University of Arkansas at Little Rock Law Review

Volume 24
Issue 1 *The Ben J. Altheimer Symposium: The Impact of Science on Legal Decisions*

Article 2

2001

Gatekeeping Stress: The Science and Admissibility of Post-Traumatic Stress Disorder

Edgar Garcia-Rill

Erica Beecher-Monas

GATEKEEPING STRESS: THE SCIENCE AND ADMISSIBILITY OF POST-TRAUMATIC STRESS DISORDER

Edgar Garcia-Rill[*]
Erica Beecher-Monas[**]

I. INTRODUCTION

Scientific evidence, increasingly drawn upon to justify legal decisionmaking, is an unavoidable facet of modern litigation. It is fundamental to criminal justice and to tort litigation, just to give two examples. Moreover, according to the United States Supreme Court, in three seminal cases,[1] and the recently amended federal rule of evidence,[2] it is up to federal judges to evaluate the scientific validity of such evidence before it may be relied upon. This is a tall order for many judges, however, who are stymied by the science component of their gatekeeping duties, and focus instead on rules of convenience that have little scientific justification. The result is unwarranted decisions at both ends of the spectrum: scientifically uncontroversial evidence that would have little trouble finding admissibility under a general consensus standard[3] is often excluded and evidence that is scientifically baseless is too frequently admitted.

---

* Professor of Anatomy and Neurobiology, Professor of Psychiatry, University of Arkansas for Medical Sciences. Director of Research, Arkansas Center for Neuroscience.

** Associate Professor of Law, University of Arkansas at Little Rock William H. Bowen School of Law. J.S.D., 2001 Columbia University; LL.M., 1995, Columbia University; J.D., 1988, M.S. (Anatomy), 1978, B.A., 1976, University of Miami. The author thanks Patti Stanley, Christian Harris, and Amy Dunn for their helpful research assistance on this article.

1. Kumho Tire Co. v. Carmichael, 526 U.S. 137 (1999); Gen. Elec. Co. v. Joiner, 522 U.S. 136 (1997); Daubert v. Merrell Dow Pharms., Inc., 509 U.S. 579 (1993).

2. Congress promulgated amendments to Federal Rule of Evidence 702, purporting to codify the *Daubert* standard. The new rule provides:

> If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

FED. R. EVID. 702.

3. Frye v. United States, 293 F. 1013, 1014 (D.C. Cir. 1923) (explaining that "while courts will go a long way in admitting expert testimony deduced from a well-recognized scientific principle or discovery, the thing from which the deduction is made must be sufficiently established to have gained general acceptance in the particular field in which it belongs").

Nowhere is this phenomenon more evident than in the admissibility of post-traumatic stress disorder (PTSD) evidence. A brain dysfunction characterized by a particular set of symptoms,[4] PTSD ought to be admissible whenever mental state or injury is at issue. In a significant percentage of people, exposure to trauma—combat, domestic violence, rape, sexual abuse, burns, disasters, violent crime, etc.—results in PTSD. The symptoms of this disorder include, as explained in greater detail below, re-experiencing the trauma, avoiding situations or activities reminiscent of the original trauma, and increased arousal or hypervigilance. Curiously, however, PTSD testimony is often excluded from evidence (generally without any analysis of its scientific validity) in precisely those circumstances where it would be the most helpful to the factfinder. In sharp contrast, psychological syndrome testimony (such as battered woman syndrome), which rests on very shaky ground indeed and cannot meet standards of scientific validity, is widely admitted (also largely without any analysis of its scientific merit).

Testimony about PTSD comes into court in a number of guises. It has been proffered as justification—self-defense—testimony in criminal cases.[5] For example, a battered woman may seek to explain why she shot her sleeping husband,[6] or an abused young man may try to explain why he thought it necessary to shoot his father.[7] PTSD testimony may also be proffered as a way of explaining why it took a rape victim several days to report the crime[8]—the issue of consent—or why a young woman felt coerced into carrying her boyfriend's gun in her purse for a robbery even without an explicit threat to her[9] (the issue of duress). It may be proffered as mitigation testimony in sentencing,[10] or in civil

4. *See infra* Part II.B.1 (discussing symptoms of PTSD).

5. Justification traditionally is explained as an absence of moral blameworthiness, resulting in legal exoneration. *See generally* John L. Austin, *A Plea for Excuses*, *in* FREEDOM AND RESPONSIBILITY 6 (Herbert Morris ed., 1957). Professor Greenawalt argues that the distinction between justification and excuse is incoherent because in both instances the result is legal exoneration. *See* Kent Greenawalt, *The Perplexing Borders of Justification and Excuse*, 84 COLUM. L. REV. 1897, 1927 (1984).

6. *See, e.g.*, Ohio v. Manning, No. 97CA006921, 1999 WL 49095 (Ohio Ct. App. Feb. 3, 1999) (proffering battered woman syndrome testimony in self-defense).

7. *See, e.g.*, Ann W. O'Neill, *Menendez Retrial Plays Differently*, L.A. TIMES, Mar. 3, 1996, at A1 (noting that the defense of the Menendez brothers was abuse in both of their two trials).

8. *See, e.g.*, Ohio v. Martens, 629 N.E.2d 462 (Ohio Ct. App. 1993) (introducing PTSD testimony to explain delay in reporting rape).

9. *See, e.g.*, United States v. Brown, 891 F. Supp. 1501 (D. Kan. 1995) (proffering testimony that battering relationship compelled defendant to commit drug-related crimes).

10. *See, e.g.*, United States v. King, No. 98-2203, 1999 WL 475562 (8th Cir. July

cases as a compensable injury in airline crashes[11] or other major catastrophes.

Because they do not understand how PTSD satisfies the *Daubert* criteria, judges frequently fail to admit PTSD evidence. By providing a background on the physiology of PTSD and a framework for the admission of such evidence, this article seeks to help judges make better evidentiary decisions. This article argues that understanding certain basic principles of brain physiology will enable judges to make better admissibility decisions. Based on criteria scientists themselves use to assess validity,[12] as well as the Supreme Court's requirements in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*,[13] *General Electric Co. v. Joiner*,[14] and *Kumho Tire Co. v. Carmichael*,[15] this article outlines a framework for sound analysis of scientific evidence regarding PTSD. Part II offers a primer on the physiology of stress, describing the essential brain mechanisms needed to understand PTSD and how it affects the body. Part III then examines the *Daubert* factors and demonstrates how PTSD evidence meets the *Daubert* standard, and explains the types of cases where PTSD evidence should be admitted. This is important information for judges because the consequence of ineffective judicial gatekeeping is significant not only for the litigants, but also for a society that depends on the proper functioning of the judicial system for an efficient balance of compensation and prevention. At stake is the credibility of the judiciary and the proper functioning of the judicial system.

II. THE PHYSIOLOGY OF STRESS

This section is a primer on the "science" of PTSD and is intended for non-scientists. The first part of the section provides a baseline understanding about the biology of stress, while subsequent parts will briefly describe the anxiety disorders in general, before dealing specifically with the symptoms, incidence, and consequences of PTSD.

6, 1999) (proffering PTSD testimony as basis for downward departure from sentencing guidelines).

11. *See, e.g.*, Ben Shimol v. TWA, Inc., No. C-93-1586 MHP, 1996 WL 724782 (N.D. Cal. Nov. 22, 1996) (airline crash victim seeking compensation).

12. *See infra* Part II.A.2 (discussing different types of anxiety disorders).

13. 509 U.S. 579 (1993).

14. 522 U.S. 136 (1997).

15. 526 U.S. 137 (1999).

A. The Stress Response and the Anxiety Disorders

1. *The Normal Stress Response*

All of the anxiety disorders are associated with a stressful event or events, either real or imagined. The event or the memory of the event induces a specific kind of physiological arousal called the stress response.[16] Normally, the stress response is a good thing. However, when the stress response becomes repetitive or chronic, it is a bad thing. The stress response is related to the fight-or-flight response. It is important to understand what actually happens to the body when the stress response is activated. This is a kind of survival reflex, conserved in evolution because animals with a good fight-or-flight response tend to survive moments of danger or emergency.

