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Trauma, Adaptation, and Resilience: A Cross-Cultural and Evolutionary Perspective

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Trauma and its consequences are a focus of intense interest. Posttraumatic stress disorder (PTSD), although not a new diagnosis – "war psychosis" and "shell shock" were long recognized – has recently been applied to a very wide range of negative experiences (Jones et al., 2003; Jones & Wessely, 2004; McHugh, 1999; A. Young, 1995). The definition has broadened beyond extremely severe and abnormal circumstances, such as war, rape, or devastating natural disasters, to encompass stresses in normal life – ongoing aspects of work and relationships and childhood emotional stress in the range that might once have been considered normal. Although physical, sexual, and severe emotional abuse – not to mention torture and concentration-camp experiences – surely deserve this label, the word *trauma* is no longer restricted to such extremes.

In the popular imagination and for some mental health professionals, it means far more – including residence in the city where a terrorist attack has occurred or viewing traumas on the television news – and we often hear recommendations for immediate psychological intervention. In fact, extensive evidence shows that resilience and/or independent recovery are by far the most common responses to potentially traumatic experiences (PTEs) in both adults (Bonanno, 2004, 2005) and children (Masten, 2001). Furthermore, research and clinical experience question the value of and point to the possible harm due to widely urged mental health interventions following PTEs (Rose, Bisson, Churchill, & Wessely, 2002; Rose, Bisson, & Wessely, 2003; Wessely, 2005; Wessely & Deahl, 2003). Although psychiatry has tried to restrict PTSD to established criteria (American Psychiatric Association [APA], 1994), some mental health professionals and many media pundits have abused the label, with potentially negative consequences for public mental health.

This chapter sets psychological trauma in the broad context of human evolution and culture. First, I consider stress in the original human environment, that of hunting and gathering, with an emphasis on the !Kung San

or Bushmen of Botswana, who are in some ways representative. Second, I review basic stress physiology and consider the distinction (originally made by Hans Selye) between *dis*tress and *eu*stress, and how the distinction aids our understanding of resilience. Third, I raise some questions about the concepts of psychological trauma and PTSD, about inappropriate uses of these concepts, and about interventions often urged or performed after PTEs. I conclude with some inferences about the role of stress in human experience.

HUNTER-GATHERER ADAPTATIONS

In the mid-twentieth century anthropologists became interested in studying living hunter-gatherers to model the circumstances in which our species evolved (Lee & DeVore, 1968a) and attempted to document their subsistence ecology and social organization. Classical studies had included those of Australian aborigines, Eskimo, Amazonian hunter-gatherers (HGs), and many others throughout the world (Lee & DeVore, 1968b). More systematic, multidisciplinary, quantitative studies were subsequently done on the Hadza of Tanzania (Hawkes, O'Connell, & Jones, 1991; Hurtado, Hawkes, Hill, & Kaplan, 1985), the Ache of Paraguay (Hill & Hurtado, 1999), the Agta of the Philippines (Griffin & Estioko-Griffin, 1985), the Efe Pygmies of Zaire (Bailey, 1991; Peacock, 1991), and the Bushmen of Southern Africa (Lee, 1979a; Silberbauer, 1981), among others (Lee & Daly, 1999).

These groups represent some aspects of our environment of evolutionary adaptedness (EEA), although given the variation among them and among past populations, the phrase should be pluralized to EEAs. These are, or were, the contexts for which natural selection prepared us, and from which we have departed only in the past 10,000 years, a short time in evolutionary terms. No one claims that the whole range of EEAs is observable among recent HGs, who have occupied only some of the wide range of ecological situations available to our ancestors. However, we also have extensive archeological, paleodemographic, and paleopathological evidence (Keenleyside, 1998; Reinhard, Fink, & Skiles, 2003; Tague, 1994) that – with the studies of recent HGs – leads to reasonable models of life during human evolution (Hewlett, 1991; Kelly, 1995; Winterhalder & Smith, 1981).

Certain generalizations are possible: (1) Groups were usually small, ranging in size from 15 to 40 people related through blood or marriage, but could be larger in ecologically rich settings; (2) they were usually nomadic, moving with changing subsistence opportunities, and flexible in composition, size, and adaptive strategies, although they could be sedentary in richer settings; (3) daily life involved physical challenge, vigorous exercise, and occasional hunger; (4) disease, mainly infectious rather than chronic, produced high rates of mortality especially in infancy and early childhood,

with consequent frequent experience of loss; (5) virtually all activities were done in a highly social context with people one knew well; (6) privacy was limited, but creative expression in the arts was possible; and (7) conflicts and problems were dealt with through extensive group discussions, but could result in separation or violence.

This applies to most of human history, so it is often said that we are HGs in business suits and skyscrapers. The industrial revolution, in evolutionary perspective, happened only a moment ago, and several of these generalizations – notably for our purposes the physical challenges of life, the role of hunger and infectious disease, and the high infant and child mortality – apply to all premodern societies. Direct fossil and archeological evidence demonstrates rates of injury consistent with substantial violence and/or accidental trauma (Keeley, 1996; LeBlanc & Register, 2003), periodic food shortages (Gaulin & Konner, 1977; Whiting, 1958), and high levels of premature mortality in premodern populations generally (Hammel & Howell, 1987). Nutritional stress and infectious disease may have increased after the transition to agriculture (Armelagos, Goodman, & Jacobs, 1991; Cohen & Armelagos, 1984), but HG life was physically and psychologically stressful. As for nonhuman animals, and by inference the prehuman phases of our evolution, premature mortality and the stresses implied by it were if anything greater than in any human populations (P. H. Harvey, 1990; Hill et al., 2001).

Baby and child care were also distinctive in HG societies (Konner, 2005), despite variations, including (1) frequent breast feeding (up to four times per hour); (2) late weaning (at least 2 and up to 4 years); (3) close mother–infant contact, including skin-to-skin carrying and adjacent sleeping until weaning; (4) prompt response to infant crying and indulgent response to other infant and child demands; (5) maternal primacy in attachment; (6) more father involvement than in most societies; (7) a gradual transition to a multi-aged play group of mixed sex; (8) usually less assignment of responsibility in the sense of chores or schooling in middle childhood, with learning through observation and play; (9) liberal premarital sexual mores with sex play in middle childhood and adolescent sexuality; and (10) late menarche, limiting childbearing until the late teens or later. These generalizations have withstood the test of sophisticated new research in at least five HG societies (Konner, 2005). Because early experience plays a role in resilience, this pattern may buffer people against lifelong stress.

However, the great majority of HG children experienced loss and grief through the death of siblings, parents, or others, as well as their own life-threatening illnesses. Thus HG childhood was far from idyllic, but most frustration and loss did not come from parentally imposed stresses. Still, physical punishment and ridicule were used by parents among the !Kung (Shostak, 1981), children were required to forage for themselves among

the Hadza (Blurton Jones, 1993), and the experience of loss was virtually universal.

The !Kung San (Bushmen) of Botswana, among whom I did field research for two years, are typical in many ways. Physiologically and psychologically they resemble human beings anywhere, but in subsistence ecology they – along with other HG groups – represent that of our ancestors (Lee, 1979a; Lee & DeVore, 1976; Marshall, 1976a). The environment is semiarid, and the soil is sandy with relatively sparse vegetation, but it provides ample plant food for people's needs. Like most HGs, their population density averaged less than one person per square mile, but was concentrated in villages with high social contact.

Women gathered plants, providing 70% of the diet by weight, retrieved water; collected firewood; and did 90% of the infant and child care. Nevertheless, they enjoyed largely equal relationships with men, had strong female friendships, and sometimes took lovers (Shostak, 1981, 2000). They gathered two to three days per week in highly social small groups. The staff of life was the fruit and nut of the mongongo tree, and women walked an average of 6 miles each way to the groves, carrying one or two infants or small children both ways plus 30 pounds of nuts on the way back. Men hunted at about the same frequency, alone or in groups, but hunts were necessarily quiet. Eland, oryx, kudu, wildebeest, duiker, steenbok, and giraffe were among their prey. Game such as oryx and warthog, which stand and resist, were hunted with dogs and spears by small groups of men, the other animals by one or two men with deadly poisoned arrows. Some carried scars of hand combat with leopards; others were killed by lions or hyenas.

