Balancing Vulnerability and Resilience in Damage Prognostication

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In civil cases alleging psychic injuries, forensic psychiatrists are asked to diagnose the injury and assign or apportion causal factors. If a plaintiff was damaged or an employee injured, the relevant inquiries are the nature and severity of the injury, the steps needed to remedy it, and the anticipated timeline for maximum medical improvement (MMI). This last item is discomfiting, especially when coupled with permanence. The insurance and disability industry's definition of MMI is when the individual's "condition has plateaued and is unlikely to benefit in a meaningful way from further medical treatment" (Ref. 1, p 90). The interpretation of this tautology falls on us, the experts, to state. Sometimes an attorney will press for a report, saying there is a forensic economist waiting to supply financial numbers for a report. The forensic expert appears to be the linchpin in this process, raising the important question of how such opinions can be supported.

Because we do not predict the future and have little science to inform prognostications, we have only the obvious guideposts. These include that the person is or is not improving, the type and quality of treatment to date, and a sense of the person's cooperation with the healing process. Depression,² posttraumatic stress disorder (PTSD),³ and mental disorders generally,⁴ examined in numerous metaanalyses, correlate with excess risk of death. Actuarial tables and statistics, while valuable to allied professionals such as vocational and rehabilitation specialists, are of little value in the assessment of an individual. Indeed, the fact that a person with a mental disorder (typically anxiety, depression, or PTSD) is slow to improve may arouse suspicion of malingering in the context of litigation or receipt of benefits. Malingering should not be inferred solely when the subject has a chronic course, which is a statistical outlier. Persons with genuine psychic injuries show a spectrum of recovery types. If the theory of personal injury litigation is to make the injured party whole,⁵ then we ignore the reality that no one is ever the same after an experience, traumatic or otherwise. There is no practicable way for expert witnesses to quantify unpleasant memories, attitudinal changes, subtle behavioral alterations, or enduring sadness.

Science may be available to apply in individual cases. We argue that forensic psychiatrists should be availing ourselves and our clients of it in addition to the standard clinical approach to prognostication. This editorial briefly explores theories of knowledge about recovery, from folk wisdom to studies of vulnerability, resilience, and neuroplasticity, concluding that we should be mindful of a vast knowledge base that could inform our reports and testimony. Note that this is not an exhaustive exploration of the science, which can be accomplished through many of the references. Rather, it is a prompt to evolve our methods, along the lines of recent editorials.^{6,7}

Received Wisdom

Recovery from psychic injuries is variable, and the dynamics of recovery are mysterious, compounded further by monetary considerations. The epidemiology of mental disorders permits us to recognize immediately that most individuals do not have anxiety or mood disorders. Of those who experience

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potentially traumatic events, many do not show posttraumatic symptoms, while others are affected severely and may have a chronic course.⁸ In the middle are those who have symptoms that dissipate variably. Each individual adapts to stress, a complex matter that involves neuroplasticity.⁹ With current technology and standards, it is difficult to predict one individual's trajectory from another's. Without scientific guidance, we are gazing into a crystal ball.

Prognostications in expert reports are often based on a folk-psychological approach (or received wisdom); that is, what everyone knows to be true, how we were taught, and what our peers do. This is not to say that it is right or wrong, only that we have limited science to apply to an individual when we are challenged on the basis for an opinion. Examples include: anyone would be traumatized by what happened here; the amount of psychic injury is proportional to the nature of the stressor; because the physical injury is permanent, the psychic injury is permanent; the individual will have mental symptoms as long as the pain condition persists; a person who has not improved by now will never recover; and the individual will get better once the case is settled. Another thread of received wisdom is the conflation of residual symptoms and permanence: because memories and feelings about the event are still present, the individual will never recover fully. This logic is more insidious because it involves a degree of insincerity on the part of the expert, namely, sidestepping what constitutes a mental disorder. With the definition of PTSD being categorical rather than dimensional, for example, expert reports that stretch the boundaries are scientifically and ethically suspect.

These questions are not new. Over 20 years ago, Robert Simon, M.D. reviewed the literature on PTSD prognosis.¹⁰ The results shed light on some of the folk-psychological ideas mentioned here. Risk factors for PTSD, he found, include magnitude of the stressor and type of stressor (e.g., physical assault or injury, rape, combat, and natural or technological disasters), chronicity of the symptoms, comorbid psychiatric and medical conditions, early life stress, and poor social supports.