Try to imagine a caveman, out foraging for food in the savanna, and around the next tree he comes face to face with a lion. What does the caveman's body need to do to survive? Run away fast. His muscles need all the energy they can muster, energy produced by burning glucose. Therefore, the stress response consists of squeezing glucose out of fat cells, liver cells, etc., for use by the muscles. To deliver all that necessary glucose to the muscles, the heart rate and blood pressure need to rise, and the need for oxygen increases so that respiration also increases. Along with the responses in the caveman's body, there are changes in his brain. The senses become sharper and memory improves during this response. This is due to an increase in awareness, because the reticular activating system (RAS)[17] of the brain is pounding away, and blood flow to the brain is boosted, especially to the frontal lobes. This cognitive component is needed in order to remember that man cannot outrun a lion, therefore, the caveman needs to devise an escape, perhaps by running to the nearest tree or rock and climbing to escape the lion's pursuit.

There are also a number of energy-consuming processes that the caveman will want to shut down in order to optimize energy use to meet the demands of this emergency. He will not need to use up energy digesting the roots he just ate, so his body shuts down digestion. He can always digest later. The caveman will not want to spend energy

16. Robert M. Sapolsky, *The Stress-Response and the Emergence of Stress-Related Disease*, *in* STRESS, THE AGING BRAIN, AND THE MECHANISMS OF NEURON DEATH 3-9 (Robert M. Sapolsky ed., 1992).

17. The RAS controls our sleep-wake cycles and arousal, as well as our fight-or-flight responses.

repairing tissue, reproducing, having sexual drive or fighting infections. He can do all that later, if he is still alive. Therefore, during the stress response, his body stops tissue repair, curtails the production of hormones related to sperm (or egg) production, and the immune system is suppressed. Pain is blunted, so that if he is clawed by the lion, he can still try to escape. The pain pathway is mercifully suppressed.

The manifestation of the stress response is well and good in an emergency, but one would not want to keep doing this all the time. The stress response is normally short-lasting and occasional. It is not intended for repeated, long-lasting responsiveness to the environment. On a chronic basis, the stress response can kill just as surely as a lion, but more slowly. If the stress response is elicited repetitively for prolonged periods, the increases in blood pressure can lead to heart disease, including heart attacks and strokes from the runaway blood pressure. During chronic stress, one does not repair the wear and tear of everyday aging. A simple paper cut can take days to heal, and it will scar ever so easily. Glucose utilization problems can lead to a form of diabetes, egg or sperm count drops, libido (sex drive) decreases, and the body is less able to fight infections, including cancer.

Recent studies on animals accentuate the contrast between acute (one shot, limited) and chronic (repetitive, excessive) stress. If rats are restrained in wire mesh containers for six hours a day, for the first week or two, they actually perform better on learning tasks, but by the third week, their performance falls off and becomes progressively worse.[18] While at first learning is improved, when the period of stress is prolonged, it can lead to shrinking of dendrites in the hippocampus, a part of the brain involved in learning and memory.[19] The shrinking of dendrites, the receiving elements of the neurons, translates into less complex function.[20] Less complex function in a brain region involved in memory and learning means diminished cognitive performance in at least some people suffering from PTSD.[21]

Suppose that you are a modern individual, rarely having to face a lion, but living in a crowded environment, toiling daily at a stressful job, facing large debts, shrinking income, increasing time demands, and

18. Victoria Luine et al., *Restraint Stress Reversibly Enhances Spatial Memory Performance*, 59 PHYSIOLOGICAL BEHAV. 27, 28, 30 (1996).

19. *See* Robert M. Sapolsky, *Glucocorticoids Endanger Hippocampal Neurons, in* STRESS, THE AGING BRAIN, AND THE MECHANISMS OF NEURON DEATH 119, 140 (Robert M. Sapolsky ed., 1992).

20. *Id.*

21. J. Wolfe & L.K. Schlesinger, *Performance of PTSD Patients on Standard Tests of Memory*, 821 ANNALS N.Y. ACAD. SCI. 208, 212 (1997).

family problems, none of which can be resolved quickly. Living daily with chronic stress produces a candidate for hypertension, diabetes, diminished sex drive (including impotence), unsuccessful reproduction, increased chance of suffering from cancer, and, to top it all off, the changes in the brain are making you dumber. When you are on edge and stressed, the RAS is constantly on edge, ready to be provoked. It seems as if the next little thing, like the faucet sprouting a leak, will be the last straw. That last stimulus produces a full-blown fight-or-flight response, in which you will strike out (verbally or physically) or escape (into the mind-numbing television set), reflexively, without "thinking." Even so, this still only represents an excessive fight-or-flight response. That is, the severity of these reactions are within "normal" limits and do not represent an anxiety disorder yet.

2. *The Anxiety Disorders*

There are three main categories of anxiety disorders,[22] namely panic disorder,[23] obsessive-compulsive disorder,[24] and PTSD. People with

22. AMERICAN PSYCHIATRIC ASSOCIATION, DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS (4th ed. 1994) [hereinafter DSM-IV]. *See id.* at 387-401, 417-23, 424-29.

23. Panic disorder is an anxiety disorder in which there are panic attacks, which are short periods of intense fear associated with impending doom. During these panic attacks, the heart starts to pound, there is sweating, trembling, shortness of breath, chest pain, dizziness, numbness, chills or hot flashes; essentially this is an unchecked episode of increased physiological arousal and stress. *Id.* at 397-401. Panic attacks represent an arousal response to a thought, to a fear-inducing idea, not to the appearance of a physical predator. Panic disorder usually first manifests itself during adolescence. A smaller population develops the disease in their mid-thirties. There is a genetic contribution to the disorder, sometimes running in families and being prevalent in twins at a higher rate than in the population at large. Panic disorder and obsessive-compulsive disorder each have a prevalence of about two percent of the population. *Id.* at 399, 420.

24. Obsessive-compulsive disorder also usually begins at or around puberty, especially in males, with gradually increasing symptoms that can also become incapacitating. Obsessions are persistent ideas, thoughts, impulses, or images that are considered intrusive and produce anxiety and distress. Compulsions are repetitive behaviors like arranging things, hand washing, or mental acts like praying or silently repeating words, designed to reduce anxiety or distress. *Id.* at 417-23. Just as avoidance is a survival strategy used by panic disorder patients, obsessive-compulsive disorder patients use compulsive actions and thoughts to ward off impending danger. For example, obsessive-compulsive behavior patients will wash their hands until the skin is macerated, hating every minute of it. These unfortunate individuals are slaves to their compulsions and obsessions knowing that these are inappropriate behaviors. If they do not perform the clearly embarrassing ritual, if they do not succumb to the urges, their anxiety becomes unbearable. The fear and impending doom are intolerable unless these tasks are performed, over and over again. These individuals are enslaved by their

anxiety disorder have a sensory gating deficit.[25] A sensory gating deficit is an abnormality in the process we use to gate, or filter out, sensory input. Normally, we habituate, that is, we have a reduced response, to repetitive stimuli. For example, if we hear two loud sounds spaced, say, one-half second apart, we will startle less to the second stimulus than to the first. The response to the second sound will be about fifteen percent of the response to the first. We will not jump anywhere near as high to the second sound as we did to the first. On the other hand, people with an anxiety disorder will show a response to the second stimulus of about eighty percent, jumping almost as high to the second sound as to the first. These individuals have an inability to gate or filter, to habituate, to repeated sensory inputs, thus the term sensory gating deficit. A sensory gating deficit implies excessive distractability. This means that the reactivity to stimuli in their world never wanes. All light, sound, and touch is intrusive, continuous, and punishing, driving you, well—crazy.

