. *Increased physiological reactivity on exposure to trauma-related stimuli*—one symptom
  16. Somatic manifestations of hyperarousal states evoked by trauma-relevant cues (e.g., sweating, heart palpitations)

THE RELATIONSHIP BETWEEN REEXPERIENCING TRAUMA AND DEFENSIVE AVOIDANCE

It is important to the understanding of PTSD to note that the distress associated with reexperiencing a traumatic experience cannot be tolerated for prolonged periods of time without periods of rest, relief, and the ability to resume activities of daily living.

The wisdom of the organism is that there are many forms of coping and of warding off the pain of reliving traumatic life experiences. Traditionally, these intrapsychic and behavioral activities have been studied as coping adaptations to stress or as ego defensive processes associated with threat, anxiety, and somatic states of tension, agitation, and intolerable affects (Wilson et al., 2001). In a broader perspective, reexperiencing phenomena are reciprocal in structure to psychic mechanisms that are designed to control degrees of distress produced by the stress-response system. McEwen (1998) found that chronic activation of the stress-response system generates wear and tear on the

organism, especially if the system fails to switch off after trauma or cannot do so because of repeated demands for use or because of the breakdown of the neurohormonal engineering system, when parts lose their effectiveness, fail, or just wear out from overuse. Therefore, we can view the avoidance cluster of PTSD diagnostic criteria as forms of coping with dysregulated stress-response systems. These PTSD symptoms, referred to as "avoidance and numbing behaviors," were not in the organism's repertoire prior to trauma and can be adaptive or maladaptive in nature.

DSM-IV-TR PTSD C CRITERIA: AVOIDANCE AND NUMBING OF RESPONSIVENESS NOT PRESENT BEFORE THE TRAUMA

How do persons distance and protect themselves from traumatic impact to their sense of well-being? The diagnostic C criteria of DSM-IV-TR (American Psychiatric Association, 2000) consists of seven symptoms of avoidance mechanisms and changed patterns of coping with stress that are different from pretraumatic baseline (see Table 1.1).

Trauma-Related Active and Passive Avoidance Tendencies

The PTSD C1 and C2 criteria involve efforts to avoid "thoughts, feelings, conversations . . . activities, places, or people" that are associated with the trauma. These symptoms reflect the aversive nature of reexperiencing trauma and the efforts the person undertakes to avoid being exposed to reminders that would stimulate unwanted memories and feelings associated with the trauma experience.

Loss of Memory and Inability to Recall Aspects of the Trauma Experience

The PTSD C3 avoidance criterion, "inability to recall an important aspect of the trauma," is indicative of amnesia or gaps in the chronology of the trauma experience itself. The inability to recall important aspects of the trauma may be the product of dissociated affects and memories of the most critical, salient, or overwhelming moments of trauma experience. The "loss" of memory for critical events may be caused by repression, blocking, or denial or by state-dependent learning (Wilson, 1989), in which the informational content of experience was encoded in extreme states of hyperarousal (e.g., terror of annihilation, defenselessness at the witnessing of a horrific death, or the total disavowal of the unimaginable circumstances that occurred within a relatively brief or protracted period of time). In a clinical situation, amnesia may be difficult to assess until the trauma narrative unfolds and the missing pieces of the jigsaw puzzle can be assembled into a complete form. In many cases, gaps in

the chronology of the trauma story are indications of the most crucial memories that required removal from active storage in order to cope with the extraordinary intensity and powerful impact of stressors to individual well-being.

Diminished Interest in Normal Activities of Daily Living following Trauma

How does PTSD affect activities of daily living? The PTSD C4 avoidance criterion specifies "markedly diminished interest or participation in significant activities." This criterion reflects a disengagement from activities that the person enjoyed or participated in prior to the traumatic experience. The loss of interest in previously enjoyed hobbies, recreation, or daily routines is often a manifestation of depression and a desire to withdraw from others in order to "lick one's wounds." A traumatic experience may precipitate a reordering of priorities, and activities once valued may not seem enjoyable, desirable, appealing, or meaningful. Nevertheless, the salient feature of the C4 avoidance criterion is that there are readily discernible posttraumatic changes in behavior.

Social Detachments and Emotional Anesthesia (Psychic Numbing)

How do PTSD and avoidance symptoms affect social and interpersonal relations? The PTSD C5 and C6 symptoms of avoidance and psychic numbing are manifestations of changes in *interpersonal* relations and *intrapsychic* capacities to tolerate affect. The C5 avoidance criteria, "feelings of detachment or estrangement from others," is characteristic of tendencies of isolation, withdrawal, social disengagement, preference for solitary activities, and geographical distance from others in a safe or secured environment. On the other hand, the C6 avoidance criteria, "restricted range of affect," is a form of shutting down emotional responsiveness; it is emotional anesthesia, psychic closing-off, or a restricted capacity for affect tolerance. Manifestations of psychic numbing take many forms, including a loss of normal capacity to experience emotions, diminished sensuality and sexuality, a loss of spirituality, and the outward appearance of being emotionally flat, nonresponsive, vapid, unfeeling, indifferent, cold, and lacking in vitality. These emotional states can be considered as coping efforts to control the level of hyperarousal inherent in PTSD as a dysregulated stress-response syndrome. The person afflicted with PTSD over controls his or her emotional responsiveness by preemptive mechanisms to prevent feeling vulnerable to the internal distress of traumatic memory and forms of reexperiencing behavior. In a simplified way, the nonverbalized thinking is: "if I don't feel, I cannot be hurt any further than I currently am by the trauma." In its basic function, psychic numbing is a security operation, attempting to impose controls on the neurohormonal engineer-