Women had if anything a greater ordeal of physical courage: At least in the higher parities, childbirth was ideally supposed to be managed by the parturient woman alone (Konner & Shostak, 1987). The loss of at least one child, usually in infancy and early childhood, was common. Because of intensive breast feeding, average birth spacing was 4 years. Infants were in skin-to-skin contact with someone at least 90% of waking hours in the first months, declining to 25% at 18 months. Mother and infant slept on the same skin mat. Toddlers made a gradual transition to a multi-aged, mixed-sex play group. Children had little responsibility but tended to forage for themselves casually, and younger children were often cared for by older ones, especially girls. Information and skills passed mainly from older to younger children, not mainly from adults to children.

Play groups were frequently out of sight of adults, and sexual curiosity took its course. Adults did not approve of sexual play but made little effort to discourage it, viewing it as inevitable and even healthy. Most children also could observe sexual intercourse at some time during childhood. Overall, the effect seems to be that sex is less taboo, less frightening, and less unknown. However, the transition from the playful sex of childhood

to the real sex of adulthood could be difficult, especially for girls, half of whom were married before their first menstruation (about age 16.5), typically to men about 10 years older. Although in principle the husband's advances would be delayed until menarche, the transition was stressful for many (Konner & Shostak, 1986; Shostak, 1981).

Overall patterns of fertility and mortality are well established (Howell, 1979). Prospective study of the age at menarche (marked by a dramatic ritual) gives a mean of 16.6 years, with the majority passing this milestone between 16 and 18 (Howell, 1979, p. 178). The average age at first birth was 19 years, almost all between 17 and 22 (p. 128). Completed fertility was 4.7 live births per woman, with the last birth in the mid-30s. Mortality patterns were typical of most human populations before the nineteenth century. Half of all children died before age 15, 20% in the first year. Life expectancy at birth was 32 years, at age 15, 55 (40 more years). Only 20% of neonates reached age 60, but some lived well into their 80s. Most deaths were due to infectious diseases such as gastrointestinal infection, pneumonia, tuberculosis, malaria, and wound infections, but some were due to accidental or violent injury, and the parasite burden was high. The central ritual was a healing dance in which trained men danced until in a trance state and attempted to heal through laying on of hands with a specific form of trembling and shrieking, a formidable display of individual and communal support (Lee, 1982; Marshall, 1981).

A village camp was a small circle of huts, each holding a family in a hemisphere of grass just large enough to lie down in. The camp included perhaps 30 people, but group structure was flexible, varying between 10 and 40, and moved with seasonal vagaries of food and water availability. People changed groups at times; conflicts were often resolved by group fission. The fragments might coalesce again months later or form the nuclei of new bands (Lee, 1979; Marshall, 1976b).

War was unknown in recent generations, although ambushes and deadly intervillage raids occurred in the past (Wilhelm, 1953). Conflicts were often resolved by the sharing of food and other goods and by talking, sometimes half or all the night, sometimes for weeks on end. Few social or economic distinctions could be maintained; the ethic of sharing strongly pressured a person to part with any visible wealth (Wiessner, 1982, 1996). Stinginess led to social ostracism, intolerable where survival requires mutual aid.

Mental illnesses both major and minor occurred. Homicide exceeded levels in American cities (Lee, 1979), despite the application of the phrase "the harmless people" to this group. Other undesirable behaviors, such as selfishness, deceit, adolescent rebellion, adultery, desertion, and child abuse also occurred, but for methodological reasons it is impossible to compare their rates to those in industrial societies. The lack of privacy provides stresses just as crowding and high levels of contact with strangers

may be stressful for us. Morbidity, mortality, and the uncertainties of the daily food quest took their psychological toll.

In summary, during the 98% of human history that took place in our environments of evolutionary adaptedness – the environments in and for which our genomes evolved – we survived periodic hunger; extreme physical exertion; natural disasters such as storms, fire, earthquakes, tidal waves, and volcanic eruptions; attacks by lions, leopards, hyenas, wolves, wild dogs, and many other predators; defensive attacks by large prey we were hunting; attacks by other humans; a heavy burden of infectious and parasitic illness; and frequent loss of loved ones. Many of these stresses persisted through almost the whole of the 10 millennia since the invention of agriculture. It is possible that our common generalized anxiety disorders (GAD) are the evolutionary legacy of a world in which mild recurring fear was adaptive (Nesse & Lloyd, 1992). Yet we not only survived; in some respects we thrived.

STRESS PHYSIOLOGY: *DIS*TRESS, *EU*STRESS, AND RESILIENCE

Because our genomes were formed in those conditions, we must be programmed to adapt to stress. Indeed, for hundreds of millions of years, stress was ubiquitous for all species ancestral to us; stress is of the essence of evolution by natural selection and close to the essence of life itself (Sapolsky, Romero, & Munck, 2000). It has been said of stress responses that "[t]hese changes are normally adaptive and improve the chances of the individual for survival," and that the behavioral component of the response includes many positive as well as negative features, such as "increased arousal, alertness, and vigilance, improved cognition, and focused attention, as well as euphoria or dysphoria" (Chrousos, 1998, p. 312). Some men speak of their combat experiences in strangely positive terms, as the time in their past when they felt most alive, or even as the best time (Terkel, 1984). Adventurous people say similar things about experiences that cause fear and stress in themselves and others, and seek such experiences (McCormick, 2001). Any model of stress effects must take into account such positive consequences, as well as the ubiquity of stress.

In mammals, a wide variety of stresses both physical and psychological results in a predictable suite of responses (Figure 15.1), sometimes called the general adaptation syndrome (GAS) (Sapolsky, 1992a; Selye, 1936, 1976, 1936/1998). Part of this syndrome is sympathetic nervous system (SNS) activation, also known as the "fight or flight" response. Essential aspects of this part of the response were established by the 1920s (Cannon, 1915/1963, 1927). The SNS initiates increases in cardiac and respiratory rates; mobilization of blood glucose; arterial dilation in heart, lung, and voluntary muscle; sweating; pupillary dilation; bladder emptying; and sensory heightening and draws blood flow and energy away from digestion

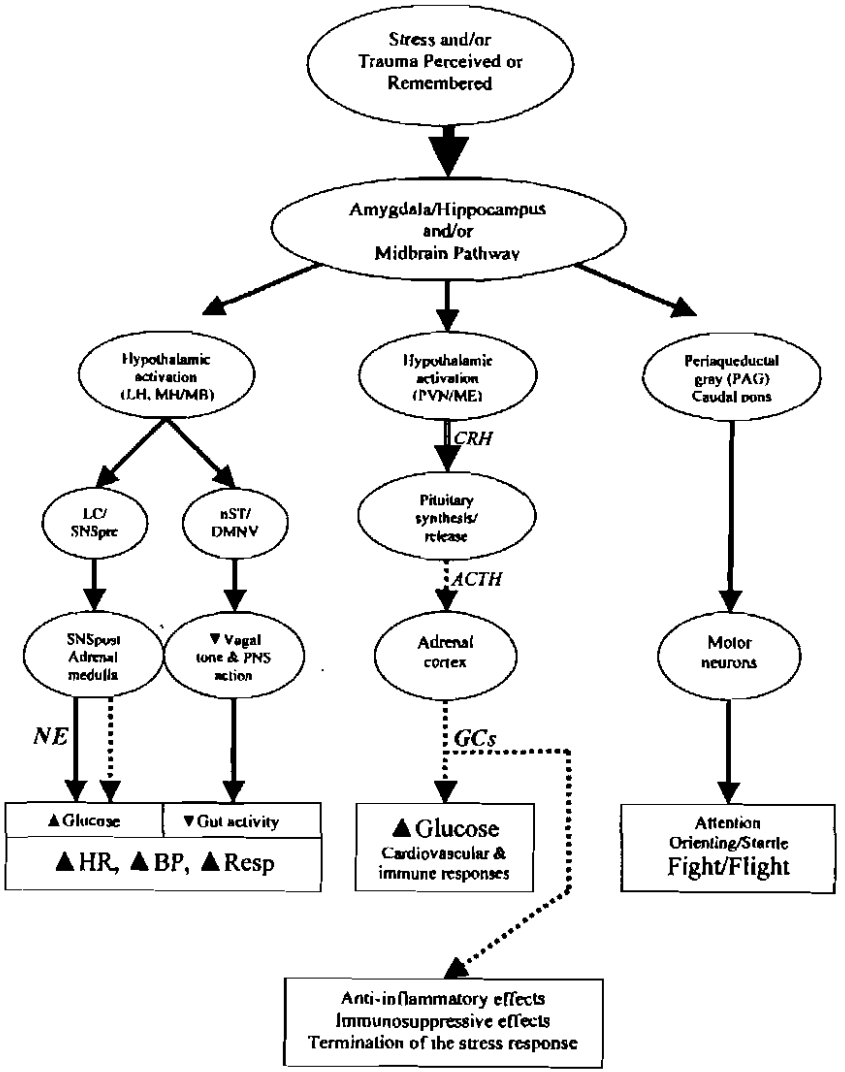

FIGURE 15.1. Simplified model of the stress response. Solid arrows, neural connections; dotted arrows, hormones in general circulation; double arrow, pituitary portal vessels; LH, lateral hypothalamus; MH, medial hypothalamus; MB, mammilary bodies; PVN, periventricular nucleus; ME, median eminence; PAG, periaqueductal gray; LC, locus ceruleus; SNSpre, preganglionic sympathetic nervous system; SNSpost, postganglionic SNS; nST, nucleus of the solitary tract; DMNV, dorsal motor nucleus of the vagus; PNS, parasympathetic nervous system; HR, heart rate; BP, blood pressure; Resp, respiration; CRH, corticotrophin-releasing hormone; ACTH, adrenocorticotropic hormone; GCs, glucocorticoids; NE, norepinephrine; Epi, epinephrine (adrenaline).