We are very good at detecting change. That is, we perceive change in the world around us and habituate—respond less and less—to stimuli that are repetitive. This is not just some quirk of nature. This capacity has great survival value and was thus preserved in evolution. After all, if you are continuously responding to the sound of the wind in the trees, you are less likely to detect the sound of an approaching predator. In anxiety disorder, the exaggerated responsiveness to all sensory inputs not only serves to overwhelm the afflicted individual, but also induces a great deal of fear and stress, a feeling of inability to cope, to detect that approaching predator. It induces a constant state of impending doom.

What causes this over reactivity to sensations? Obviously, the brain system controlling arousal, the startle response, and fight-or-flight responses is disturbed. This means that the RAS is not working properly in anxiety disorders. Recent studies have confirmed the well-established observation that these people also have disturbed sleep patterns.[26] These individuals are not only hyper-reactive to sensory input, but are also hyper-aroused in general. These people have difficulty falling asleep and staying asleep. When they do sleep, they awaken tired and unrefreshed. Their dreams are vivid, their nightmares intense. These are all symp-

---

brain's dysfunction.

25. R.D. Skinner et al., *Reduced Sensory Gating of the P1 Potential in Rape Victims and Combat Veterans with Post Traumatic Stress Disorder*, 9 DEPRESSION & ANXIETY 122, 127-28 (1999).

26. *See* Ruth M. Benca et al., *Sleep and Psychiatric Disorders: A Meta-Analysis*, 49 ARCHIVES GEN. PSYCHIATRY 651 (1992); Richard J. Ross et al., *Sleep Disturbance as a Hallmark of Posttraumatic Stress Disorder*, 146 AM. J. PSYCHIATRY 697 (1989).

toms of increased arousal and hyper-vigilance, and of a problem in the RAS. One of the major problems in this disease is that the RAS, allowed to throb unchecked, propels the brain into delusion and hallucination.

B. The Neurobiology of Post-Traumatic Stress Disorder

1. *The Symptoms of PTSD*

PTSD is perhaps the most common anxiety disorder in this trilogy.[27] Experiencing or witnessing an event involving death, injury, or threat, coupled with the intense fear that event generated, along with a feeling of helplessness, induces a characteristic set of symptoms.[28] First, there is *avoidance* of thoughts, feelings, places, situations, or activities that remind the person of the traumatic event. This symptom is characteristic not only of PTSD but of all of the anxiety disorders. Avoidance of places, people, things, of situations or events from which you cannot escape or flee would seem to be a natural tendency. After all, it is a strategy designed for survival, to save oneself from the unseen danger. Fear of something causes avoidance. However, fear of everything can be terribly incapacitating. For those unafflicted, this is difficult to understand. A sudden, unexplainable, intense fear will induce an escape response, a flight, avoidance. Forcing a victim of a panic attack to remain in that situation, to face the fear, to fight the unseen predator, is not appropriate. These individuals consider their lives to be in danger and will fight to survive, and even strike out in order to flee. A calm voice and a quiet environment is one way to try to ameliorate the situation. By keeping the sensory input down, at least the world outside will not add to the roar inside the brain.

By itself, avoidance can be terribly disabling in one's life, but, unfortunately, it is only the beginning. A second symptom is *hyperarousal and hypervigilance*, which are often present with the disturbed sleep. This symptom obviously indicates dysregulation of the RAS. There is a third and particularly damaging component to this disorder, a persistent *re-experiencing* of the traumatic event. Every time the traumatic event is recollected, it triggers a physiological arousal and

27. *See* R.A. KULKA ET AL., TRAUMA AND THE VIETNAM WAR GENERATION 53 (1990); Naomi Breslau & Glen Davis, *Posttraumatic Stress Disorder in an Urban Population of Young Adults: Risk Factors for Chronicity*, 149 ARCHIVES GEN. PSYCHOL. 671, 671 (1992); Dean G. Kilpatrick et al., *Criminal Victimization: Lifetime Prevalence, Reporting to Police and Psychological Impact*, 33 CRIME & DELINQ. 479, 488 (1987).

28. *See* DSM-IV, *supra* note 22, at 424.

stress response, intense fear, and anxiety of the event. Our memories are recollections from which we cannot escape. A color, a light, a sound, a word, almost anything can trigger a reminder of that feared incident, causing the same response every time, reliving the entire event in our memories. The fear, pain, helplessness, and all of the physical reactions that accompanied the initial event, are all re-experienced, along with the inevitable stress response, an exaggerated one at that.

2. *The Incidence of PTSD*

PTSD can be caused by sexual or physical abuse during childhood or adulthood, but it is not known what percent of abused children and adults (including battered women) will develop the condition. On the other hand, scientists do know that over one half of rape victims will develop PTSD.[29] About one third of combat veterans and, probably, a higher proportion of prisoners of war become affected.[30] Hostage or crime situations, torture, and even witnessing man-made or natural disasters that involve loss of life, death, or serious injury to family members or close associates, all can lead to this disorder. Estimates of the lifetime prevalence of PTSD range from one to fourteen percent in the general population.[31]

Within that significant segment of the population, the severity of the disease will vary. Some people may remain undiagnosed for years, perhaps even their entire lives. Perhaps they are Vietnam veterans who returned "upset" by their combat experiences and became "difficult." This group of veterans were different when they came home—they became jumpy and nervous, combative, and argumentative. These very ill individuals were "out of control." Other patients with PTSD had a "violent" childhood or were sexually molested when young.[32]

They now resist or fight authority or any attempt at controlling their behavior, and have trouble concentrating and finishing tasks. These individuals cannot sleep, and their interpersonal skills are atrocious; their marriages are "on the rocks" or terminate in divorce. These patients cannot abide loud sounds, sudden actions, or unexpected touching. They are not just "stressed out," they may be mentally ill.

---

29. Kilpatrick et al., *supra* note 27, at 485.
30. *See* KULKA ET AL., *supra* note 27, at 53.
31. Breslau & Davis, *supra* note 27, at 672.
32. *See* Kilpatrick et al., *supra* note 27, at 486-87.

3. *Where is the Problem in PTSD?*

The stress response is a chain reaction of hormonal activity. A stressor, any stressor, (1) induces physiological arousal in the RAS, then (2) triggers activity in the hypothalamus, then (3) signals the pituitary gland, located underneath the hypothalamus, to then (4) secrete ACTH, a hormone, into the circulation, which then (5) reaches the adrenal gland, located on top of the kidney, causing (6) production of adrenaline, the substance that leads to the stress response (that is, glucose mobilization, accelerated heartbeat, etc.). Adrenaline has a rapid effect on the various organs on which it acts, and then dissipates or is broken down. Adrenaline is the ideal substance for a quick get-away. The adrenal glands also produce glucocorticoids, which are hormones with longer action on these organs, prolonging the stress response beyond the few seconds during which adrenaline is active. Cortisol is the best-known of these "stress hormones."[33]

The brain has a nifty mechanism to terminate the stress response. Being able to end this response allows the body to go back to its housekeeping duties, like digesting, reproducing, and fighting infection. Circulating glucocorticoids in the blood reach the hypothalamus in the underside of the brain, and there shut off the further release of ACTH. That is, the same substance that is released as a result of the stress response feeds back to the regions which started the process. This process is called negative feedback, which is a typical control system, like the thermostat in your house. The air conditioning shuts off when the thermostat detects a temperature drop (too much cool air). Too much glucocorticoid in the blood leads to feedback inhibition of the hypothalamus, leading to the shutdown of ACTH release from the pituitary, which no longer induces the adrenals to release glucocorticoids. The termination of the stress response is also important because excess glucocorticoids can have a deleterious effect on the brain. The occasional stress response is not going to do any harm, but, if you are under continuous stress, high circulating levels of glucocorticoids may lead to the shrinking of dendrites, probably wherever there are glucocorticoid receptors[34] in the brain.