ing systems that have failed to switch off, leaving the person feeling vulnerable to turbulent affects that persist *as if* the trauma is still occurring. In severe cases of chronic PTSD, the person may feel that to become emotionally vulnerable to the painful, unresolved aspects of trauma is tantamount to dying. Elsewhere, this phenomenon has been described as self-dissolution and deintegration, wherein the component parts of the self-structure (e.g., coherence, connection, autonomy, vitality, agency, etc.) fragment and result in a loss of ego continuity, self-sameness, and personal identity (Wilson, 2002b, 2003b; Wilson & Drozdek, in press).

Psychological Myopia: Changes in Future Orientation

The last avoidance criterion for PTSD, C7, is identified as a "sense of a foreshortened future." This symptom means that the person feels as though his or her expected course of lifespan development will be truncated, short-lived, or profoundly altered in uncertain and anxiety-provoking ways. The specter of a sense of foreshortened future may lead to an urgency to live life fully in the present and, consequently, to engage in risk-taking and acting-out behaviors. When a strong sense of foreshortened future predominates the individual's future orientation and planning, the immediacy of the present is overvalued. The result is *psychological myopia*, in which tomorrow may never exist in the eyes of the person. Such a worldview and diminished sense of future orientation may mask depression, feelings of learned helplessness, and loss of control over outcomes in daily living. In response, the individual may engage in risky, impulsive, and self-destructive patterns of behavior.

DSM-IV-TR PTSD D CRITERIA: PERSISTENT SYMPTOMS OF INCREASED AROUSAL NOT PRESENT BEFORE THE TRAUMA

How do persons manifest hyperarousal behaviors as stress-related changes in behavior? Five interrelated symptoms define DSM-IV-TR PTSD D criteria of "persistent symptoms of increased arousal" that were not present before the trauma. These symptoms reflect psychobiological changes in allostasis, hyperarousal of the adrenergic response system, and their behavioral expressions as PTSD symptoms (see Table 1.1).

Sleep Cycle Disturbances

The PTSD D1 hyperarousal criterion (difficulty falling or staying asleep) reflects sleep disturbances and includes disruptions of the early, middle, or terminal phase of the cycle. Accompanying the difficulties with sleeping are night sweats, problems returning to sleep upon early awakening, nightmares, night

terrors, somnambulism, agitation, and restless activity while sleeping, which may be attended by vocalizations (e.g., gasps, screams, crying, talking, making references to the trauma experience, etc.).

Anger, Irritability, and Hostility

The PTSD D2 hyperarousal criterion, "irritability or outbursts of anger," is another manifestation of dysregulation of the stress-response system. Persons with PTSD are sometimes quick to react with irritability, hostility, anger, cynicism, confrontation, and anxious agitation at annoying circumstances. They are often restless and impatient. They have proverbial short fuses, quick tempers, and "fast draw" dispositions. Recent studies (Bremner, 2002) have shown that the basal ganglia area of the limbic system associated with anger and aggression are effected as part of the prolonged stress-response pattern. For some persons, especially those with a history of combat, aggression, or self-defense being necessary to survival, the subcortical brain structures associated with aggression appear to be in a state of kindling, a neurological ready-alert mode of functioning. On provocation, even minimal, they may be predisposed to act automatically in irritable, angry ways that, in turn, may trigger a sequence of increased aggressiveness.

Impairment in Cognitive Processing of Information

How do states of hyperarousal in PTSD influence thinking, concentration, academic performance, and other cognitive functions? The PTSD D3 criterion, difficulty concentrating, is another manifestation of hyperarousal. We can think of this symptom as part of a larger constellation of cognitive processing deficits in PTSD, which include difficulties in encoding, processing, and retrieving information. These cognitive impairments of executive function also include attention deficits (e.g., shifts in focus, drifting, inability to solve problems or follow directions, etc.). Students who have PTSD may also manifest hyperarousal in the form of irritability, anxiety, tension, agitation, inattention, "zoning out," oppositional tendencies, problems with conduct and following rules, restlessness, and discomfort associated with reexperiencing phenomena. PTSD symptoms of this type are easily misdiagnosed as hyperactive attention-deficit disorder (ADD or ADHD) because of the similarity in overt behavioral patterns. Problems of cognitive information processing in PTSD can be understood as the effect of accumulative, hyperarousal states jamming the information processing centers of the brain, impairing concentration, attention, and memory. The somatic dysregulation of affect in PTSD creates "noise," "signals," or "interference," which disrupts the normal operation of the brain's CPU for high-order cognitive processes. The dysregulated affects in PTSD are like weather storms that disrupt radio and television reception, resulting in "lost" information and poor quality reception.