and reproduction. Its activation is twofold: faster, mediated by noradrenergic (norepinephrine-secreting, NE) neurons, and slower, mediated by secretion of adrenaline (epinephrine, E) by the adrenal medulla, or core of the adrenal gland. Although E acts as a hormone, this is phylogenetically and embryologically neural tissue similar to NE neurons, but E, which the adrenal medulla releases into the bloodstream, is a modified form of NE that is hundreds of times more potent. NE neurons within the brain are also activated.

Another component of the stress response, added to Cannon's model by Hans Selye (Selye, 1936), is activation of the hypothalamic–pituitary–adrenal (HPA) axis. Perception of stress by the limbic system, especially the amygdala and hippocampus, activates hypothalamic neurons that secrete corticotropin-releasing factor or hormone (CRF/CRH) into the anterior pituitary, where it stimulates the synthesis and secretion of corticotropin (adrenocorticotropic hormone, ACTH). At the adrenal cortex (the outer adrenal gland), ACTH stimulates the synthesis and release of cortisol, a glucocorticoid (GC) and principal stress hormone. Like E, GCs mobilize blood glucose for fight, flight, or other responses to stress and have both positive and negative effects on cognitive function (K. Erickson, Drevets, & Schulkin, 2003). GCs, over time, also can damage hippocampal and other neurons (Sapolsky, 1992b). But the discovery in the late 1940s that GCs have healing properties changed views about the GAS, suggesting the HPA axis has a role not just in mobilizing the organism in acute stress, but also in modulating and terminating that response.

The two components of the stress response are synergistic. During vertebrate evolution the adrenal medulla and adrenal cortex tissues changed their anatomical positions (Norris, 1997). In lower vertebrates they are adjacent, but mammalian evolution brought them into increasing juxtaposition until the medulla was surrounded by the cortex (hence, their names). The functional explanation is that the cortex supplies GCs to the medulla, where they are cofactors for the enzyme (phenylethanolamine *N*-methyl transferase) that converts NE to the far more potent E, greatly increasing the efficiency of the stress response. Brain NE neurons are also probably involved in directing the hypothalamus to release CRF.

We can now understand what is obvious to all who study natural history or evolution: Life itself is stress and coping, and because of the competition entailed in natural (including sexual) selection, individuals are continually providing stresses for each other even beyond other environmental stresses (Konner, 2002; Sapolsky, 2001; Sapolsky et al., 2000). Success in evolution requires superior coping with stress. In an important sense, every change in the stimulus envelope is a kind of stress, or at least a challenge, to which the organism must respond both physiologically and psychologically according to genetic programs modulated by individual experience.

Attention is the minimal change needed for an organism to respond to changing stimuli; the next step is arousal, then frustration, fear, or pain, depending on the nature and strength of the stimulus (Davis & Whalen, 2001; Ursin & Kadda, 1960; Zald, 2003), a process that is largely subcortical (Liddell et al., 2005; Ohman, 2005). This continuum of arousal and stress demands a corresponding continuum of response and action – adaptation or coping. Because of the internal discomfort, however mild, caused by many stimulus changes, successful coping is rewarding and sometimes exhilarating. Human infants as young as 3 or 4 months of age show wary attention to a moderately unfamiliar stimulus and smile when they have recognized or assimilated it (Super, Kagan, Morrison, Haith, & Weiffenbach, 1972; Zelazo & Komer, 1971). This is how we learn, grow psychologically, and liberate ourselves a little from the grip of the genes. It is also how evolution liberated higher organisms from the simple, mechanical, genetically dominated behavior of lower ones. Some helping professionals seem to think that the ideal condition for an organism is the absence of stress. This notion runs counter to all we know about life under natural conditions and violates the logic of our own subjective experience.

This is why Selye named the stress response the *general adaptation syndrome*: It is at the heart of adaptation itself. He also made a vital, often overlooked distinction: Some stress is negative and can impair future function, whereas other stress is positive, producing effective coping and enhancing the organism's long-term function. He called these "*dis*tress" and "*eu*stress," respectively (Selye, 1975). Unfortunately for simplicity, this is not a categorical distinction, and it is not always clear where to draw the line.

What *is* clear is that humans, to one degree or another, are resilient, even in the face of severe stresses. We have already considered the environments of human evolutionary adaptedness, where both eustress and distress are ubiquitous; so is resilience. Some people seek severe stresses, as in extreme sports and dangerous occupations (Haynes, Miles, & Clements, 2000), and people can be arrayed on a continuum of sensation seeking that has cross-cultural validity (Neria, Solomon, Ginzburg, & Dekel, 2000; Wang et al., 2000). Sensation seekers differ physiologically from others (Zuckerman, 1984, 1990; Zuckerman, Buchsbaum, & Murphy, 1980); they are not immune to trauma, but they are courageous and resilient. Several studies show that the most resilient athletes are also the best (Holt & Dunn, 2004; Martin-Krumm, Sarrazin, Peterson, & Famose, 2003; Mummery, Schofield, & Perry, 2004; Schinke & Jerome, 2002). Decorated Israeli war veterans score higher than other war veterans on sensation seeking and have low levels of PTSD symptomatology (Neria et al., 2000).

More relevant to the average person are life history studies of ordinary individuals. A longitudinal, prospective study of 94 men who were in college in the early 1940s followed the subjects for half a century. After 35 years of follow-up (Vaillant, 1977), many of the men were happy and successful by self-report as well as external criteria, whereas others were unhappy or failures. Extensive data from the men's childhoods, with follow-up from college up to advanced age, supported several conclusions.

First, a stable, loving early family life appears to confer advantage; men with bleak childhoods tended to remain unhappy despite externally defined success. Some seemed well adapted but could not form intimate relationships; one, aware of his impairment, stated that he could do nothing about it. This observation is consistent with other longitudinal studies, with growing clinical evidence (Heim, Plotsky, & Nemeroff, 2004), and with voluminous experimental data from animal models showing that early positive nurturance enhances lifelong resilience both psychologically and physiologically (Bennett et al., 2002; Champagne, Francis, Mar, & Meaney, 2003; Francis, Szegda, Campbell, Martin, & Insel, 2003; Sanchez, Ladd, & Plotsky, 2001; Sanchez et al., 2005; Suomi, 2002).

A second conclusion was that stress is not necessarily unhealthy or bad. One third of the men in this study spent at least 10 days in continuous combat in World War II. All 94 subjects suffered major personal grief, setbacks, disappointments, and losses during adulthood. None of these life events, per se, predicted poor adjustment. About a famous man not in the study but whom he interviewed, Vaillant asked, "How can I give a logical explanation for the growth of Roy Campanella, a great Brooklyn catcher who at thirty-six broke his neck, was paralyzed in all four limbs; yet at fifty the crippled Campanella seemed a greater man . . . than Campanella the baseball star had seemed at thirty" (Vaillant, 1977, p. 239). Similar things have been said about Christopher Reeve, the actor who became quadriplegic after a fall from horseback and who went on to greater performances and a forceful leadership role in the cause of the disabled. Vaillant echoed Selye: "It is not stress that kills us. It is effective adaptation to stress that permits us to live" (Vaillant, 1977, p. 374).

In the 1990s, the follow-up was extended another 15 years and compared with longitudinal studies of two other samples: 40 women who, as children in the 1920s, had been intellectually gifted, and about 300 men from poor families in Boston, followed from junior high school into their 70s. All three groups displayed considerable variation, but many subjects had transcended adversity, suggesting that human mental life has a self-healing bias. Eleven men, chosen from the poor sample because of extremely bad childhoods, had seemed at age 25 to be psychologically damaged beyond repair; 50 years later 8 of the 11 were doing well. "Man is born broken," Vaillant concluded, but "he lives by mending" (Vaillant, 1993, p. 287).