---

33. *See* Robert M. Sapolsky, *An Introduction to the Adrenocortical Axis*, *in* STRESS, THE AGING BRAIN, AND THE MECHANISMS OF NEURON DEATH 11 (Robert M. Sapolsky ed., 1992).

34. Receptors are like "locks" which are opened by neurotransmitter and hormonal "keys," leading to the opening of channels, "doors" in the cell membrane.

The parts of the brain with significant numbers of these receptors are the hypothalamus, the hippocampus, and the RAS. These are the three brain regions primarily involved in anxiety disorders. There is no clear scientific evidence to suggest that the difference between a normal individual living in a stressful society and an individual with anxiety disorder is the extent of damage or dysfunction in these regions of the brain. Obviously, something else happens to these brain centers in a person with anxiety disorder, especially after puberty. Likewise, something else leads some combat-exposed soldiers to develop PTSD, while their fellow combatants remain normal. Why do only one half or so of rape victims manifest this disorder but the rest do not? There must be other factors that, when combined with repeated or excessive stress responses, may lead to sufficient damage by high circulating levels of glucocorticoids to kill or render inactive many of these neurons (hippocampal, hypothalamic, RAS). The exaggerated effect of glucocorticoids is probably a major element contributing to the disorder.[35] Remember that one of the duties of glucocorticoids is to block glucose uptake into tissues and release it into the bloodstream. If glucocorticoids block glucose uptake into neurons, will these cells not die or be weakened by a lack of energy production? One possible predisposing factor placing individuals at risk for developing an anxiety disorder is head injury. If the brain has already been racked by a concussion or two, or if there is another disease like epilepsy in that tissue, are these not "debilitated" cells? The chances that the loss of glucose due to glucocorticoid exposure, sending these cells "over the edge" of recovery, are probably fairly high. Attempting to confirm or deny these effects is a very active area of research at the present time. It is also a very important area of research, one with implications for our overall health in today's stressful world.

4. *The Sound of Stress*

During the stress response, the music of the brain is driven by the percussion section, the drumbeat rising in volume with every stimulus. In anxiety disorder, the sensory gating abnormality ensures that the volume will keep increasing. If, as some researchers believe, there is damage to the noradrenergic neurons of the RAS in anxiety disorder,

35. *See* Robert M. Sapolsky, *Glucocorticoid Neurotoxicity, in* STRESS, THE AGING BRAIN, AND THE MECHANISMS OF NEURON DEATH 95-96 (Robert M. Sapolsky ed., 1992).

their activity will decrease.[36] This means that the neighboring cholinergic neurons of the RAS will be less inhibited, less regulated by noradrenergic input. The result is that arousal will be heightened during waking, and rapid eye movement (REM) sleep will be intensified, producing more vivid dreams and nightmares. In some people, this overactivity could induce hallucinations, or dreaming while awake. The pounding of the bass drums of the cerebral orchestra would be wrenching. On an everyday basis, these individuals would be less likely to withstand the psychological pressures of a stressful occupation, choosing to avoid noisy or disturbing environments and activities. These individuals would be the ones who scream and jump the highest at the action-suspense movies, if they view them at all. Those exposed to abuse or trauma would flinch at (and avoid) the violent scenes of a movie or news program. Fear and impending doom cast a dark shadow over their lives, in which imagined catastrophes will kill them or their loved ones. The music of the brain would be constantly playing a requiem, albeit very loudly. The overwhelming pounding would lead to exaggerated, hair-trigger fight-or-flight responses. Chronic overactivity in the RAS can also produce "hypofrontality," decreased blood flow to the frontal lobes.[37] That means that the part of the brain most responsible for critical judgement is less active, so that the making of decisions is impaired. It should be noted that baseline cortical metabolism is decreased in PTSD patients,[38] but these individuals will show increased blood flow compared to normal controls when exposed to trauma-related stimuli or abuse-related imagery.[39]

An individual previously abused physically and mentally is likely to respond in the same manner when stressed, to respond violently, to lash out indiscriminately, reflexively. If beaten as a child for crying, that person as an adult could be more likely to abuse his own children when stressed. Those children could, in turn, grow up to beat their children, and the behavior becomes a "sick" family tradition. This could be one

36. Edgar Garcia-Rill, *Disorders of the Reticular Activating System*, 49 MED. HYPOTHESES 379, 382-83 (1997).

37. *Id.*

38. J. Douglas Bremner et al., *Positron Emission Tomography Measurement of Cerebral Metabolic Correlates of Yohimbine Administration in Combat-Related Posttraumatic Stress Disorder*, 54 ARCHIVES GEN. PSYCHIATRY 246, 246, 250 (1997).

39. Israel Liberzon et al., *Brain Activation in PTSD in Response to Trauma-Related Stimuli*, 45 SOC'Y BIOLOGICAL PSYCHIATRY 817, 821-25 (1999); Lisa M. Shin et al., *Regional Cerebral Blood Flow During Script-Driven Imagery in Childhood Sexual Abuse-Related PTSD: A PET Investigation*, 156 AM. J. PSYCHIATRY 575, 581-83 (1999); Jon-Kar R. Zubieta, *Medial Frontal Cortex Involvement in PTSD Symptoms: A SPECT Study*, 33 J. PSYCHIATRIC RES. 259, 262-63 (1999).

reason why anxiety disorders run in families. At some point, however, one of these individuals may seek help, stop responding excessively to stressful stimuli, and attempt to break the cycle of violence. Otherwise, the family legacy of abuse will be passed down from generation to generation.

If there is damage to the cells of the hippocampus, the music of the brain will be less expressive, less brilliant. Memory and learning will be somewhat impaired, but some memories will be repeated, reinforced, and amplified. Those vivid memories of the trauma, of the abuse, will be written in stone. Every time they are recalled, every time any little thing reminds them of that event, the memory will induce another full-blown stress response. The re-experiencing of the traumatic event in PTSD is extremely damaging because it will boost the stress response and lead to high circulating levels of glucocorticoids, putting even more brain cells at risk. The next stress response will then be a little stronger, producing more damage. A vicious cycle of brain damage caused by a bad memory from which they cannot escape.

This may be one reason why PTSD can last a lifetime. While about fifty percent of patients with PTSD can be said to be in remission three years after the trauma (can live normal lives, even with occasional symptoms), over forty percent are still considered to meet criteria for PTSD ten years after the traumatic event.[40] That is, for many victims, this is a lifelong disorder,[41] complicating issues related to compensation. PTSD can also have a delayed onset, sometimes of many years, such as in cases of combat veterans who developed PTSD after retirement.[42]

5. *Turning Down the Volume*

After a hard day at the office, we reach the relative safety of home to the news that the daughter has a new tattoo, the son has been suspended from school, the dog threw up on our favorite chair, and the spouse was fired. It is time to open the bar. One of the easiest ways to calm down the RAS is with alcohol.[43] Alcohol turns down the volume

---

40. *See* Ronald C. Kessler et al., *Posttraumatic Stress Disorder in the National Comorbidity Survery*, 52 ARCHIVES GEN. PSYCHIATRY 1048, 1057, 1059 (1995).

41. *See* Caron Zlotnick et al., *Chronicity in Posttraumatic Stress Disorder (PTSD) and Predictors of Course of Comorbid PTSD in Patients With Anxiety Disorders*, 12 J. TRAUMATIC STRESS 89, 89 (1999).

42. *See* Craig Van Dyke et al., *Posttraumatic Stress Disorder: A Thirty Year Delay in a World War II Veteran*, 142 AM. J. PSYCHIATRY 1070, 1072 (1985).