Hypervigilance: Excessive Alertness to Threat and Danger and Readiness to Respond

The PTSD D4 (hypervigilance) and D5 (exaggerated startle response) criteria both reflect psychobiological changes in behavioral dispositions. These symptoms can be considered as manifestations of hyperarousal of the SNS, reflecting allostatic processes of altered thresholds of response and initial response patterns (Wilson et al., 2001). The person suffering from PTSD continues to function in a "red alert" status of readiness, behaviorally primed for another stressful event.

Hypervigilance is a behavioral disposition and a readiness to respond to stimuli, especially cues that have trauma-specific relevance to their traumatic experience (TSCs). As part of PTSD-related hypervigilance (*hyper* = excessive; *vigilance* = alert, awake, watchful), the individual is on guard and scans the environment for cues, signs, or situations that signify a threat or potential problem. Hypervigilance consists of cognitive, affective, somatic, and behavioral dimensions. In regard to perception and cognition, persons with PTSD automatically, and often unconsciously, scan the environment for signs of threat. Based on their own trauma experiences and the activation of the fight–flight stress response (i.e., hyperarousal), they have a faster recognition threshold of threat stimuli. Upon perception, or actual recognition of a threat source, affective responses intensify—typically fear, anxiety, anger, or terror. Somatically, the increase in hyperarousal is experienced in physical reactions of muscle tension and increased heart rate, blood pressure, respiration, and sweating. The person is behaviorally ready to deal with fear, anxiety, and the possible need to act in response to the actual or perceived threat. It is important to note that hypervigilance is not an all-or-none phenomenon. Similar to dissociative reactions, there are degrees of hypervigilance as an expression of increased autonomic nervous system arousal. There are also varying levels of conscious awareness (LCA; Wilson et al., 2001) and self-monitoring of internal states of increased arousal and hypervigilant behaviors.

Hyperarousal and Self-Monitoring Difficulties

The problem of self-monitoring of PTSD states of hyperarousal is yet another manifestation of allostatic dysregulation. Persons suffering from PTSD often have a decreased capacity to accurately self-monitor ("read") their internal states of arousal, emotions, and thought patterns. In extreme cases, the failure to accurately monitor and process internal states can lead to the possibility of misinterpreting others' intentions, actions, and verbal expressions and subsequently result in defensive action that includes withdrawal, avoidance, and overt aggression (Wilson et al., 2001).

In extreme states of hypervigilance (as a symptom of PTSD rather than of other mental disorders), the person filters his or her perceptions of the environment through a finely meshed screen that is tightly woven together from

the trauma experience. Perception and information processing are then filtered through an ultra-high-grade sieve constructed from the individual trauma encounter, which sifts out irrelevant information and focuses attention on those actions of others or the environment with the highest potential for danger and threat. It must be recognized that as a component of PTSD, hypervigilance is a biologically conditioned (learned) response. Hyperarousal during the trauma was associated with a range of actions that ultimately resulted in survival, albeit with physical or psychological consequences. *Hyperarousal states during the trauma were reinforced by survival itself*. Thus PTSD hypervigilance is an automatic, psychobiologically determined response pattern designed to adapt to the perception, or existence of, threat to the well-being of the organism. It is for this reason that it is very difficult to extinguish a learned survival response. However, extreme hyperarousal may result in misperception of cues and lead to maladaptive responses, including those with potential legal consequences (e.g., self-defense resulting in a criminal assault; see Wilson & Moran, Chapter 21, this volume).

Abnormal Startle Response

The PTSD D5 hyperarousal criterion of exaggerated startle response is perhaps the purest example of a psychobiologically conditioned response. The startle response is an instinctive reaction to unexpected stimuli, such as loud noises, bright flashes of light, and aversive or classically conditioned odors. An exaggerated startle response is an amplified pattern of such reactions, usually with trauma-specific associations that are discernible from the person's history of involvement in the trauma. For example, former combat veterans often manifest exaggerated startle to loud explosion-like noises, the sounds of helicopters, aircraft engines, sandy wind storms, and whistle-like noises associated with mortar rounds or rockets. Further, although exaggerated startle reactions are expectable in patients with PTSD, they may also manifest in unusual, idiosyncratic ways, as in the case of a former torture victim who jumps anxiously at a routine medical examination (Juhler, 1993, in press) or of sexually abused children who become agitated at bedtime. In some situations of torture, medical personnel assist in overseeing torture to ensure its effectiveness and to minimize observable, external scars produced by the process. Exaggerated startle reactions are both generalized and idiosyncratic in nature (Wilson & Drozdek, in press).

In summary, the psychobiological criteria for PTSD presented in the DSM-IV-TR (American Psychiatric Association, 2000) contain four subcategories of hyperarousal phenomena that are important to understand in clinical assessments because the symptoms were not present before the occurrence of the trauma. We can summarize these four psychobiological clusters as: (1) sleep cycle disturbance; (2) cognitive processing deficits (e.g., concentration, attention, memory, information processing, executive functions); (3) perceptual and sensory sensitivity (e.g., startle responses, hypervigilance); and (4)

hyperarousal phenomena (hypervigilance, startle response, risk-taking behaviors, sensation seeking, agitation, feeling keyed up, or edgy, etc.).