Summarizing another longitudinal study of adult Americans, Jean MacFarlane wrote, "Many of the most outstanding mature adults in our entire group, many who are well integrated, highly competent and/or creative . . . are recruited from those who were confronted with very difficult

situations and whose characteristic responses during childhood and adolescence seemed to us to compound their problems" (Vaillant, 1977, p. 299). In still another prospective longitudinal study, 698 infants born in Kauai, Hawaii, in 1955 were followed for over 40 years (Werner, 1989, 1997; Werner & Smith, 2001). About one third of them had severe deprivations and losses in early childhood, yet in their 30s and 40s one third of those – about one ninth of the total sample – developed into "competent, confident, and caring adults" (Werner, 1997, p. 103). In retrospective interviews the participants most frequently cited two protective factors: a significant supportive person outside their dysfunctional immediate families and a strong individual tendency to make the best of life.

POSTTRAUMATIC STRESS DISORDER: SOME QUESTIONS

PTSD is unusual among psychiatric diagnoses (Breslau, Chase, & Anthony, 2002). First, it is one of few diagnoses in the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (*DSM–IV*; APA, 1994), dependent on etiology, which has otherwise largely been eliminated from *DSM* because of uncertain causality. Although considering etiology is the rule in the rest of medical diagnosis (as insisted on by Rudolf Virchow, Robert Koch, and other founders of modern medicine), in most physical illnesses the anatomical, physiological, and/or metabolic pathways of the disease process are largely known. With the exception of those caused by chemical agents, no psychiatric diagnosis, certainly not PTSD, has met these criteria.

Second, PTSD is unusual in that the criteria prominently include the characteristics of an *external* event (Eagle, 2002); for most *DSM* diagnoses, criteria are mainly symptoms, signs, course of illness, and other characteristics of the patient. On such criteria PTSD overlaps extensively with other diagnoses, hence the need for reference to external events. Again, in the rest of medicine, external factors – infectious agents, toxins, trauma – are often cited in diagnostic criteria. However, the pathways in the body from external agent to physical or metabolic breakdown are largely known and thoroughly justify the diagnostic consideration of the agent.

Third, most patients resist being labeled with psychiatric diagnoses, and PTSD may result in such resistance for some patients, but other patients want and seek the PTSD label. Psychiatric symptoms may be less stigmatizing if caused by a traumatic experience, especially if others were responsible. Legal issues of compensation enter into some patients' efforts to receive the diagnosis just as they do with back pain, whiplash, and other potentially trauma-induced physical ailments. Up to 94% of individuals receiving the PTSD diagnosis within the Veterans Administration system apply for compensation benefits, and high levels of disability can result in payments of $2,000 per month or more. Factitious PTSD in this context

has been repeatedly described, and some veterans carrying or seeking the PTSD diagnosis have been shown to have had no combat exposure, despite their claims to the contrary (Lynn & Belza, 1984; Sparr & Pankratz, 1983). Exaggerated or imagined episodes of combat stress may be deliberate or unintentional in different cases.

Among veterans who are inpatients being treated for PTSD, compensation appears to worsen symptom description (Fontana & Rosenheck, 1998), although this effect may not apply to outpatients. A number of studies have shown that memories of combat become worse over time and that the severity of the remembered combat exposure is correlated with the severity of current PTSD and other symptoms (Southwick, Morgan, Nicolaou, & Charney, 1997; Wessely et al., 2003). In a study of 460 U.S. soldiers who had served in Somalia, subjects remembered more combat exposure as time passed, and "severity of posttraumatic symptomatology was uniquely associated with this change, indicating a possible systematic bias in which severity of symptoms leads to increased reports of stressor frequency" (Roemer, Litz, Orsillo, Ehlich, & Friedman, 1998, p. 597).

In the civilian context, worker's compensation may serve as an incentive, as does some litigation. Authorities on psychiatry and law state that PTSD forms an important new kind of tort, increasingly figuring in civil cases (Lindahl, 2004; Pitman & Sparr, 1998; Sparr & Pitman, 1999), and that the diagnosis in these settings is subject to trivialization and abuse both within (Grisso & Vincent, 2005; Rosen, 1996) and outside the United States (Eagle, 2002; Fabra, 2002, 2003; Tennant, 2004). Efforts to develop tests that discriminate between PTSD sufferers and people trying to simulate the symptom pattern factitiously have not been successful (Hickling, Blanchard, Mundy, & Galovski, 2002), and experiments show that coaching simulators of PTSD enhances their ability to evade detection (Bury & Bagby, 2002).

In addition, PTSD is among the psychiatric diagnoses that have been in flux for many years (McHugh, 1999). During World War I "shell shock" was defined but was thought to be more physical than psychological, due literally to the shock wave of the explosion (A. Young, 1995). However, it was recognized that some men came back with specifically psychological damage, and such diagnoses as hysteria and neurasthenia were applied. By World War II, a book titled *Traumatic Neuroses of War* defined emotional disorders resulting from the psychological stress of combat (Kardiner, 1941).

The recognition of a disorder called "Vietnam War: Post-Vietnam Syndrome" markedly raised awareness of combat-related psychological damage (Davis, 1992). Veterans returning from this particular war may have been especially vulnerable due to hostility at home and a general sense of failure as the war ended unfavorably for the United States. This is consistent with research on the importance of cognitive framing of trauma to the likelihood of later symptoms. However, the essence of the etiology for

many men was combat itself. It is also clear that similar symptoms can arise in the wake of many other kinds of trauma (Schnurr, Friedman, & Bernardy, 2002; Yehuda, 2002b).

PTSD entered the *DSM* in 1980 (*DSM–III*) and expanded greatly thereafter (A. Young, 1995, 2000). There is no doubt that this is partly due to the recognition of an important and previously under-recognized disorder. However, it is well established that culture influences *DSM* diagnoses (Konner, 1995; Nuckolls, 1992; Summerfield, 2002). For example, in the early *DSM*s there was an illness called "homosexuality." This was transformed over the decades until the sole reference to sexual orientation became a subset of Sexual Disorder Not Otherwise Specified: "persistent and marked distress about sexual orientation" (APA, 1994, p. 538), which also occured in heterosexuals. Premenstrual syndrome (PMS) evolved into a diagnostic category for research purposes only: premenstrual dysphoric disorder, a subset of Depressive Disorder Not Otherwise Specified (pp. 715–18).

In addition to changes in diagnostic categories in response to cultural influence, application of the labels may depend on cultural differences. For example, through the 1970s diagnoses in the schizophrenia spectrum were applied far more frequently in the United States than in the United Kingdom, which diagnosed bipolar disorder and other mood disorder spectrum labels to the same patients (Pope & Lipinski, 1978), with important implications for treatment. In the past decade there has been a very large expansion in the diagnosis attention-deficit/hyperactivity disorder, including a widening application to adults (Kanapaux, 2002; Rutter, 1998). Bulimia is a relatively new diagnostic category clearly influenced by culture; binging and purging was a culturally accepted practice among well-to-do ancient Romans. This does not condone the practice nor deny its different meaning in a culture that disapproves it; here, bulimia is a disorder. But it does show the power of culture over behaviors we consider symptoms. Probably a culture of exaggerated concern about body image and a subculture of extreme dieting and even binging and purging have increased bulimia's frequency. Finally, there are marked sex differences in the application of diagnoses on *DSM* Axis II, personality disorders that tend to be stable. Antisocial personality disorder is far more often applied to males than females, for example, whereas histrionic personality disorder is far more often applied to females (Nuckolls, 1992). This could be because of a legitimate sex difference in the underlying processes, but it could also be influenced by gender stereotypes.

Regardless of the specific application of the labels, psychiatric diagnosis has important weaknesses as well as negative consequences (McHugh, 1999). It was shown decades ago that it is relatively easy to feign psychosis well enough to be given a diagnosis of major mental illness. Eight different individuals, all free of noteworthy mental illness, were admitted to

12 different psychiatric hospitals and held several weeks until discharge (Rosenhan, 1973). Even when appropriately applied, psychiatric labels carry a significant social stigma with economic consequences and sometimes tend to shift the locus of control from the individual to the health care system, potentially weakening self-reliance and self-healing. They can result from or encourage malingering, and some patients succumb to the temptations of the secondary gains of illness, which can also slow recovery (Franklin, Repasky, Thompson, Shelton, & Uddo, 2002). With the exception of substance abuse disorders, some organic syndromes, and possibly PTSD, no diagnosis in *DSM–IV* has a known etiology, and none has a medical test that qualifies for routine use. Given that psychiatric treatment, whether pharmacological or psychological, is not without risk, these general questions about diagnosis should give us pause.