Thus far, psychiatric expert witnesses have relied on untested nostrums. There may be ways to reconcile, or at least temper, received wisdom with science. The following sections highlight areas of knowledge that have the potential to inform our opinions. Whether such refinements will be applauded or rejected by attorneys is itself unknown. Because discussions of trauma and PTSD are ubiquitous and causality is often examined, most of the examples below will concern PTSD.

Allostatic Load

We were taught in biology class that organisms, when perturbed, are programmed to return to status quo ante, Cannon's principle of homeostasis.¹¹ This tenet of biology, in the case of simple systems such as body temperature and blood pressure, is applicable to humans.12 The model predicts that, over time, persons experiencing stress will return to the way they were before. In humans, it is not automatic that injured persons return to preinjury normal or that they become either stronger or more disease-prone as a result of their experience. The traditional view of stress, promoted by Selye, called it "general adaptation syndrome," implying human resilience.¹³ Our complexity, memory, and consciousness itself, however, may frustrate a robust application of homeostasis to psychic trauma. Cannon's principle has been refined by appreciating that humans change constantly with experience and that adaptation, comes at a price, the concept termed allostasis.^{12,14,15}

Allostasis suggests adaptation to circumstances occurs outside of conscious control. To cope with stress, an organism may reset its "operating range" to accommodate the new system. It may do this by displaying what we would consider pathological symptoms. The cumulative result of the changes is allostatic load (i.e., wear and tear).15 McEwen and Stellar¹⁵ cite PTSD as an example of a consequence of allostatic load linked to hypervigilance and hostility. Other examples of the burden of allostatic load are less apparent than the syndrome of PTSD, which most psychiatrists are comfortable identifying and diagnosing. By the time we see them in litigation, some individuals will already have altered physiology, but it will be hiding as changes in stress hormones, which we do not measure regularly. These changes will not be apparent on examination but are likely to have long-term health consequences. When severe or toxic stress results in cascades of neuroendocrine and metabolic consequences, there is evidence that it is associated with early senescence.³

In a more subtle biological causation, an individual with PTSD who self-treats allostatic load with alcohol may perceive temporary benefits but experience significant risks to health and life expectancy.¹⁵ For forensic purposes, it may not be practical to use indices of allostatic load as verification of PTSD and its course. Autonomic activation is a key clinical feature of PTSD, useful in recording a psychiatric diagnosis but rarely measured. To complicate matters, there is a spectrum of reaction types resulting in phenotypic variants. For example, individuals with psychopathic traits override autonomic responses to stress through emotional intelligence.¹⁶ By contrast, one could question whether persons with over-reactive stress responses deserve more compensation than those who equilibrate with minimal allostatic consequences. It would be hard to say without objective correlates. There might be usable applications when we conduct screening or fitness-for-duty examinations in police officers and soldiers. In disability assessments, where impairment is based on observable and reported capacities and behaviors, information about underlying physiology could be relevant. AAPL's practice resource on disability evaluations¹⁷ is silent on physiological correlates of functioning, presumably because this domain is neither required by the gatekeepers nor considered standard practice. Relying on manuals, operational definitions, and self-reported inventories (e.g., the WHODAS 2.0¹⁸) cannot be the limit of what behavioral science has to offer.

There may be guidance on the question of documenting, if not quantifying, allostatic load. About 10 years ago, a group of researchers compiled what was known about allostatic load biomarkers, which comprised a substantial body of research, from 1999 through 2009, which is beyond the scope of the present discussion.¹⁹ The biomarkers reflected neuroendocrine (e.g., cortisol) and metabolic (e.g., lipid profiles) indices. There was predictive value in tracking the biomarkers;

e.g., the appearance of metabolic syndrome was associated with cognitive decline. Some of the studies affirmed epidemiological health and mortality surveys including biomarkers. Correlations between outcome and adverse life experiences such as poverty confer optimism on clinically based prognostications. The effects of stress during the developmental period can give rise to diverse outcomes: ways, such that the physiological expression of the stress response system to stressors is processed differently as either positive, tolerable, or toxic stress (Ref. 19, p 13).