ACUTE STRESS DISORDER

In DSM-IV (American Psychiatric Association, 1994), acute stress disorder (ASD) was added as a complementary diagnostic category to PTSD. ASD is similar to PTSD except that its onset and offset as a stress-response syndrome occur within 1 month of the traumatic event. ASD is a form of short-term stress-response syndrome and as such reflects a shorter cycle of the disorder

ASD differs from PTSD as a diagnostic entity in several ways relevant to clinical assessments. First, its duration is shorter and does not have the PTSD subtype specifiers of delayed onset, chronic or acute (i.e., symptoms less than 3 months). Second, to be diagnosed with ASD, a person needs to manifest only *one* symptom from each cluster of the core PTSD triad: (1) reexperiencing, (2) avoidance and numbing, or (3) hyperarousal. Third, ASD, unlike PTSD, has a separate diagnostic category for dissociative symptoms. In the DSM-IV-TR (American Psychiatric Association, 2000), the 'B' criteria for ASD states "either while experiencing or after experiencing the distressing event, the individual has three (or more) of the following dissociative symptoms," which include: "(1) a subjective sense of numbing, detachment, or absence of emotional responsiveness; (2) a reduction in awareness of his or her surroundings (e.g., 'being in a daze'); (3) derealization; (4) depersonalization; and (5) dissociative amnesia (i.e., inability to recall an important aspect of the trauma)" (p. 471). Thus, *only* for the dissociative cluster of symptoms for ASD must the person manifest three of the five forms of dissociation listed to have a positive diagnosis.

ASD AND DISSOCIATIVE PROCESSES

Several aspects of the criteria for ASD require further examination. First, the B set of diagnostic criteria for *dissociative symptoms* uses the words "distressing event" rather than "traumatic event." It also states that the dissociative symptoms may occur during the event (i.e., "while experiencing") or after its termination ("after experiencing"). The proximity of the dissociative reaction to the traumatic or distressing event reflects what are known as *peritraumatic dissociation* and posttraumatic dissociation (Wilson et al., 2001).

Moreover, Marmar, Weiss, and Metzler (1997) have provided conceptual and clinical clarity to the understanding of peritraumatic dissociation:

> One fundamental aspect of the dissociative response to trauma concerns immediate dissociation at the time the traumatic event is unfolding . . . dissociation at the time of trauma may take the form of altered time sense, with time being experi-

enced as slowing down or rapidly accelerated; profound feelings of unreality that the event is occurring, or that the individual is the victim of the event; experiences of depersonalization; out-of-body experiences; altered pain perception; altered body image or feelings or disconnection from one's body; tunnel vision; and other experiences reflecting immediate dissociative responses to trauma. (p. 44)

PTSD VERSUS ASD: DIAGNOSTIC INCONSISTENCIES IN DISSOCIATIVE CRITERIA

The distinction between pre- and posttraumatic forms of dissociation in response to trauma or distressing events is quite useful, as it helps in assessing how the person coped with the distressing situation. *Peritraumatic dissociation reflects the individual's need to control the degree of threat experienced at the time of the event by using dissociation to cope with perceived danger*. In this regard, dissociation is an ego defense designed to alter awareness of potential harm (Chu & Bowman, 2000). Posttraumatic dissociation is a form of reexperiencing trauma, as noted in the PTSD B3 criteria, or as a response to current life situations involving the perception of threat. However, it is important to point out a contradiction within DSM-IV-TR (American Psychiatric Association, 2000) because *different sets* of criteria are being used to differentiate ASD from PTSD. PTSD does not require any dissociative symptoms following the traumatic event to establish a positive diagnosis. ASD requires three symptoms to make the diagnosis.

Space limitations do not permit a further analysis of the discrepancies in the diagnostic criteria for PTSD and ASD. It should be noted that persons suffering from either type of stress disorder (i.e., ASD or PTSD) can exhibit dissociative symptoms. As noted by Marmar et al. (1997), peritraumatic dissociative symptoms involve perceptual and cognitive alterations in time, space, person, location, emotions, body image, and reality in general.

ASD was included in DSM-IV (American Psychiatric Association, 1994) to be conceptually congruent with a diagnostic sense that there is a stress-response continuum that may be acute or chronic in nature. Without ASD as a diagnostic subtype of PTSD as an anxiety disorder, there is no correct way to diagnose disruptive, clinically significant impairments in functioning that have an onset and offset within a short period of time.

COMPLEX PTSD AND POSTTRAUMATIC DAMAGE TO THE SELF

A scientific awareness has been emerging that the diagnostic criteria for ASD and PTSD are "skeletal structures" of a much larger set of psychological impacts to organismic functioning in posttraumatic states. The core triad of PTSD symptoms define the primary modalities of the psychobiology of the

stress-response syndrome, which has been extraordinarily useful in understanding various parameters of the disorder. Our knowledge of PTSD prevalence, gender differences, chronicity, neurobiological changes in brain morphology, and assessment technologies has advanced the front line of scientific knowledge (Friedman, 2000; Wilson et al., 2001). Moreover, it has long been recognized that trauma's impact on persons is more than an aggregate of symptoms that manifest as posttraumatic changes in behavior patterns and coping adaptation.