As for PTSD itself, the diagnosis has come into very widespread use on the basis of research that does not always meet the highest scientific standards (McHugh, 1999). In some settings political and moral judgments play important roles (Eagle, 2002). Media sources have repeatedly invoked the label inappropriately after PTEs in the news, but mental health professionals have often cooperated in the abuse of this and related diagnoses – as after September 11, 2001.

For example, Richard Mollica, MD, director of the Harvard Program in Refugee Trauma, was quoted in the October 17, 2001, issue of the *Journal of the American Medical Association* as saying, "starting around the Thanksgiving holiday and through the New Year, a major mental health crisis will emerge in the city and surrounding area" (Stephenson, 2001, p. 1824). Allen Keller, MD, director of the Bellevue/NYU Program for Survivors of Torture, was quoted as saying,

> Arguably, the entire city has been exposed to horrible trauma, and primary care physicians . . . need to touch base with all of their patients and be very conscious that when individuals present with somatic complaints – stomachaches, headaches, what have you – those problems may be manifestations of stress reactions from these recent horrible events. (p. 1824)

Spencer Eth, MD, medical director of Behavioral Health Services at Saint Vincent Catholic Medical Centers in New York, was quoted in September 2001 on the mental health website HealthRising.com as expecting "huge increases in the prevalence of traumatic grief, depression, posttraumatic stress disorder and substance abuse in the New York City metropolitan area at the least. This is an unprecedented disaster, and its psychiatric toll will be enormous" (Kaplan, 2001). James Nininger, MD, then president of the New York State Psychiatric Association, wrote in a letter in the *New York Times* of September 30, 2001, that the psychiatric problems caused by the attacks would continue to emerge for years, not just in people directly affected, or even just in New Yorkers, but "also among those who viewed

the horrific scenes on TV." In other words, billions of people throughout the world.

The projected avalanche of trauma-related mental illness never materialized. A random-digit-dialed telephone survey of adult Manhattanites 6 months after 9/11 showed a prevalence of probable PTSD of 0.6% (Galea et al., 2003). Total utilization of mental health services in Manhattan went from 16.9% in the 30 days before that date to 19.4% in the 30 days following it (Boscarino, Galea, Ahern, Resnick, & Vlahov, 2002). A study comparing the 22-week period following 9/11 with the same period in the 2 previous years surveyed Washington, D.C.–area residents for mental health clinic utilization (Hoge, Pavlin, & Milliken, 2002); there was no overall increase, although there were significant increases in utilization by children with anxiety and stress reactions, as well as an increase in adult adjustment reactions, which are not mental disorders. A Centers for Disease Control random-digit-dialed telephone survey of residents of Connecticut, New Jersey, and New York between October 11 and December 31, 2001, was published in their respected *Morbidity and Mortality Weekly Report* (Centers for Disease Control, 2002). They found that 50% of people participated in a memorial service; 75% had "problems attributed to the attacks," of whom 12% reported "getting help," mostly from family, friends, and neighbors; 48% experienced anger; 3% of alcohol drinkers said they drank more; and 21% of smokers said they smoked more. One percent of nonsmokers said they had started to smoke. (Percentages of those who decreased or ceased usage were not reported.) Twenty-seven percent reported that they missed work, most because of evacuation or transportation problems. Eighty percent said they watched more media coverage than usual. Nothing mentioned in this document falls into the category of morbidity, much less mortality; nowhere does it suggest that all these responses are adaptations, most of them healthy ones. In fact, one study that considered the possibility of positive psychological effects of the September 11 tragedy found them. A self-report on-line questionnaire based on the Values in Action Classification of Strengths was completed by 4,817 Americans in the two months following that date, and showed increases in gratitude, hope, kindness, leadership, love, spirituality, and teamwork; ten months after September 11, the effect was attenuated but still present (Peterson & Seligman, 2003).

Who gets PTSD after a PTE, and why? This holds great practical and theoretical interest. Most prospective studies begin just after the trauma, which limits our knowledge of what symptoms may have preceded it. Pretrauma neuroticism strongly predicted PTSD among women who suffered a pregnancy loss (Engelhard, van den Hout, & Kindt, 2003). However, in a prospective study of World War II combat exposure, psychological vulnerability before combat exposure predicted later non-specific psychiatric symptoms but not PTSD, while combat exposure itself predicted PTSD

symptoms but not other kinds of psychopathology (K. A. Lee, et al., 1995). A meta-analysis of 68 studies showed that seven variables predicted PTSD: prior trauma, prior psychological adjustment, family history of psychopathology, perceived life threat during the trauma, posttrauma social support, peritraumatic emotional responses, and peritraumatic dissociation, with the last factor having the strongest association (Ozer, Best, Lipsey, & Weiss, 2003).

Some studies of identical twins discordant for combat exposure support the role of combat in symptoms of PTSD (Roy-Byrne et al., 2004) and in one of its physiological markers, increased heart-rate response to a sudden loud noise (Orr et al., 2003). In another genetically controlled study, however, 222 monozygotic and 184 dizygotic twin pairs were compared on exposure to trauma and PTSD symptoms; concordance was higher in the monozygotic twin pairs for both risk of exposure to trauma and (given a trauma) the likelihood that PTSD would develop (Stein, Jang, Taylor, Vernon, & Livesley, 2002), suggesting a genetic continuum of vulnerability. Lower intelligence and negative personality traits are pretrauma behavioral predictors (McNally, 2003a). Smaller hippocampal volume has been found in several studies of PTSD victims (Hull, 2002; Lindauer et al., 2004; Villarreal et al., 2002). However, a study of twins discordant for combat exposure showed that the non-combat-exposed twin had reduced hippocampal volume comparable to that of the exposed twin, and that PTSD symptom severity in the combat-exposed twin could be predicted from the hippocampal volume of his non-combat-exposed brother as well as from his own (Gilbertson et al., 2002; Sapolsky, 2002).

A related problem is comorbidity, which complicates the diagnosis and raises questions about pretrauma symptoms and vulnerability. A variety of psychiatric disorders, prominently including substance abuse disorders, appear comorbidly with PTSD and often have symptoms that overlap with those of PTSD. In the National Comorbidity Survey (NCS) of almost 6,000 subjects, pretrauma history of affective disorder predicted PTSD in women and both a history of anxiety disorder and parental mental illness predicted it in men (Bromet, Sonnega, & Kessler, 1998). Other studies have found a variety of comorbid psychiatric disorders. In one typical study over 40% of subjects with PTSD also met criteria for major depression, although these were considered separate and distinguishable, especially by peritraumatic anxiety and dissociation (Shalev et al., 1998). A sample of Gulf War veterans were prospectively studied in a cross-lagged model that showed that PTSD and major depression interacted reciprocally over time to worsen each other's symptoms, except that one PTSD symptom, hyperarousability, appeared to precede but not follow major depression (D. J. Erickson, Wolfe, King, King, & Sharkansky, 2001).

Another approach to the comorbidity question is factor analysis of psychiatric disorders in large community samples such as the NCS (Krueger,

1999), which yielded three dimensions: an anxiety–misery factor (representing mainly mood disorders), a fear factor (phobias and panic), and an externalizing disorders factor (antisocial personality and substance abuse). A subsequent study using a separate subsample of the NCS yielded the same three factors using a different factor-analytic method (Cox, Clara, & Enns, 2002). This second study considered PTSD in relation to the factors and showed that PTSD loads moderately (.39), with mood disorders and generalized anxiety, on the anxiety–misery factor but weakly on the externalizing (.14) and fear (.10) dimensions.

DSM–IV Axis II disorders, especially borderline, obsessive-compulsive, avoidant, and paranoid but also schizotypal and self-defeating personality disorders, have also been shown to be very common among PTSD patients (Southwick, Yehuda, & Giller, 1993). Axis II disorders are by definition not the result of substances, injury, or particular stresses, so they must precede the trauma in many PTSD patients with Axis II diagnoses and could increase vulnerability.