ACEs and Eggshells

An individual's lifetime of stress is a factor in forensic psychiatric assessments. Data from the landmark Adverse Childhood Experiences (ACE) Study indicate that adults with exposure to trauma in childhood were at much higher risk of long-term physical, mental, and behavioral problems.²⁰ It has been fashionable to include ACEs²¹ as background features of individuals facing criminal sentencing. On the 20th anniversary of the ACE study, it was critiqued by McEwen and Gregerson,²² who cited the narrowness of the 10-item index. Among their points were that the index leaves out other elements of childhood adversity and social inequity; it "underplays the effects of adversity throughout childhood and across generations" (Ref. 22, pp 790-91); it ignores protective factors by focusing on deficits; and it emphasizes interventions over prevention.

ACEs, in the broad sense, are associated with deviations in physical and mental health. In criminal justice, for example, attorneys can argue that the defendant was subject to uncontrollable influences and was therefore less culpable. It can be inferred from the presence of ACEs that the affected individual had increased allostatic load during the developmental period. Perhaps it should be quantified. As with all attempts at mitigation, however, the presence of ACEs can be used by the prosecution as evidence that the defendant is damaged permanently and therefore incorrigible and a continued threat. Beyond the presence of ACEs, the totality of childhood deprivation and abuse is fair game in mitigation. Failure of trial counsel to proffer testimony about it can result in a claim of ineffective assistance of counsel.²³

In the civil domain, at least in the adult disability and personal injury sector, there is little, if any, focus on either allostatic load or the general effects of development on the prognosis for conditions acquired during adulthood. That is, adverse developmental experiences may affect the course and permanence of psychiatric conditions brought about by trauma and stressors in adult life. This suggestion, too, is arguable. The defendant in a personal injury case, such as a motor vehicle accident, may claim that the damage caused is mitigated by a preexisting condition. The plaintiff will fall back on the "eggshell plaintiff"

Accumulated damage over time and the biological embedding of adversities during sensitive developmental periods are nevertheless experienced and manifested in heterogeneous

principle: that the defendant is responsible for the damage caused to the plaintiff with an unknown preexisting condition, regardless of the prior degree of impairment.²⁴ Thus, from the plaintiff's point of view, accumulated allostatic load that may have created predisposition to PTSD could be used legitimately. Making this argument would require expert testimony, however, which at this time is speculative without biomarkers or valid correlates of stressinduced susceptibility.

Resilience and Neuroplasticity in Recovery

Personal injury and disability reports are concerned with causality, diagnosis, and expected recovery or permanence. Because we know there are pathways to recovery and not all persons with the same injury or descriptive diagnosis have permanent conditions, an actuarial approach tends to leave out properties of the individual that may aid predictions. These properties are resilience^{25–27} and neuroplasticity,⁹ the former being a composite indicator of return to health and the latter of enduring changes in brain and related systems that correspond to acute, chronic, and toxic stress.

A compact definition of resilience is "a non-pathologic or adaptive behavioral and neurobiological response to traumatic stress" (Ref. 28, p 1268). We can understand resilience as a multifactorial phenotype that reflects central nervous system and peripheral changes in response to traumatic incidents that confer a positive or protective outcome on wellbeing. Psychological resilience as a scientific concept emerged in the 1970s, stemming from observations that not everyone experiences a negative outcome following childhood trauma.²⁹ Indeed, some people exhibit positive adaptations (e.g., the adjacent concept of posttraumatic growth) instead of or in addition to adverse effects following traumatic incidents. An inventory of positive posttraumatic growth features, compiled by Tedeschi and Calhoun, appeared in 1996³⁰ and was followed by empirical research.³¹ Their original sample of approximately 600 college students responded to about 34 items suggested by the literature to represent possible consequences of adverse events.³⁰ From the responses, the researchers selected 21 items grouped into five domains: relating to others, new possibilities, personal strength, spiritual change, and appreciation of life. Through these domains, the researchers were able to correlate cognitive and attitudinal changes with positive psychological growth. In their follow-up report, the authors