Understanding and assessing PTSD in a holistic framework includes a sensitive understanding of how the inner self-processes of the person are affected by trauma. Traumatic events, especially those involving acts of interpersonal assault, violence, abuse or prolonged coercive internment under degrading conditions, attack the bases of the self and systems of personal meaning. The results of traumatic injury to the self and personhood are deleterious, diverse, and, in some cases, pathologically lethal. Our understanding of persons with dissociative identity disorders, war veterans, interned political prisoners, prisoners of war, rape victims, Holocaust survivors, and those who have suffered repeated, prolonged, and multiple forms of abuse have provided clinical descriptions of the various ways that trauma damages the inner self, the very "soul" of the person (Krystal, 1968; Lifton, 1967; Niederland, 1968; Wilson et al., 2001; Ulman & Brothers, 1988; Herman, 1999). Such terms as "soul death," "broken spirits," "soul bruising," "walking dead," "catanoid state," "empty shells," and "vacuum states" have been used to characterize traumatic damage to the self that extends beyond the mere presence of the core triad of PTSD symptoms (Krystal, 1988; Lifton, 1967; Simpson, 1993; Gabbard, 1992; Wilson, 2003a). The self-structure is a central organizing component of personality (Stern, 1985) and has both *structural* and *functional* dimensions that are critical to understanding responses to trauma. Extreme trauma attacks the individual's core self, resulting in structural damage to the organization of self. As a consequence, self-dissolution, dissociative processes, fragmentation in ego processes, and a loss of self-sameness in continuity in identity may be evident. Wilson (2002, 2003a, 2003b, in press) has identified 11 separate typologies of posttraumatic self-configurations that exist on a continuum from severe pathology (e.g., the inert self) to optimal health (e.g., the integrated self). This typology also includes related aspects of posttraumatic pathology and personality processes and illustrates the wide range of possible ways in which self-transformation occurs following trauma.

It is not possible to describe and assess how the self is damaged by trauma without understanding the inner world of traumatization (Kalsched, 1996). Descriptions of complex PTSD have abounded in many sources published on the severe effects of traumatization dating back to the early Greeks (Friedman, 2000). Judith Herman (1992) reviewed some of the major categories that made up her description of complex PTSD—including suicidality, self-mutilation, dissociation, substance abuse, depression, psychosomatic complaints, character and identity changes, and disruptions in intimacy,

sexuality, and patterns of interpersonal relationships. Herman (1992, 1999) notes quite correctly that "concepts of personality developed in *ordinary circumstances* are frequently applied to survivors without an understanding of the deformation of personality which occur under conditions of coercive control" (p. 93, emphasis added). Viewed in another way, we have no separate psychology of how trauma affects the self and personality processes in lifespan development. Traditional theories of personality have a useful but limited relevance to understanding assaults on the self-structure in situations of repeated, prolonged, or extreme abuse and trauma. For example, who would consider a death camp an ordinary life experience? Or surviving years of secretive childhood sexual or physical abuse? Or being a torture victim of a political regime? Or the perpetrator and recipient of trauma in prolonged combat exposure under conditions of guerrilla warfare? Or being a wrongfully convicted murderer living for years in prison on death row and later exonerated? Herman (1992) suggests that the misapplication of personality disorders is among the most common diagnostic errors for trauma survivors whose personality characteristics and self-structures have been altered and sometimes warped by extreme stress experiences. This diagnostic insufficiency is typically the case in clinical assessments in which an inadequate trauma history fails to establish the level of optimal functioning prior to the traumatic event.

IMPACT OF PTSD ON THE SELF, EGO PROCESSES, AND IDENTITY

How is the inner core of self-esteem and personal identity damaged by trauma? Trauma's impact on the self-structure, ego processes, and identity is a complex intrapsychic phenomenon of critical importance to a holistic–dynamic understanding of PTSD. Wilson et al. (2001) and Wilson (2002a, 2003a, in press) have reviewed the various conceptualizations of trauma's impact on the self and its functional properties. Based on the works of Freud (1916/1957), Lifton (1967, 1979, 1993), Krystal (1968, 1988), Kalsched (1996, 2003), Ulman and Brothers (1988), Niederland (1968), Putnam (1997), Erikson (1968) and others, it was possible to extract similarities and consistencies in clinical findings concerning posttraumatic damage to the self. In particular, Wilson (2002b, 2002c, 2003b) notes that six core dimensions of the self are affected by traumatic events: (1) coherency, (2) connection, (3) continuity, (4) energy, (5) autonomy, and (6) vitality. Each of these dimensions of the self can be adversely affected by trauma and can result in varying degrees of self-dissolution, disintegration, fissility, disunion, dissociation, fracturing, or annihilative effects. Moreover, it is historically interesting to note that Freud (1916/1957) believed that "*a person is brought so completely to a stop by a traumatic event which shatters the foundation of his life that he abandons all interest in the present and remains permanently absorbed in*

mental concentration upon the past" (p. 342, emphasis added). Clearly, Freud understood the potential transformative power of trauma, especially to ego processes, as a protection against harm (see *Beyond the Pleasure Principle* [Freud, 1920/1959] for a complete discussion). Similarly, Erik Erikson (1968) developed the concept of *identity diffusion* from his therapeutic work with World War II veterans and stated, "Most of our patients had neither been shell-shocked nor became malingerers, but through the exigencies of war lost a sense of personal sameness and historical continuity. . . . *I spoke of a loss of ego-identity"* (1968, p. 17, emphasis added).