Twin studies are particularly useful. A study of comorbidity in combat-discordant Vietnam-era twins showed that identical twins among men with PTSD had significantly more mood disorder symptoms than identical twins among non-PTSD combat controls or dizygotic twins among those with PTSD; this and other findings in the study suggested that major depression, GAD, and panic disorder are part of a postcombat syndrome and that a shared genetic vulnerability contributes to the association between PTSD and major depression, and between PTSD and dysthymia (Koenen et al., 2003). A similar study showed that part of the vulnerability for PTSD comes from preexisting childhood conduct disorder or adult antisocial personality disorder (Koenen, 1999). Clearly some comorbid psychiatric disorders precede the trauma and may be markers of preexisting vulnerability. Is the PTSD label becoming a substitute for such diagnoses as depression, anxiety, and panic disorder, among many others – not to mention normal emotions such as grief, fear, and rage? We simply do not at present have research that answers this question.

Consider the analogy of certain disorders associated with pregnancy and delivery. Gestational diabetes mellitus Type 2 (GDM) and preeclampsia (hypertension of pregnancy with proteinuria) are by definition pregnancy-induced diseases. However, even if these conditions are not apparent before pregnancy, they often persist long after it and may become chronic or chronically recurring. The incidence of chronic noninsulin-dependent diabetes mellitus (NIDDM) may be as high as 50% of women who previously received the diagnosis of GDM (O'Sullivan, 1991; Tan, Tan, Lim, Tan, & Lim, 2002) without prior evidence of diabetes. In one study, about a fourth of women with GDM had NIDDM 1 year postpartum and another 15% had impaired glucose tolerance (Metzger et al., 1985). Some of these

women probably had undetected pregestational impairment of glucose tolerance unmasked by pregnancy whereas in others the chronic problem was induced by pregnancy. Another prospective study showed that 14.8% of women with severe preeclampsia or eclampsia (diagnoses given only in pregnancy) went on to develop chronic hypertension, as opposed to 5.6% of a control group (Sibai, el-Nazer, & Gonzalez-Ruiz, 1986). Patients may be euglycemic or normotensive, respectively, for months to years, before late-onset NIDDM or chronic hypertension develop.

Physicians are interested in the patient's history and want to know, for example, that a 35-year-old woman with NIDDM developed glucose intolerance or hypertension for the first time during a pregnancy at age 28. But they do not use the terms "gestational diabetes" or "pregnancy-induced hypertension" to refer to such a patient. PTSD patients, however, are referred to by this label regardless of how long it has been since the trauma or whether there are comorbid disorders (depression, anxiety-spectrum disorders, OCD, substance abuse) that overlap in symptom picture with PTSD. Standard psychiatric references state that the differential diagnosis of PTSD and these disorders (Davidson, 1995) is difficult and that the most important clues are the first onset after occurrence of a trauma and the presence of trauma-specific intrusive memories and dreams. Neither of these differentiating criteria, considered critical, would clearly distinguish an underlying disorder unmasked by trauma from a disorder caused by trauma.

Depression and the anxiety disorders are now known to be genetically linked (Kendler, Neale, Kessler, Heath, & Eaves, 1992), and some anxiety-spectrum disorders respond well to the same neurotransmitter reuptake inhibitors effective in depression, even when highly serotonin selective; the same medications are also effective in OCD, although often at higher doses (Boerner & Moller, 1999; Kilts, 2003; Vaswani, Linda, & Ramesh, 2003). Even the HPA-axis abnormalities considered distinctive of PTSD are found in some studies to occur in depression and other disorders and to be tied more to depression than to trauma history (Newport, Heim, Bonsall, Miller, & Nemeroff, 2004; Smith et al., 1989). These findings suggest the possibility of a biological continuum with PTSD of some important psychiatric disorders that can be difficult to distinguish from PTSD. Furthermore, stressful life events in the recent past have always been taken into account in the *DSM* diagnosis of mood disorders. The term "diathesis" was used by older physicians to mean "a constitution or condition of the body which makes the tissues react in special ways to certain extrinsic stimuli and thus tends to make the person more than usually susceptible to certain diseases" (Dorland, 1965). It is likely that a constitutional predisposition to mood and/or anxiety disorders (and perhaps dissociative disorders) is a diathesis for psychiatric trauma, which can unmask these disorders

or the underlying tendency toward them. This concept has received little attention in the PTSD research literature and in the clinical pragmatics of differential diagnosis.

Many studies have begun with subjects with psychiatric symptoms and probed strongly for a variety of past experiences presumed to have been traumatic, including events and processes common in family life. We know that this approach often distorts memory and that overly eager mental health workers, police officers, and other authority figures can create "memories" of things that did not happen (Loftus, 2000, 2003). This alone should encourage caution in our efforts to elicit memories of trauma retrospectively, sometimes many years after the fact. That the diagnostic criteria for PTSD include "inability to recall an important aspect of the trauma" (APA, p. 428) increases the risk of memory distortion by authority figures during interviews. Repressed memories exist (Loftus, 1993), but the process of eliciting or reconstructing them is fraught with problems.

During the 1980s some clinicians began expanding the diagnosis of multiple personality disorder (MPD), itself presumed to be linked to PTSD, from a rare disorder to a very common one and then to a rare one again (McHugh, 1995; McHugh, Lief, Freyd, & Fetkewicz, 2004; McHugh & Putnam, 1995). During the heyday of the diagnosis, popular clinical manuals advised clinicians to reinterpret a remarkable range of symptoms as evidence of MPD and other dissociative disorders and to presume the existence of past trauma that, it was believed, would explain the "MPD" (McNally, 2003b). Many patients spontaneously or under strong "therapeutic" encouragement remembered things that could not have occurred – abduction by space aliens and baby-sacrificing rituals – and were clearly products of dissociation, suggestion, or both. This wave of clinical enthusiasm had the unfortunate affect of raising doubts about the suffering of real PTSD patients (Ofshe & Waters, 1994; Wright, 1994).

Is there a risk of repeating this mistake today? Some studies enroll subjects with continuing stresses – advanced cancer, for example – confounding PTSD with ongoing stress itself (Jacobsen et al., 2002). Some purport to investigate the PTSD resulting from head trauma, completely confounding the psychological sequelae of the trauma with physical brain damage (Mollica, Henderson, & Tor, 2002; Williams, Evans, Wilson, & Needham, 2002). Some mental health professionals claim to be able, through retrospective interviews, to find trauma in the past of a large proportion of depressed patients; given the broad definition of trauma and the fact that we have all had unpleasant experiences, this is a questionable research strategy. Furthermore, given the weight of evidence supporting genetic influences on depression, we may be in danger of using the trauma concept to turn a trigger of illness into a presumed cause.

As with false memories of childhood abuse, preconceived notions about PTSD treatment have sometimes led to interventions that were neither

welcome nor helpful (McHugh, 1999). It is fortunate that two widely accepted treatments for PTSD, psychotherapy (Bradley, Greene, Russ, Dutra, & Westen, 2005) and both selective serotonin and serotonin/NE reuptake inhibitors (SSRIs and SNRIs; Gorman & Kent, 1999; Schoenfeld, Marmar, & Neylan, 2004), are also effective in depression, GAD, and OCD, reducing the risk that misdiagnosis and/or comorbidity will result in inappropriate treatment. Exposure therapy is effective in PTSD (Rothbaum & Schwartz, 2002) but may be inappropriate in comorbid or misdiagnosed depression or OCD. Other approaches to PTSD may be ineffective or harmful. A 20-year retrospective on inpatient treatment found its results to be disappointing by objective and subjective measures (Rosenheck, Fontana, & Errera, 1997). Debriefing has been repeatedly shown in meta-analyses to be ineffective in preventing PTSD (Rose et al., 2002; Wessely & Deahl, 2003), and some studies, including two randomized controlled trials, have suggested that debriefing and similar immediate posttrauma counseling may increase the risk (Bisson, Jenkins, Alexander, & Bannister, 1997; Mayou, Ehlers, & Bryant, 2002). Because it is well established that cognitive framing of trauma affects the likelihood of PTSD (Ali, Dunmore, Clark, & Ehlers, 2002; Dunmore, Clark, & Ehlers, 2001; Ehlers & Clark, 2000), such effects may be the result of negative cognitive framing in some debriefing procedures.

PTSD is real (Schnurr et al., 2002; Yehuda, 2002b). People who have been through concentration camps, combat, natural disasters, serious auto accidents, and rape (among other stresses outside of normal life) are vulnerable to it and deserve help. Its physiology may be distinct from that of ongoing stress – depressed instead of elevated cortisol (Yehuda, 2002a) – although this remains controversial (Newport et al., 2004; E. A. Young & Breslau, 2004). It is disabling to many, with symptoms such as vigilance, fear, anger, light and easily disturbed sleep, revisiting and rehearsing the trauma, avoiding people and places associated with the trauma, withdrawing generally, and maintaining a muted level of affect. All these disturbing symptoms can become chronic and impairing and deserve to be clinically addressed.