cited widespread acceptance of the concept of posttraumatic growth as a process.³¹ Among their conclusions was that outcomes left individuals with what amounts to positive allostasis: "Posttraumatic growth is not simply a return to baseline-it is an experience of improvement that for some persons is deeply profound" (Ref. 31, p 4). More recently, investigators have piloted a 46-item, seven-subscale battery for risk and resilience, based on a sample of about 300 volunteers.³² After detailed item analysis, the seven salient domains included self-reliance, positive relationships, negative relationships, peer victimization, emotional dysregulation, neighborhood danger, and stressful events. These domains, the authors conclude, will require correlation with biological substrates of risk and resilience. In that vein, there have been recent attempts to characterize stress resilience across multiple levels of individuals' biology and functioning in socioecological realities³³ and in anatomically oriented studies of brain functioning.³⁴

We do not endorse the immediate use of quantitative measures of risk and resilience in relation to prognostication in personal injury determinations. While perhaps not ready for incorporation into expert reports, valid and reliable indices of resilience would contribute to a precision-psychiatry approach to forensic work.³⁵ Such information will be especially useful in demonstrating the actual effect of stress on an individual, avoiding speculation or reliance on traditional wisdom to prognosticate outcomes. In the case of neuroplasticity, forensic experts will want to know if changes in brain structure or function that give rise to a variety of symptoms (e.g., memory deficits, reexperiencing, and hyperarousal) are reversible. It has been pointed out that neuroplasticity, by itself, does not protect against psychopathology and may be the source of clinical symptoms; that is, maladaptive physiology can increase allostatic load.⁹

As one might expect, the literature on the psychology and biology of resilience is vast. A panel convened in 2013 concluded that the determinants of resilience should include "genetic, epigenetic, developmental, demographic, cultural, economic, and social variables" (Ref. 25, p 1). Each of these items, in turn, has its own infrastructure, rendering a distilled version for forensic purposes merely aspirational until there is a signal from courts that such information would be admissible. The massive research on neural pathways of mood, anxiety, learning, and memory can be found elsewhere (e.g.,

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Cathomas *et al.*,³⁶ Horn and Feder³⁷). Though psychiatrists believe stress and trauma result in enduring neuronal and epigenetic changes, forensic-grade biomarkers for the presence of PTSD and pathways leading to either adaptation or pathology have yet to be elucidated.³⁸ On the psychometric side, while resilience scales have some predictive power in determining who is at risk for PTSD,²⁷ they are applied generally in military screening, not forensic assessments that are done after a traumatic event.

As to the question of prognosis in PTSD, folk psychology eventually will be augmented by genetic, epigenetic, and other quantifiable information on markers of vulnerability and comorbidity, studies of which are ongoing.³⁹ A related question is whether traumatic memories can be modified or even erased.⁴⁰ If that were feasible, ethical, and safe, there could be a duty imposed on a plaintiff to mitigate damage; refusal could result in reduction of the award. Extinction learning, in which a new memory is formed that reduces expression of a fear memory, represents the primary model for traumatic memory modification, and enhancing that process is a focus of research.⁴¹ Reconsolidation, in which a fear memory is disrupted at the time of recall, represents another model for modification or even erasure.⁴² Presently, however, noninvasive fear-extinction therapies predominate while animal models explore more permanent solutions.43 Similarly, while medications such as propranolol and prazosin are sometimes used to mitigate autonomic symptoms, perhaps treatment with MDMA (3,4-methylenedioxymethamphetamine)-assisted psychotherapy will become an industry standard that bears on the question of MMI.44 Such a development could illuminate the limits of a plaintiff's duty to mitigate damage and whether everything must be tried before MMI can be invoked.

Resilience can be inferred from the absence of clinical PTSD after a traumatic event but could be corroborated via measurable brain characteristics. A 2016 study of 40 persons with PTSD and 36 matched trauma-exposed healthy controls measured hippocampal volume with magnetic resonance imaging.⁴⁵ The subjects with PTSD received 10 weekly sessions of prolonged exposure therapy. Similar to previous studies, treatment did not affect hippocampal volume. Greater volume, however, was shown in the subjects who later responded to treatment and in those who were in the control group. Thus, there was a signal

that greater hippocampal volume had predictive value. This type of information could be used by a defense attorney in a personal injury case to argue that awarded damages should be reduced because treatment will be more effective. It is unlikely that such a tactic would prevail over traditional wisdom, at least as a stand-alone factor.