In his pioneering studies of Hiroshima survivors of the first atomic bomb, Robert J. Lifton (1967, 1979, 1993) spoke of how the self-structure fragmented and dissolved for many survivors of the bombing in 1945. Lifton spoke of *psychic numbing*: a loss of capacity of the self to experience emotions and life experiences. He also noted that survivors showed distinct changes in their sense of continuity in time, space, and future orientation. Lifton (1967) observed, for example, that some bombing survivors believed they were "walking dead," living in penance in a Buddhist hell. He reported that they experienced a loss of continuity and future orientation, as well as a sense of fragmented personal and physical integrity. In his psychoformative theory of the self, Lifton (1976) suggested that self-alteration could be expressed as stasis, separation, and *self-discontinuity* in posttraumatic states. Nearly identical observations were made by William Niederland (1968) in his clinical studies of Holocaust survivors, in which impairments to personal identity and self-function were paramount issues in recovery. More recently, studies of survivors of the terrorist attacks on the World Trade Center in 2001 now show similar findings on self-changes (Cardenas, Williams, Wilson, Fanouraci, & Singh, 2003).

Many other areas of traumatic stress research support the undeniable deleterious impact of trauma for aspects of self-functioning. Space limitations restrict our analysis, but research on dissociative identity disorders (DIDs), childhood sexual abuse, torture victims, rape victims, refugees, prisoners of war, and battered women have all confirmed that traumatic events can cross-cut all dimensions of the self-structure and result in self-fragmentation, discontinuity, loss of drive, loss of autonomy and vigor, and loss of the will to thrive (Chu & Bowman, 2000).

POSTTRAUMATIC DAMAGE TO IDENTITY, EGO PROCESSES, AND THE SELF-STRUCTURE

Wilson and colleagues (2001) presented a detailed, tetrahedral model of PTSD with five symptom clusters that include the core injuries of the PTSD triad, as well as injuries to the self-structure, attachment, intimacy, and interpersonal relations that *were not present before the traumatic event*. In terms of PTSD

and the self-structure, they identified 13 symptoms that are manifestations of traumatic injury to the self-structure, ego processes, personal identity, and personality processes:

1. Narcissistic and other personality characteristics that reflect damage to the self-structure associated with trauma.
2. Demoralization, dispiritedness, dysphoria, and existential doubt as to life's meaning.
3. Loss of ego coherence and dissolution of the self-structure.
4. Loss of a sense of sameness and continuity to ego identity or capacity for ego stability.
5. Fragmentation of ego identity and identity disturbance (e.g., identity diffusion).
6. Shame, self-doubt, loss of self-esteem, guilt, and self-recrimination.
7. Fluctuating ego states; proneness to dissociation and lack of ego mastery.
8. Hopelessness, helplessness, and self-recrimination; masochistic and self-destructive tendencies.
9. Suicidality; patterns of self-destructiveness or self-mutilation.
10. Chronic feelings of uncertainty and vulnerability; levels of depression, helplessness, and hopelessness.
11. Existential personal or spiritual angst; dread, despair, and a sense of futility in living.
12. Loss of spirituality, essential vitality, willingness to thrive, religious/cosmic belief systems, and so forth.
13. Misanthropic beliefs, cynicism, and a view of the world as unsafe, dangerous, untrustworthy, and unpredictable.

Posttraumatic damage to the self-structure may be manifested in degrees of injury, impairment, or deficits anywhere on the continuum of fragmentation to integration *of the structure itself (i.e., coherence, connection, continuity, vitality, autonomy, energy*). In extreme cases of PTSD, the entire self-structure dissolves (Goodwin, 1993, 1999; Kalsched, 2003; Jung, 1953–2000). Extreme fragmentation of the self-structure caused by traumatic injuries may result in a loss of energy to thrive, a loss of autonomy and "free" self-regulation, a loss of self-continuity with the past, a loss of a meaningful sense of connection to others, and a loss of the capacity for intimacy. The person with severe PTSD may manifest a profound loss or altered sense of continuity in personal identity; the threads of self-sameness with the flow of one's past dissipate, leaving a sense of interrupted life sequence (Wilson, 1980, 1981, 1994a, 1994b, 1995). The inner core of the self may be experienced as empty or dead or as existing in an abyss of nothingness.

The 13 symptoms of PTSD identified by Wilson et al. (2001) are useful to clinical assessments because they employ the same criteria that were used for

the B, C, and D PTSD symptoms in the DSM-IV-TR (American Psychiatric Association, 2000); that is, these symptoms of traumatic injury to the fabric of the self-structure and its functional capacities *were not present before the trauma*. The symptoms are manifestations of allostatic psychobiological adaptations to traumatic stress impact on the *inner agency of the self*. Trauma produces changes in psychological functions that were not present in the same functional or structural manner as before the stressful event. *As reexperiencing, avoidance, and hyperarousal symptoms have a specific and traceable relationship to the precipitating trauma, so do the symptoms of altered self-functions*. Furthermore, these symptom manifestations are not only directly caused and/or correlated with trauma exposure, but there are also not more viable, logical, or meaningful explanations for their presence in the repertoire of posttraumatic adaptive behaviors of the person.