Nevertheless, the typical response to acute psychological trauma is recovery over time. Symptoms in the immediate aftermath of the trauma, now known in *DSM-IV* as acute stress disorder (A. G. Harvey & Bryant, 2002), decline with time in most and resolve in many. For example, 52 men who experienced a severe avalanche showed a decrease in stress reactions from 3 weeks to 4 months and a persistent reduction at 12 months, whereas only subjects who experienced repeated stress exposure over the 12 months had increased symptoms (Johnsen, Eid, Laberg, & Thayer, 2002). Of 84 primary care patients who met the criteria for PTSD on presentation, 2 years later 69% no longer met the full criteria and 18% had a full recovery, with comorbid disorders predicting worse outcomes (Zlotnick et al., 2004). In

106 consecutive patients admitted to a trauma unit with severe accidental injuries, 5 met full and 22 met some criteria for PTSD 2 weeks after the trauma; at 12-month follow-up, the numbers had declined to 2 and 13, respectively (Schnyder, Moergeli, Klaghofer, & Buddeberg, 2001).

Of course, not everyone exposed to even severe stressors develops PTSD (Yehuda, 2002b). A review of PTSD following terrorist attacks worldwide showed an incidence of 28% in those closely affected (Gidron, 2002). The *lifetime* prevalence of PTSD in 140 war journalists, who often experience multiple and ongoing stressors, was 28.6% (Feinstein, Owen, & Blair, 2002). Of 77 individuals exposed to a mass shooting spree at a courthouse, 5% developed PTSD (Johnson, North, & Smith, 2002). A 3-year follow-up of victims of serious automobile accidents showed an 11% incidence of PTSD, predicted by persistent health and financial problems, litigation, and several peritraumatic variables (Mayou et al., 2002). Studies of resilience are far less common than studies of PTSD itself, yet these may hold the clues to primary and secondary prevention.

For certain victims of trauma the pre- or posttraumatic cognitive framing of the experience may be critical (Ehlers & Clark, 2000). PTSD after combat is strongly associated with low motivation to serve in the military (Z. Kaplan et al., 2002). Ordinary citizens are much more likely than members of security forces to suffer PTSD after terrorist attacks (Gidron, 2002); the latter's objective experience is the same or worse, but the cognitive preparation and framing are very different. In 181 male firefighters who worked as rescue workers in the Oklahoma City bombing of 1995, 13% had PTSD, compared with 23% of male primary victims (North et al., 2002). Vietnam veterans who suffer from PTSD show improvement if they care for their grandchildren (Hierholzer, 2004); might similar positive, active experiences help other trauma victims cope, even though these activities take place outside the mental health system? Many ongoing investigations are considering the value of intervention by mental health professionals after acute stress, but we should not ignore the healing resources of family and community.

CONCLUSION: STRESS, RESILIENCE, AND ADAPTATION

This chapter began with a description of life in our environments of evolutionary adaptedness, finding it to be stressful and subject to frequent trauma, yet indicative of the great human capacity for resilience. It went on to consider the physiological bases of normal and abnormal responses to stress and, to the extent we understand it, resilience. Finally, it raised some questions about the diagnosis and treatment of PTSD as currently construed in the mental health professions and the media.

A greatly simplified and speculative model of three possible responses to acute stress is shown in Figure 15.2. It proposes a possible continuum of stress responsiveness, construed to include three broad types of

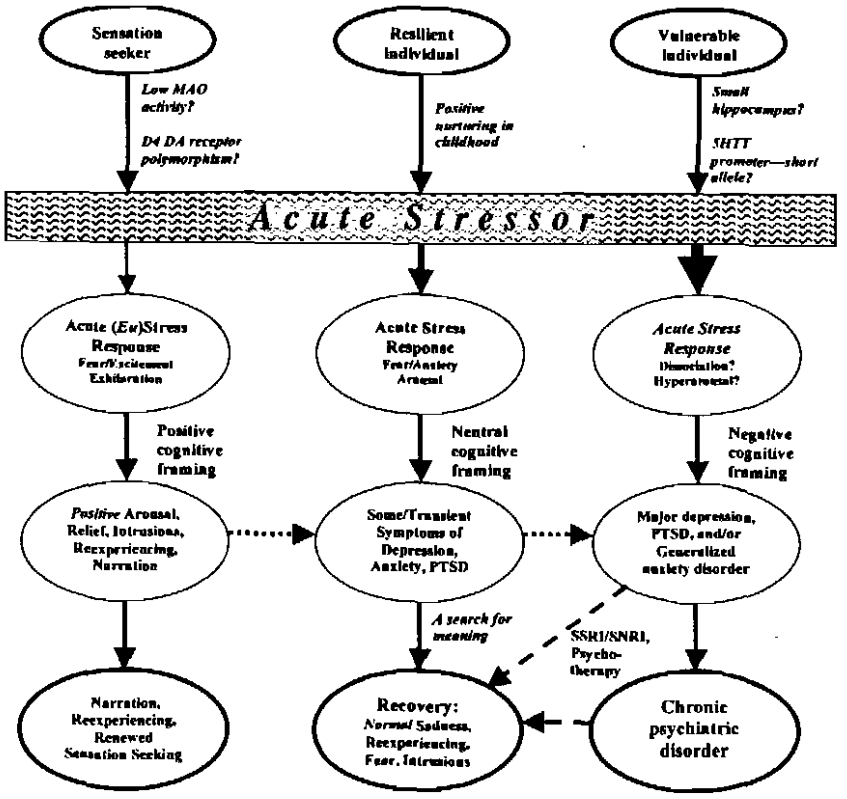

FIGURE 15.2. A speculative model of three responses to acute stress. MAO, monoamine oxidase; DA, dopamine; 5HTT, serotonin transporter; SSRI/SNRI, serotonin selective/serotonin-norepinephrine reuptake inhibitors; dotted arrows, unlikely but possible transformations; dashed arrows, response to treatment.

people. The first, sensation seekers, are the small proportion who desire and seek out experiences that would be stressful to most people. Studies suggest that they may share biological markers including low activity of the enzyme monoamine oxidase (MAO) and a particular polymorphism of the D_4 dopamine receptor (Zuckerman & Kuhlman, 2000). These people find novel, stressful, and dangerous experiences desirable and exhilarating, especially if positively framed and sought. The second and largest group is fairly resilient in the face of serious stress but develops acute stress responses which, if they persisted, might qualify as psychiatric disorders. However, few do persist. The third category consists of the minority who are vulnerable to the development of psychiatric illness during and after acute stress. This group may develop PTSD, depressive disorders, and/or GAD in the wake of the stress, and symptoms may persist for years. Small

hippocampal volume may be a marker of vulnerability. Carriers and/or homozygotes for an allele for the shorter of two promoters of the serotonin transporter gene are vulnerable to depression and some other psychiatric problems following serious psychological stress (Caspi et al., 2003; Grabe et al., 2005; Kendler, Kuhn, Vittum, Prescott, & Riley, 2005).

This is a speculative model advanced for heuristic purposes, and it has many possible flaws. For example, the first and third categories may not be independent or mutually exclusive and therefore may not be actual ends of the proposed continuum. However, because it is clear that some people are exceptionally resilient in the face of severe stress, the continuum is probably real.

I did not and do not challenge the existence of PTSD nor the all-too-real suffering of those who have it. I have simply reviewed studies and trends that suggest that PTSD is a problematic diagnosis, that it has been overapplied, that it is sometimes exaggerated or factitious, that there are incentives for such distortions, that culture and media influence it and other psychiatric diagnoses, that a great majority of PTSD sufferers also have other psychiatric diagnoses whose symptoms often overlap with those of PTSD, and that little is known about the antecedents of PTSD that make some individuals more vulnerable than others. Most important, I have tried to emphasize that most of us, like our evolutionary and historical predecessors, are resilient.

Where does this resilience come from? I would point to three possible sources. First, social and family supports in ancestral settings were extremely strong, even more so during childhood, and these bonds of aid and empathy are protective. Second, people are far more resilient than some mental health professionals would have us believe, and the experience of self-reliance and survival in these challenging environments must have strengthened resilience. Third, cognitive framing of stress and responses to stress in these cultures emphasized strength, resilience, and the necessity to survive to meet other stresses and to protect dependents.

Because these were the conditions under which we evolved, it is not surprising that we have a GAS consisting of predictable and often appropriate, although sometimes excessive, physiological responses encoded in the genes. Following Selye, we distinguished between *dis*tress and *eu*stress and emphasized that eustress – normal stress – is the essence of life itself. *Dis*tress is also central to life, and the responses organisms make to it determine whether they survive and reproduce. Thus, coping with distress has determined the course of our evolution, and consequently we are relatively good at it. What about the exceptional distress represented by trauma? This too was common in evolution, and the adaptive response produced by thousands of generations of selection was "Let's get on with life."