43. *See* John C.M. Brust, *Ethanol, in* NEUROLOGICAL ASPECTS OF SUBSTANCE ABUSE 190, 193 (1993).

of the music. The conduction of impulses, of action potentials, along the multiple synapses of the RAS is slowed by alcohol. Just as one might use alcohol to relax, to recover from the stress response, so does the anxiety disorder patient self-medicate with alcohol. In virtually all disorders in which the output of the RAS is amplified—schizophrenia, anxiety disorder, depression, and manic-depression—patients will self-medicate excessively with alcohol.[44] This is not necessarily an addiction to alcohol, for these individuals will tell you that they hate the hangovers and the loss of control, but alcohol turns down the blaring music, the intrusive world.

Sometimes this creates a problem in diagnosis. For example, if a teenager has a silly idea that she does not want to leave the house and she drinks a lot, her parents may conclude she has a drinking problem. Or does she? Drinking may be masking a panic disorder. Only a competent clinician may be able to arrive at an accurate diagnosis. Before embarking on an expensive treatment regimen for alcoholism that will clearly fail in this case, alternative diagnoses need to be considered. Alcohol is a very good anxiolytic (anxiety-reducing) drug.[45] Unfortunately, it also has serious side effects. Alcohol preferentially affects little neurons in the brain called granule cells.[46] These are located in the cortex, cerebellum, and hippocampus. What happens then? The music is missing the high notes, and the control is gone. The cortex is playing simpler music, so one is not a great intellect when drunk. The hippocampus is having a hard time remembering anything. The cerebellum is where the real danger lies. The regulation of rapid, highly integrated movements by the cerebellum is impaired, along with balance and equilibrium. Performing accurate movements after consuming alcohol is difficult at best. Driving is impaired and should not be attempted. The policeman who stops the drunk driver gives him a neurological exam of cerebellar function, including walking a straight line heel-to-toe, placing the feet together and arms apart, then touching the nose, closing the eyes (so that visual input cannot be used to orient),

---

44. *See* Edward J. Khantzian, *The Self-Medication Hypothesis of Addictive Disorders: Focus on Heroine and Cocaine Dependence*, 142 AM. J. PSYCHIATRY 1259 (1985); Alexander C. McFarlane, *Epidemiological Evidence About the Relationship Between PTSD and Alcohol Abuse: The Nature of the Association*, 23 ADDICTIVE BEHAVS. 813 (1998); Roger D. Weiss et al., *Drug Abuse as Self-Medication for Depression: An Empirical Study*, 18 AM. J. DRUG & ALCOHOL ABUSE 121 (1992).

45. *See* Brust, *supra* note 43, at 193.

46. *See* Gerhard Freund, *Neuropathology of Alcohol Abuse*, *in* ALCOHOL AND THE BRAIN: CHRONIC EFFECTS 3, 10 (Ralph E. Tarter & David H. Van Thiel eds., 1985).

tilting the head back (to test balance control), and standing on one leg (all tests used by the neurologist when assessing cerebellar control).

There are other drugs that can be used to turn down the volume of the RAS.[47] One type of agent is a benzodiazepine, like clonazepam, that amplifies the action of inhibitory neurons all over the brain, including those in the RAS. The good news is that it calms down the drumbeat of the brain. The bad news is that use of benzodiazepines calms down activity in many other regions, interfering with cognitive processes, producing drowsiness, sleepiness, and lethargy, leading patients to resist therapy. This is a typical problem in the treatment of anxiety, when treatment includes the use of tranquilizers, sedatives, and barbiturates to calm the agitated patient. These drugs basically turn down all of the RAS, making it difficult to stay awake, much less carry out everyday activities. More recently, compounds with more specific actions have been used in the treatment of anxiety disorder. Xanax is a benzodiazepine-like compound which also has antidepressant effects. The good news is that Xanax relieves anxiety without making people sleepy, but the bad news is that it is very short-lasting, requiring repeated dosing, and it can be very addictive, producing repeated withdrawal symptoms as each dose wears off. The search is on for a longer-lasting, less addictive substitute. There are some agents currently being tested which may act more specifically in the RAS, with promising anxiolytic actions.

6. *Managing Stress, Preventing Anxiety*

Just as child abuse (or adult battering) can ultimately lead to PTSD, the opposite kind of treatment can allow the child brought up in a loving environment to cope better with the stresses of life. Even animal studies have shown, for example, that rat pups raised by mothers that tend to lick and groom them a lot, compared to those whose mothers do not, will have amazing lifelong effects.[48] These pups will release less glucocorticoids when placed in a stressful environment, even as adults. They will show more exploratory behavior, indicative of decreased anxiety. They will also show larger hippocampal formations (probably due to greater elaboration in the growth of dendrites). These pups, just

47. *See* Linda S. Brady, *Stress, Antidepressant Drugs, and the Locus Coeruleus*, 35 BRAIN RES. BULL. 545, 549-51 (1994).

48. *See* Monique Vallee et al., *Long-Term Effects of Prenatal Stress and Postnatal Handling on Age-Related Glucocorticoid Secretion and Cognitive Performance: A Longitudinal Study in the Rat*, 11 EUR. J. NEUROSCI. 2906, 2914 (1999).

as well-loved and nurtured children, will be better able to cope with stress, and will possibly be less likely to develop conditions like PTSD when exposed to trauma. Giving our children love and affection not only makes them feel good, but protects them for the rest of their lives. What better gift to give a child?

What about battered adults? The key to successfully dealing with stress or surviving an anxiety disorder is to cope.[49] Coping strategies are mostly common sense but are well-supported by scientific study. For example, in coping with stress, those who find an outlet for life's frustrations will do better (that is, release less glucocorticoids). Prayer, meditation, sports, and music all help release the pressure of stress. One's attitude is important. In the face of terrible news or conditions beyond our control, hoping for the best but preparing for the worst will reduce our stress response during a crisis, and if things get worse. Controlling the situation also relieves stress, but only if we do have control. However, if something is broken beyond repair, we should not try to fix it, as it will only add to the stress. In times of stress, those with a support group of family and friends will cope better than those without. Those of us who develop these good coping strategies will have abated stress responses, good digestion, better sexual and reproductive lives, and probably live longer.

All of these coping strategies also apply to anxiety disorders. In fact, these strategies are even more important for dealing with such disorders but we must remember that there is structural damage in anxiety disorder—that there is an organic problem—not just a psychological one.[50] This means that an accurate diagnosis must be made, and

---

49. *See Managing Stress, in* ROBERT M. SAPLOSKY, WHY ZEBRAS DON'T GET ULCERS, A GUIDE TO STRESS, STRESS-RELATED DISEASES, AND COPING 248, 279 (1994).

50. Victims of PTSD show decreased volume of the hippocampus, an area of the brain responsible for memory and orientation in space. *See* J. Douglas Bremner et al., *Magnetic Resonance Imaging-Based Measurement of Hippocampal Volume in Posttraumatic Stress Disorder Related to Childhood Physical and Sexual Abuse—A Preliminary Report*, 41 BIOLOGICAL PSYCHIATRY 23, 24 (1997) [hereinafter Bremner et al., *Preliminary Report*]; J. Douglas Bremner et al., *MRI-Based Measurement of Hippocampal Volume in Patients with Combat-Related Posttraumatic Stress Disorder*, 152 AM. J. PSYCHIATRY 973, 977 (1995) [hereinafter Bremner et al., *MRI-Based Measurement*]; Tamara V. Gurvits et al., *Magnetic Resonance Imaging Study of Hippocampal Volume in Chronic, Combat-Related Posttraumatic Stress Disorder*, 40 BIOLOGICAL PSYCHIATRY, 1091, 1096 (1996). In addition, there is decreased metabolism in the frontal lobes, as mentioned, which may be related to a decrease in inhibitory-type receptors in the cerebral cortex in PTSD patients. *See* J. Douglas Bremmer et al., *Decreased Benzodiazepine Receptor Binding in Prefrontal Cortex in Combat-Related Posttraumatic Stress Disorder*, 157 AM. J. PSYCHIATRY 1120, 1123 (2000) [hereinafter Bremner et al., *Decreased BZ Receptor Binding*]. Moreover, these individuals face the likelihood of a number of disorders to which they become more

a course of treatment prescribed by a professional, which may include anxiolytic drugs. In addition, psychotherapy may be of immense benefit. Why? Because of the memories. We must learn to deal with the memories that we cannot escape. Rape victims who learn to deal with the incident, and succeed in re-experiencing the trauma less often, will be better able to avoid developing PTSD. Similarly for combat- or battering-induced PTSD. Despite the stigma attached to the traumatic event or to the disorder itself, victims need to seek help, and the sooner the better.[51]