IMPACT OF PTSD ON ATTACHMENT, INTIMACY, AFFILIATIVE BEHAVIORS, AND INTERPERSONAL RELATIONS

How does trauma affect the nature and quality of interpersonal relationships? As noted in DSM-IV-TR (American Psychiatric Association, 2000) under the "associated descriptive features and mental disorders," other symptoms of complex PTSD are also important to clinical assessment and treatment protocols. The diagnostic manual lists such symptoms as interference in relationships, marital conflict, and poor job performance (p. 465). Although these are useful observations, it is possible to gain greater conceptual and diagnostic clarity by specifying the domain of PTSD symptoms that represent traumatic damage to attachment, intimacy, sexuality, and interpersonal relationships. Wilson et al. (2001) list 13 symptoms for this criterion that were not present before the trauma:

1. Alienation: social, emotional, personal, cultural, spiritual.
2. Mistrust, guardedness, secretive behaviors, non-self-disclosure, reticence toward social encounters.
3. Detachment, isolation, withdrawal, estrangement, and feelings of emptiness.
4. Anhedonia: loss of pleasure in living; loss of sensuality, sexuality, feeling, capacity for joy.
5. Object relations deficits; loss of capacity for healthy connectedness to others.
6. Self-destructive or self-defeating interpersonal relationships which are repetitive in nature.
7. Impulsiveness, sudden changes in residence, occupation, or intimate relationships.

8. Impaired sensuality, sexual drive, capacity for sexuality or loss of libidinal energy in general.
9. Inability to relax; discontent with self-comfort activities and an inability to receive nurturing, affection, or physical touching from others.
10. Unstable and intense interpersonal relationships whose origin is in trauma experiences.
11. Problems with establishing or maintaining boundaries in relationships based on trauma experiences.
12. Anxiety over abandonment or loss of loved ones, which is either conscious or unconscious in nature and based in traumatic experiences.
13. Repetitive self-defeating interpersonal relationships which reflect unmetabolized patterns of attachment behavior from abusive developmental experiences.

These symptom clusters are manifestations of trauma's adverse impact on *interpersonal* and *intrapersonal* functioning. Traumatic impact on the domain of affiliative and attachment behaviors creates problems with: (1) healthy boundary maintenance, (2) trust of others, (3) repetitive self-defeating relationships, (4) impulsiveness in areas of sexuality, friendships, and economic consumption, (5) personal, social, and cultural alienation, (6) geographical isolation from others, (7) self-care, (8) fears of abandonment, (9) intense, unstable relationships, (10) secretiveness, guardedness, or an unwillingness to self-disclose, and (11) impaired capacity for enjoyment of work, play, exercise, sensual relations, and sex drive.

BEYOND COMPLEX PTSD: ORGANISMIC IMPACTS OF TRAUMATIC STRESS

The complexity of PTSD and personality functioning allows a summary of the organismic nature of these phenomena as prolonged stress-response syndromes that are manifestations of allostatic adaptive processes (Wilson et al., 2001). As such, they are direct manifestations of changes in the primary psychological substrata (see Figure 1.1).

Holistic Stress Response Syndromes

PTSD and self-impairments are holistic stress-response syndromes and function synergistically in their psychodynamics. Trauma impacts on the organism are multidimensional and affect psychological systems of functioning (e.g., memory, learning, information processing, self-esteem, personality development, interpersonal relations, motivational striving, system of meaning and belief, etc.).

Synergistic Dynamics

A holistic, dynamic understanding of allostatic processes indicates that there are five distinct but interrelated sets of symptom clusters that make up ASD, PTSD, and complex PTSD: (1) reliving; (2) avoidance and numbing; (3) increased physiological reactivity (hyperarousal); (4) changes in self-structure, personal identity, and ego processes; (5) changes in affiliation, attachment, intimacy, and interpersonal relationships. *For each of the five clusters of symptoms, there are a discernible set of symptom indicators of posttraumatic changes in baseline organismic functioning that were not present before the trauma*. These symptom manifestations are synergistic. Changes in one cluster produce changes in the others, which can be described as a tumbling, cascade set of effects. The five clusters have *reciprocal interaction effects* with one another, creating cycles or episodes of symptom manifestation until a steady state of allostatic stabilization is achieved.

New Organismic Baseline in PTSD

The five clusters of PTSD-related symptoms reflect organismic shifts in adaptive functioning, a new baseline (i.e., set point) of functioning after trauma. The new baseline of functioning applies to each set of symptom clusters, as well as to the integrated posttraumatic functioning of the organism as a whole (Wilson et al., 2001).

Periodicity in PTSD Symptom Clusters

The variability in manifestations of PTSD symptom clusters consists of allostatic alterations in symptom functions. It is possible for some symptom patterns (e.g., avoidance) to persevere longer than others. Symptom patterns may be episodically activated through conditioned learning and may evoke psychobiological response patterns. Other symptom patterns (e.g., self-dissolution) are manifestations of a relatively permanent injury to the organism. PTSD has permutations in the severity, duration, intensity, and frequency of symptom manifestation in all five dimensions of the syndrome. Thus there is variability in periodicity in PTSD symptom manifestation.