Still, many must have failed. It does no good to say they were selected against, because in our modern culture we properly insist on buffering

those who are less resilient against some severe stresses that others weather more easily. We do not say, with natural selection, "The devil take the hindmost." We intervene. This is fine, provided the intervention does more good than harm.

HGs had levels of mortality from infectious disease that we would not countenance for a moment. The fact that deadly microbes were ubiquitous and natural carries little weight, and that is as it should be. However, we have often prescribed antibiotics unnecessarily, producing adverse instead of salutary effects for many individuals (for example, by killing good bacteria) and damaging the public health by selecting for and breeding antibiotic-resistant organisms. Furthermore, we overestimate the importance of antibiotics in bringing about the great decline in mortality in modern times; in reality, such variables as nutrition, plumbing, pest control, and other community-level factors accounted for almost all of it (McKeown, 1995). We also underestimated the ability of patients to fight infectious diseases with the adaptations provided by a highly evolved immune system. Analogies are limited, but it is possible that our scientific and medical arrogance could once again lead us to overtreat and overintervene, this time in the psychological realm.

Consider the symptoms of PTSD itself in the light of our evolutionary history. In the human EEAs, an adaptive response to stress might include a needed vigilance (with lighter sleep), appropriate fear and/or anger, revisiting and rehearsing the trauma to consolidate its lessons, and avoiding or withdrawing from sources of danger. In some situations even a general withdrawal and a pattern of muted affect – for example, if bravado or anger had helped to cause the trauma – might be adaptive. These symptoms may be less adaptive in a culture like ours, and in any case they would not be adaptive if they become chronic. But the question is not whether PTSD can be debilitating; rather, it is where we draw the line between resilience and vulnerability and whether our interventions sometimes do more harm than good.

Would the Vietnam veterans, with the same combat experience, have been as vulnerable to PTSD if they had been welcomed home as heroes? Would the people of London suffering the blitz have been better off if Churchill, instead of saying, "Death and sorrow will be companions of our journey; hardship our garment; constancy and valor our only shield. We must be united, we must be undaunted. We must be inflexible" (Churchill, 2003), had said, "We'd better send in thousands of trauma counselors"? The stiff upper lip is a famous British cultural adaptation; Churchill believed in it, and he addressed his beleaguered nation in words that helped to make it real. Many people rise to stresses when they are encouraged to believe that they can and that much depends on them, and succumb when they are told that they cannot. After September 11, some mental health authorities predicted a nationwide epidemic of PTSD. Similar predictions were made

in the media immediately following the July 7, 2005, terrorist bombings in London, and the counterproductive potential for such predictions raised legitimate concern (Wessely, 2005). This is the kind of thing that prevents people, legislators included, from taking the mental health professions seriously. It also diminishes the suffering of those at or near (in distance or relationship) ground zero.

What actually happened after September 11, of course, was that the American people entered into a new era of vigilance, revisiting and rehearsing the event, learning from it as much as possible, and, yes, getting very angry at the perpetrators and all who gave them comfort. This was a healthy and adaptive response to the trauma and has allowed almost all Americans to adjust without clinical intervention. No doubt many of those who experienced the event at or near ground zero or who lost loved ones in it required and will continue to require help. But many others, even among those close to the event, channeled their grief into adaptive paths, such as agitating for and getting an independent commission to revisit and rehearse the event with them at a national level, going to fight against Al Qaeda in Afghanistan, or learning more about Islamic culture and Islamist terrorism. It is intriguing that in some studies the intrusive memories in PTSD patients are of the warning signs of the forthcoming trauma, rather than or in addition to the trauma itself (Ehlers et al., 2002); this strongly suggests an adaptive mechanism for avoidance learning.

Finally, it is possible to construe some of the symptoms of PTSD as a search for meaning. Revisiting and even to some extent obsessing about the trauma is in part a normal urge to integrate and understand a dreadful experience. Thinking about it and incorporating it into one's own life story is in part a product of the human drive toward narrative, and talking about it can reflect a normal need for a listening ear. Memory – remembrance – has value in itself; it is a part of our selves even when it is bad.

Consider the stories of two combat veterans. One, a Vietnam veteran speaking many years after the war, said,

> I can't get the memories out of my mind! The images come flooding back in vivid detail, triggered by the most inconsequential things, like a door slamming or the smell of stir-fried pork. Last night, I went to bed, was having a good sleep for a change. Then in the early morning a storm-front passed through and there was a bolt of crackling thunder. I awoke instantly, frozen in fear. I am right back in Viet Nam, in the middle of the monsoon season at my guard post. I am sure I'll get hit in the next volley and convinced I will die. My hands are freezing, yet sweat pours from my entire body. I feel each hair on the back of my neck standing on end. I can't catch my breath and my heart is pounding. I smell a damp sulfur smell. Suddenly I see what's left of my buddy Troy, his head on a bamboo platter, sent back to our camp by the Viet Cong. Propaganda messages are stuffed between his clenched teeth. The next bolt of lightning and clap of thunder makes me jump so much that I fall to the floor. (Davis, 1992, p. 470)

The second, a World War II veteran, spoke more than 60 years after his event:

> I was hid behind a big tree that was knocked down or fallen. And I could see these Germans in the woods across this big field. And I saw this young kid crawling up a ditch straight towards my tree. So I let him crawl, I didn't fire at him, but when he got up, within three or four foot of me, I screamed at him to surrender, and instead of surrendering, he started to pull his gun towards me, which was instant death for him. But this young man, he was a blond, blue-eyed, fair-skinned, so handsome, he was like a little angel, but I still had to shoot him, and it didn't bother me the first night, because I went to sleep, I was so tired, but... the second night, I woke up crying [voice breaks] because that kid was there [voice breaks]. And to this day I wake up many nights crying... over this kid. I still see him in my dreams. And I don't know how to get him off my mind. (Robertson, 2005)

At first glance the two statements seem similar. The first contains evidence of symptoms meeting most of the *DSM-IV* criteria for PTSD, and the speaker's pain and need for help are palpable, even many years after his horrific experience. The second, especially when heard in spoken form, feels very different. It is a poignant reminiscence of a tragic event in which a soldier had no choice but to kill a beautiful young man much like himself. Yes – 6 decades later – his sleep is disturbed, his thoughts are intrusive, he sometimes wakes up crying, he does not quite know how to get the boy he killed completely off his mind. If he should ask for help, of course he should get it, but he does not appear to view his condition as psychopathology. This, I would argue, is not PTSD. It seems, rather, a fairly normal reexperiencing of a life-changing event, a tragic moment in which he was forced to kill another human being. Why should such an event not be remembered, intrusively or otherwise? Why should it not cause sadness? These memories are part of the meaning of this man's life, of a conscience troubled permanently by a uniquely powerful act, of an identity forged in part by that act and that experience. They show, to himself and others, how very much he cares about human life, and they help to keep the memory of that angelic-looking German boy alive.

Viktor Frankl, a psychiatrist who spent 3 years as a prisoner in Auschwitz, emerged with the belief that suffering must be dealt with through a search for meaning, and he developed a method of psychotherapy based on that belief (Frankl, 1984). Finding meaning in suffering and in life is, he believed, the best and perhaps the only way for a human being to adapt. He recounted the story, first set down by a German bishop, of a Jewish woman who a few years after World War II wore a bracelet with baby teeth set in gold. Questioned by a doctor, she explained, "This tooth here belonged to Esther, this one to Miriam," and so on. These teeth had been saved, one from each of her nine children, all of whom were murdered in Auschwitz. "How can you live with such a bracelet?" the doctor asked.

"I am now," she answered quietly, "in charge of an orphanage in Israel" (Frankl, 2000, p. 142).

Not everyone who experiences severe trauma – even violent rape, even Auschwitz – develops PTSD. It is essential for us to understand who does and who does not, and what the psychological markers are, not just of vulnerability but also of resilience. Unless research is designed with this question in mind, PTSD runs not just the risk of becoming a passing fad, like the MPD of a decade ago, leaving truly needy people in its wake, but a self-fulfilling prophecy in which some people become psychologically debilitated because they are told that they will. Most people should be told that they are resilient, not just because it is a healthy message, but because it is the legacy of our biological evolution and is usually true. Then, and only then, can we identify the minority among us that is not so resilient and direct the scarce resources of clinical intervention where they are needed and where they belong.

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