The inevitable question is whether to incorporate scientific information into an expert report; it is a mixed blessing on both sides of civil litigation. On the plaintiff's side, counsel would like to see hard evidence of a trauma-related disorder beyond descriptive and self-reported data. Ideally, counsel would prefer biomarkers indicating a plateau in resilience, which would support permanence. If one or both is absent, however, and if the standard of practice permits such evidence, defense counsel may take the opportunity to raise the specter of malingering. On the defense side, counsel would be looking for absent PTSD biomarkers or for positive markers of resilience. By the time arguments get to this level, such evidence may already be factored into the actuarial tables of forensic economists and related fields. Admissibility of novel evidence may be hampered if opinions are inferred from aggregated data. For example, whereas neuroimaging is interpreted from a valid cohort group, courts tend to be more interested in individual data rather than group data.⁴⁶ The challenge of using group data to infer the condition of an individual (i.e., the G2i problem) is not insurmountable:

Should research on the biological correlates of mental disorders develop to the point where neuroimaging and genetic data can identify accurately the presence or absence of a disorder, concerns about the validity and reliability of many psychiatric diagnoses may diminish considerably (Ref. 46, pp 750–51).

Discussion

Both individuals who do and those who do not develop symptomatic post traumatic conditions exist on a spectrum of both susceptibility and recovery factors. Hemingway⁴⁷ put it this way:

The world breaks every one and afterward many are strong at the broken places. But those that will not break it kills. It kills the very good and the very gentle and the very brave impartially. If you are none of these you can be sure it will kill you too but there will be no special hurry (Ref. 47, p 239).

Forensic professionals should make no such assumptions and need an upgrade in the basis for

prognostication. Looking at PTSD, for example, across time and culture, Konner stated in 2007, "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" (Ref. 13, p 326).

With the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III),⁴⁸ in 1980, the classification of PTSD among anxiety disorders reified what we knew, that psychiatry had given back to veterans something that had been missing during the Vietnam War era and afterward: respect for what they had experienced. It was a sociopolitical gesture. Since then, trauma has become a household word, like depression and anxiety. Nonmilitary types have emerged (e.g., genocide survivors; abused children; and persons exposed to violent crimes, rape, and natural disasters), and victimology has flourished as have industries of therapeutics and research. It is time to move beyond descriptive criteria and to broaden the basis for forensic predictions of recovery.

In 2007, McHugh and Treisman published a blistering critique of PTSD, which they considered an overworked and misleading construct.⁴⁹ In addition to observing widespread political and social overlay that obfuscates the core clinical topics, they were especially critical of the replacement of "bottom-up" assessments (e.g., Meyerian, comprehensive, thoughtful) with "top-down" diagnostics (e.g., checklist- or criterion-based, phenomenological). They also observed psychiatry's ambivalence about forensic entanglements: "Perhaps the only issue about mental disorders following trauma that psychiatrists identified, quarreled about, but never resolved was the role of compensation and self-serving litigation in either provoking or sustaining these states of mind" (Ref. 49, p 215). Since this critique, it has been increasingly apparent that PTSD has become a source of income for attorneys, who require expert testimony.⁵⁰ It is no wonder that the battlegrounds are labeling (e.g., PTSD versus adjustment disorder) and permanence.

As McHugh and Treisman remarked, with the publication of DSM-III, aided by antiwar psychiatrists and veterans' advocates, PTSD arose "[l]ike Athena full-grown from the forehead of Zeus" (Ref. 49, p 216). Aside from verification that the person actually experienced a traumatic event, reliance on self-reported data adds awkwardness for both clinicians and expert witnesses. We find resilience is overlooked in forensic matters in favor of a focus on recovery when these are not mutually exclusive concepts. Indeed, human beings are not the same after trauma, and this may manifest as both negative and positive psychological outcomes, even in the same person, with underlying neurobiological adaptations that we may or may not understand. For these reasons, forensic psychiatrists should be aided by independent markers of disturbances that correlate with vulnerability, resilience, and other indices, so that we can retire the crystal ball.

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