7. *Dealing with the PTSD Patient*

It should be emphasized that victims of PTSD cannot be expected to react "normally" to such stressful conditions. These individuals will overreact to most stimuli, will fight or flee when pushed, and can "lose it" for no obvious reason other than a memory. These responses can be "illogical," "mindless," "insane," probably because critical judgment is decreased (perhaps due to decreased blood flow or "hypofrontality"). Even if such individuals can handle these proceedings without major external signs, delayed reactions to such stressors should be expected.[52]

Given this background, the well-informed individual will think long and hard about how best to deal with the PTSD patient. Descriptions of the traumatic event will be stressful, producing a stress response, perhaps exaggerated, along with active avoidance and agitation, even irritability. In gleaning such recollections, consideration should be paid to the distress being induced, because, after all, as the stress level increases, avoidance, distress, and hypervigilance will grow. Even

---

susceptible. These include shortened lifespan; increased risk for arterial, lower gastrointestinal, dermatologic, and musculoskeletal diseases; and decreased quality of life. Suicide attempts are 6% for people without such traumatic experiences, and 30% in trauma plus PTSD subjects; hospitalization is higher (48% versus 30%); major depression is higher (76% versus 53%); as well as alcohol abuse (38% versus 21%); and increases in the incidence, by 1.5 to 2 times, of circulatory, digestive, musculoskeletal, endocrine, nervous system, and nonsexually transmitted diseases are seen as long as twenty years after the trauma. Joseph A. Boscarino, *Diseases Among Men 20 Years After Exposure to Severe Stress: Implications for Clinical Research and Medical Care*, 61 PSYCHOSOMATIC MED. 605, 608-10 (1997); Meredith G. Warshaw, *Quality of Life and Dissociation in Anxiety Disorder Patients with Histories of Trauma or PTSD*, 150 AM. J. PSYCHIATRY 1512, 1514 (1993).

51. *See supra* note 50 and accompanying text.

52. An appropriate model for functioning under the influence of PTSD has been published as "The Road of Life." *See* Erica Beecher-Monas & Edgar Garcia-Rill, *The Law and the Brain: Judging Scientific Evidence of Intent*, 1 J. APP. PRAC. & PROCESS 243, 254 (1999).

hypofrontality may come into play, distorting critical judgement. A similar state can be induced during, for example, a deposition should questioning become particularly combative. Under such circumstances, an exaggerated fight-or-flight response may occur, the patient striking back or escaping, not to return. Obviously, a calm environment in the presence of supportive individuals is needed during such events, perhaps even the presence of a trusted counsellor/therapist to smooth out the bumps. Shortening the duration of such "stressors" should be considered, and could be tailored to the threshold of each patient. We must also be aware that anxiolytic agents may distort memory and cloud judgement, while lack of treatment may produce a hyperalert, over-questioning, over-sensitive individual. There may be a combative attitude or an uncommunicative one, in either case, great patience is required because these individuals are not in complete control of their emotions. Similarly, we must give these individuals the benefit of the doubt when overreactions ensue, particularly when they are placed in a stressful situation. When communicating with these individuals and their spouses, special care should be given to the presence of depression and suicidal ideation. It must be remembered that, to the depressed individual, suicide is a "logical" conclusion, in fact, the most logical end to the current turmoil. Such conclusions are, of course, being formed by a brain without the benefit of sufficient critical judgement. Psychiatric help should be sought at the mere mention of such "options."

The biological consequences of PTSD are considerable. For example, while forty to sixty percent of people exposed to such traumatic events will recover, the rest may carry the symptoms for many years.[53] Symptoms may develop with a delay of months, even years, but can last a lifetime.[54] The constant stress of PTSD can be considered as equivalent to accelerated aging of some brain areas.[55] PTSD is character-

53. These authors found that fifty-seven percent of their PTSD cases had a duration of more than one year. Breslau & Davis, *supra* note 27, at 672.

54. Symptoms can be delayed for years. *See generally* Van Dyke et al, *supra* note 42; Zlotnick et al., *supra* note 41, at 89. This recent study calculated that "the typical person with PTSD has a duration of active symptoms lasting more than two decades." Ronald C. Kessler, *Posttraumatic Stress Disorder: The Burden to the Individual and to Society*, 61 J. CLINICAL PSYCHIATRY 4, 7 (2000). Another study by the same author found that the median (50% of subjects) time for remission was thirty-six months for those who sought treatment, and over 30% of subjects had not recovered after ten years. Kessler et al., *supra* note 40, at 1057.

55. The degenerative effects of PTSD on the hippocampus have been proposed to be equivalent to accelerated aging. J. Douglas Bremner & Meena Narayan, *The Effects of Stress on Memory and the Hippocampus Throughout the Life Cycle: Implications for Childhood Development and Aging*, 10 DEV. & PSYCHOPATHOLOGY 871, 881 (1998).

ized by dysfunction or damage to the cortex, making our world less bright, inducing attention and logic problems.[56]

Damage to the hippocampus makes concentration and memory less effective.[57] The hyperarousal and sleep/wake problems have been attributed to dysregulation of the RAS.[58] All kinds of body systems are

---

56. There is decreased metabolism in the frontal lobes of PTSD subjects compared to controls when given an anxiety-inducing drug. The frontal lobes are involved in critical judgement, learning and memory. Bremner et al., *supra* note 38, at 246, 251. This finding suggests that, when a PTSD sufferer is exposed to a stressor, the function of the frontal lobes may be impaired to some extent, and so will critical judgement. On the other hand, patients with PTSD showed increased blood flow to limbic (emotional context) and parietal (spatial and sensory context) regions of the cortex. Neena Sachinvala et al., *Increased Regional Cerebral Perfusion by ^{99m}Tc Hexamethyl Propylene Amine Oxime Single Photon Emission Computed Tomography in Post-Traumatic Stress Disorder*, 165 MIL. MED. 473, 478 (2000). The interesting finding here is that every PTSD patient, not just the mean, showed abnormally high blood flow to emotional/sensory areas compared to normal subjects. There is recent evidence showing abnormalities in neuronal metabolism in limbic cortex of childhood PTSD patients. Michael D. De Bellis et al., *N-Acetylaspartate Concentration in the Anterior Cingulate of Maltreated Children and Adolescents with PTSD*, 157 AM. J. PSYCHIATRY 1175, 1176-77 (2000). Another study found activation of limbic and subcortical areas in PTSD patients subjected to trauma-related stimuli. Liberzon et al., *supra* note 39, at 822-25. There is a decrease in benzodiazepine receptors in the prefrontal cortex; these receptors mediate inhibition, so that control of this area is disturbed in PTSD. Bremner et al., *Decreased BZ Receptor Binding*, *supra* note 50, at 1123-24. Deficits in short-term memory are present in PTSD patients many years after trauma. J. Douglas Bremner et al., *Deficits in Short-Term Memory in Posttraumatic Stress Disorder*, 150 AM. J. PSYCHIATRY 1015, 1018 (1993). These deficits are not global, but rather affect primarily attentional mechanisms (which are decreased) and trauma-related memories (which are exaggerated). Wolfe & Schlesinger, *supra* note 21, at 210-11. In keeping with the selectiveness of trauma-related deficits in PTSD, there are significant increases in medial frontal lobe blood flow when PTSD patients are exposed to combat sounds compared to controls. Zubieta et al., *supra* note 39, at 262.