Severe and Prolonged Stress-Related Damage to the Core Psychological Processes

The most severe damage in PTSD occurs to the two core structures of the syndrome: (1) neurobiological responses of the adrenergic system and (2) structural components of the self. Damage to the genetically driven and biologically hardwired, instinctive components of the stress-response system reflects allostatic loads described by McEwen (1998) as: (1) repeated hits (i.e., recurrent or prolonged exposure to stressors); (2) lack of capacity for adaptation;

(3) prolonged stress response; and (4) inadequate response or system failure, that is, the capacity of the neurohormonal engineering mechanisms to begin to fail. In severe PTSD, prolonged activity of the neurobiological components becomes deleterious to the organism, as repeated demands on the system to function effectively may not allow sufficient time and resources for reparative maintenance of the system itself. Empirical research demonstrates that prolonged stress responses, without relief and allostatic restabilization, are associated with changes in the cardiovascular, endocrine, and other systems of the body (McEwen, 1998; Bremner, 2002).

In an analogous way, damage or prolonged stress to the core dimensions of the self-structure (i.e., coherence, continuity, connection, vitality, energy, autonomy) may result in severe personality damage in terms of: (1) self-esteem; (2) personal identity and sense of self-identity; (3) loss of striving in life; (4) suicidality and self-destructiveness; (5) fragmentation in ego processes and self-components (e.g., alternate personalities in DID); (6) capacity for intimate relationships; (7) changes in systems of meaning, beliefs, values, and faith.

Etiology, Clinical Assessment, and Lifespan Development

An organismic approach to PTSD enables assessment of a broader, more encompassing set of psychological and psychosocial functions associated with the syndrome. These include, but are not limited to: (1) understanding of the etiology of the disorder in five subsystems and functioning affected by trauma; (2) understanding of the changes in pretraumatic baseline functioning to a new, allostatic set point in organismic functioning; (3) assessment of profile configurations in terms of frequency, periodicity, severity, intensity, and duration of symptoms within and among the five interrelated clusters of symptoms; (4) knowledge of how core, inner dimensions of the self are altered in ways that are associated with posttraumatic self-typologies that fall along a continuum of fragmentation (i.e., loss of structural coherence) to integration and transformation (i.e., unity transcendence, resilience); and (5) understanding of how trauma affects biosocial functioning in terms of epigenetic development and personality functioning in the life cycle.

Diagnoses: PTSD and Axis I and Axis II Mental Disorders

The clinician assessing PTSD, especially for purposes of formulating a treatment plan, or in forensic evaluations (see Chapter 21, this volume) must attempt to obtain as complete a picture of the patient's functioning as possible. To do this requires taking a comprehensive trauma history and determining whether or not there is a prior history of abuse, victimization, or a preexisting Axis I or Axis II mental disorder. A five-step decision-making process to gain information as to the pretrauma level of functioning and psychobiological stress response set point of adaptation is recommended.

CONSIDERATIONS IN THE DIFFERENTIAL DIAGNOSIS OF PTSD

1. *Premorbidity*. Is there any psychiatry history prior to the traumatic event that would suggest or document an Axis I or Axis II clinical disorder?

2. *Substance abuse*. Is there any history of drug or alcohol abuse prior to the traumatic event?

3. *Changes in personality and behavior*. Are there identifiable and independently verifiable changes in personality and behavior that were not present before the trauma but are posttraumatically manifest as personality processes? If so, are the changes discernible in the self-structure and ego processes of the individual?

4. *Transient behavioral and personality changes*. Are there transient changes in personality and behavior that reflect allostatic dysregulations in behavior as attempts at coping with trauma in proximity to the event (e.g., one month to one year posttrauma)?

5. *Interaction of premorbidity, trauma, and PTSD*. If a preexisting Axis I or Axis II disorder exists, to what extent does it contribute to the current symptoms, personality processes, and forms of coping? It is important to determine in what ways, if any, a preexistent psychiatric disorder or history influences the clinical presentation of PTSD and the processing of the trauma experience.

6. *Multiple diagnoses*. PTSD can coexist with any Axis I or Axis II clinical disorder. There are multiple combinations of comorbid diagnoses possible with PTSD. These combinations may present complex and difficult assessment and treatment issues.

CONCLUSION

In conclusion, the analysis of PTSD as a prolonged stress response syndrome has enabled us to gain a panoramic, wide-lens view of the disorder. In the aftermath of trauma, the psychobiological nature of the human stress response involves the characteristic development of reactions, symptoms, and integrated syndromes of adaptive functioning. Traumatic events activate primary biological and psychological processes of the organism that control the mechanisms of adaptive responses. PTSD is a synergistic syndrome, and the symptom clusters that develop have reciprocal, interactive influences on each other. As an organismic stress response, PTSD involves five clusters of symptoms that develop from the two primary psychobiological substrates of the underlying neurohormonal engineering systems of the body. The five PTSD clusters are epiphenomenal manifestations of the psychobiological substrates and include reexperiencing phenomena, avoidance and numbing, hyperarousal states, impaired self-function, and effects on interpersonal relations.

These integrated symptom clusters are unique to PTSD as a psychiatric disorder because they: (1) were not present before the traumatic event and (2) are specific manifestations of the normal, adaptive stress response pattern in a prolonged and potentially impairing way to psychological functioning. Once ASD or PTSD develops, various forms of behavioral adaptations may become evident in the person's repertoire of coping and adaptive capacities. These behavioral adaptations configure in dysregulated affects, personality alterations, altered interpersonal relations, and psychosocial functioning until recovery, restabilization, and healing occur.

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