57. *See supra* note 50. A measure of neuronal density has found decreased density in the right temporal lobe of patients with PTSD. Thomas Freeman et al., *In Vivo Proton Magnetic Resonance Spectroscopy of the Medial Temporal Lobes of Subjects with Combat-Related Posttraumatic Stress Disorder*, 40 MAGNETIC RESONANCE MED. 66, 66 (1998).

58. Sleep disturbances are typical of PTSD, and include inability to fall asleep, inability to remain asleep, frequent awakenings, vivid nightmares, and the like. In terms of sleep studies these symptoms are reflected in increased REM sleep drive; decreased slow-wave, or non-REM sleep, and hypervigilance and hyperalertness during waking. Richard J. Ross et al., *Rapid Eye Movement Sleep Changes During the Adaptation Night in Combat Veterans with Posttraumatic Stress Disorder*, 45 BIOLOGICAL PSYCHIATRY 938, 938 (1999); Ross et al., *supra* note 26, at 700; Paul Sandor & Colin M. Shapiro, *Sleep Patterns in Depression and Anxiety: Theory and Pharmacological Effects*, 38 J. PSYCHOSOMATIC RES. 125, 125 (Supp. 1994); Steven H. Woodward et al., *Sleep and Depression in Combat-Related PTSD Inpatients*, 39 BIOLOGICAL PSYCHIATRY 182, 183 (1996). It should be noted that such sleep disturbances are indicative of dysfunction in a very stable, evolutionarily-conserved system, the RAS. That is, dysfunction/damage in this system is indicative of serious physiological problems that affect the metabolic

affected besides the brain. The wear and tear on the heart, immune system, digestion, etc., all contribute to a higher incidence of a large number of diseases in PTSD sufferers, and, of course, a shortened lifespan.[59]

---

balance of the brain. Prolonged, unresolved sleep-wake abnormalities lead to cognitive dysfunction, motor disturbances, and even psychotic events.

59. In a study fifty years after combat-induced PTSD, 59% of men who experienced heavy combat and PTSD symptoms were dead or chronically ill, whereas only 39% of combat exposed men without PTSD were chronically ill or dead. Kimberly A. Lee et al., *A 50-Year Prospective Study of the Psychological Sequelae of World War II Combat*, 152 AM. J. PSYCHIATRY 516, 520 (1995). People with PTSD may have at least a 20% greater chance of dying earlier than people their age, and since women live longer, this number may be higher for female survivors. PTSD was found to increase the risk for arterial, lower gastrointestinal, dermatologic, and musculoskeletal diseases. Paula P. Schnurr et al., *Physician-Diagnosed Medical Disorders in Relation to PTSD Symptoms in Older Male Military Veterans*, 19 HEALTH PSYCHOL. 91, 94 (2000). PTSD severely affects quality of life. *See supra* note 50 (listing statistics that suggest a higher incidence of various health problems in PTSD patients). People with PTSD will be 5 times more at risk for suicide, 1.5 times more likely to be hospitalized, 1.5 times more likely to develop major depression, and 2 times more likely to develop substance abuse than people their age. Alcohol abuse, whether as self-medication or as developed alcoholism, is increased by over 20% after PTSD. V. Gruden et al., *PTSD and Alcoholism*, 2 COLLECTIVE ANTHROPOLOGY 607, 608 (1999); McFarlane, *supra* note 44, at 814. The number of symptoms increases 29% per decade, regardless of combat or noncombat trauma experienced, and subjects with any type of trauma were more likely to show PTSD symptoms. Paula P. Schnurr et al., *Physical Symptom Trajectories Following Trauma Exposure: Longitudinal Findings from the Normative Aging Study*, 186 J. NERVOUS MENTAL DISEASE 522, 526 (1998). People with PTSD may have escalating effects with age. It should be noted that almost one half of patients with PTSD will develop major depression, and episodes of major depression predispose to suicide later in life. Up to 50% of patients who commit suicide suffer from major depression, 25% of patients with major depression attempt suicide in a lifetime, and 15% of patients with major depression die by suicide. Significantly, these attempts increase in frequency with the age of the patient. Kevin M. Malone et al., *Clinical Assessment Versus Research Methods in the Assessment of Suicidal Behavior*, 152 AM. J. PSYCHIATRY 1601, 1601-02 (1995). An added problem with major depression is that depression is a risk factor for coronary artery disease in men, making them twice as likely to develop coronary artery disease. Daniel E. Ford et al., *Depression Is a Risk Factor for Coronary Artery Disease in Men*, 158 ARCHIVES INTERNAL MED. 1422, 1423 (1998). Another study found that patients with depression had 4.5 times the risk of myocardial infarction. Laura A. Pratt et al., *Depression, Psychotropic Medication, and Risk of Myocardial Infarction*, 94 CIRCULATION 3123, 3126-27 (1996). There was a 25% greater risk of death from myocardial infarction in patients with depression. Sylvia Wassertheil-Smoller et al., *Change in Depression as a Precursor of Cardiovascular Events*, 156 ARCHIVES INTERNAL MED. 553, 557 (1996). Twenty years after severe stress, lifetime PTSD increases the incidence of a variety of health problems. *See supra* note 50. Twenty years after chronic, primarily combat-related, PTSD is also associated with clinically elevated leukocyte and total T-cell counts. Such measures are suggestive of disease in general medicine. These changes in leukocyte and T-cell physiology are implicated in autoimmune disorders, including diabetes, multiple sclerosis, atherosclerosis, and rheumatoid arthritis. Joseph A. Boscarino & Jeani Chang, *Higher Abnormal Leukocyte and Lymphocyte*

III. A FRAMEWORK FOR ADMISSION OF PTSD EVIDENCE IN CIVIL AND CRIMINAL CASES

Whatever the reason for proffering PTSD testimony, it must first pass the gatekeeping standards set out by the Supreme Court in a revolutionary trio of cases: *Daubert v. Merrell Dow Pharmaceuticals, Inc.*,[60] *General Electric Co. v. Joiner*,[61] and *Kumho Tire Co. v. Carmichael*.[62] This section lays out an analytical framework for making evidentiary decisions regarding PTSD. First, Part A demonstrates that PTSD satisfies the *Daubert* criteria. Next, Part B gives factual contexts in which PTSD satisfies the *Joiner* requirement of "fit" in both criminal and civil cases. The section concludes with an explanation in Part C of how social context evidence differs from PTSD evidence.

A. PTSD Evidence Meets *Daubert* Standards

Because of the *Daubert*, *Joiner*, and *Khumo Tire* cases, judges must now evaluate the scientific validity and the fit (or relevance) of expert testimony—including PTSD testimony—before it may be admitted into court. *Daubert* requires that the judge examine the proffered testimony for falsifiability, error rate and the existence of protocols, peer review and publication, and general acceptance.[63] *Joiner* insists that the testimony fit with the facts of the case, and *Kumho Tire* extends the validity inquiry to all types of expert testimony, including psychology.

Counts 20 Years After Exposure to Severe Stress: Research and Clinical Implications, 61 PSYCHOSOMATIC MED. 378, 383-84 (1999). That is, people with PTSD are at higher risk for developing diabetes, multiple sclerosis, atherosclerosis, and rheumatoid arthritis.

60. 509 U.S. 579 (1993).

61. 522 U.S. 136 (1997). In *Joiner*, the Court reiterated the abuse of discretion standard of review for evidentiary issues, and expanded upon the idea of "fit." *Id.* at 143. The Court explained that expert conclusions and the methodology used to reach them are not entirely separate and that one must examine both to determine relevance. *Id.* at 146.

62. 526 U.S. 137 (1999). The expert testimony involved in *Kumho Tire* was engineering testimony. *Id.* at 141. The Court ruled that the *Daubert* inquiry applies to all types of expert testimony. *Id.*

63. In *Daubert*, plaintiffs had suffered multiple birth defects, which they claimed were caused by their in utero exposure to Bendectin, an antinausea drug manufactured by the defendant. *Daubert*, 509 U.S. at 590. The trial court ruled that the proffered causation testimony was inadmissible, because it was incapable of meeting the general consensus standard. *See* Daubert v. Merrell Dow Pharms., Inc., 727 F. Supp. 570, 575-76 (S.D. Cal. 1989), *aff'd* 951 F.2d 1128 (9th Cir. 1991), *rev'd* 509 U.S. 579 (1993). The Supreme Court reversed and ruled that Federal Rule of Evidence 702 requires the trial judge to examine the validity of proffered expert testimony and whether the testimony "properly can be applied to the facts in issue." *Daubert*, 509 U.S. at 590.

These three cases have transformed the way courts must approach scientific evidence.

PTSD has been empirically tested, it has been subjected to critique for several decades, and PTSD studies have been published and peer reviewed. PTSD has been accepted as textbook science by the scientific community for twenty years.[64] Applying the *Daubert* factors, we have a falsifiable hypothesis and data that has been tested to support the theory. PTSD studies have been published in peer-reviewed journals and the diagnostic features are accepted in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV),[65] so it fits well within the scope of general consensus. As with any medical diagnosis, there may be variations in judgment, but the underlying studies have met statistical criteria for validity.

Syndrome testimony, on the other hand, has little basis in science.[66] There are so many syndromes out there in litigation land that one commentator calls such testimony the "abuse excuse" and lumps battered woman syndrome, rape trauma syndrome, urban rage, and the battered child syndrome into this category.[67] None of these syndromes are recognized by the DSM-IV. The empirical support for these separate disorders is shaky.[68] The error rate is high.[69] The survey evidence on which they are based is so fraught with errors it cannot possibly meet the *Daubert* standards.[70] There simply are no such syndromes. Rather, there is PTSD, and it may affect battered and traumatized people of all stripes: women, children, and combat veterans.

PTSD testimony, unlike syndrome testimony, meets all of the *Daubert* criteria. So why is this type of testimony controversial? Courts are poorly informed about the scientific basis for PTSD, and consequently handle such evidence poorly. As a result, syndrome testimony with its shaky basis is routinely admissible without even a nod toward

---

64. *See generally* DSM-IV, *supra* note 22, at 424-29.

65. *See id.*

66. *See* David L. Faigman et al., *The Battered Woman Syndrome and Other Psychological Effects of Domestic Violence Against Women, in* 1 MODERN SCIENTIFIC EVIDENCE: THE LAW AND SCIENCE OF EXPERT TESTIMONY § 8-1.0 (1997).

67. *See generally* ALAN M. DERSHOWITZ, THE ABUSE EXCUSE AND OTHER COP-OUTS, SOB STORIES AND EVASIONS OF RESPONSIBILITY (1994).

68. *See, e.g.*, Faigman et al., *supra* note 66, § 8-1.0 (arguing that battered woman syndrome "rests on less than sound foundations"); *see also* Robert P. Mosteller, *Syndromes and Politics in Criminal Trials and Evidence Law*, 46 DUKE L.J. 461, 487-90 (1996) (asserting that battered woman syndrome rests on shaky science).

69. *See* Faigman et al., *supra* note 66, § 8-2.1.2 .

70. *See id.*

Daubert,[71] while PTSD evidence is frequently excluded where it would be extremely helpful.[72] For example, battered woman syndrome (which argues for a cycle of violence in abusive relationships and learned helplessness that prevents escape) has been a particular focus of justified attack for its poor research methods.[73] Yet it is admissible as justification (self-defense) evidence in nearly every jurisdiction.

On the other hand, excuse evidence—that a defendant who participated in a crime was not responsible for her actions because of PTSD (or what many judges consider its equivalent, battered woman syndrome)—is routinely excluded. For example, a mother failing to seek medical help for her ailing or abused child is unlikely to have expert PTSD testimony admitted; similarly, an abused woman who carried her boyfriend's gun to a robbery because she was afraid of his abuse will commonly have her proffered PTSD testimony excluded. Courts overwhelmingly exclude such evidence, either as unscientific, or as irrelevant even though it is both relevant to the defendant's state of mind and scientific.

B. Cases Where PTSD Testimony "Fits"

So how should lawyers educate judges and how should judges respond to this information? What kind of cases are "right" for this evidence? What aspects of a case can PTSD evidence explain? PTSD evidence should come in as evidence whenever state of mind is an issue. Not only in criminal cases, where responsibility is an issue, but in any case, including civil cases, where state of mind is ordinarily relevant and admissible, or where damages may be at stake.

---

71. *See* Janet Parrish, *Trend Analysis: Expert Testimony on Battering and its Effects in Criminal Cases*, 11 WIS. WOMEN'S L.J. 75, 84 (1996) (noting that battered woman syndrome testimony is admitted routinely as self-defense testimony, but that only a small percentage of those cases require such testimony to meet the *Daubert* standards).

72. Sue Osthoff, *Preface to* Parrish, *supra* note 71, at 79-81 (noting that testimony about the defendant's abuse is routinely admissible to support self-defense, but rarely admissible to show the effects of abuse on the beliefs, perceptions and experience of the defendant).

73. *See* Robert F. Schopp et al., *Battered Woman Syndrome, Expert Testimony, and the Distinction Between Justification and Excuse*, 1994 U. ILL. L. REV. 45, 54-60 (detailing problems with the empirical data).

1. *Criminal Cases*

As self-defense testimony, PTSD testimony may be relevant in explaining why a woman reacted with lethal force to a seemingly innocuous triggering event: having been beaten many times following her spouse's quarrels with her, she may have responded to cross words with the first blow.[74] Or responded to physical blows with a gunshot. That is the issue of self-defense: was the defendant's behavior justified?[75] Most jurisdictions require that a defendant's belief in the necessity of her use of force be reasonable; that is, understandable to a person who sees what she sees and knows what she knows.[76] Although battered woman syndrome testimony is widely admissible to answer these issues, its shaky foundation indicates that it should be replaced with expert testimony for which there is more empirical support: PTSD evidence to explain the effects of trauma, and social science testimony to explain the circumstances in which the accused acted—the reasonableness of her conduct.

As excuse testimony, PTSD may be proffered to show mental incapacity or duress.[77] Even if the defendant's actions cannot be characterized as self-defense, PTSD evidence may be useful in showing

74. By the way, the overwhelming percentage of women who kill abusive partners do so during a confrontation, not as popular myth has it, while the spouse is asleep or unarmed. *See* Holly Maguigan, *Battered Women and Self Defense: Myths and Misconceptions in Current Reform Proposals*, 140 U. PA. L. REV. 379, 382-84 (1991).

75. Justification and excuse are the are the predominant bases for transforming an otherwise criminal act, such as the intentional shooting of one person by another, into a noncriminal event. *See* Greenawalt, *supra* note 5, at 1897-98.

76. *See* Schopp, *supra* note 73, at 98.

77. The distinction between justification and excuse is "between warranted action and unwarranted action for which the actor is not to blame." Greenawalt, *supra* note 5, at 1927 (arguing that the "criminal law should not attempt to distinguish between justification and excuse in a fully systematic way"). Under the Model Penal Code, the defense of duress is available if a person has been coerced to commit criminal conduct by force or threat of force that a reasonable person would have been unable to resist. MODEL PENAL CODE § 2.09(1) (1985). The "traditional defense of duress . . . covers some behavior that is justified and other behavior that is only excused." Greenawalt, *supra* note 5, at 1912.

lack of moral agency.[78] PTSD may come in as evidence for mitigating murder to manslaughter.[79]

In some instances, PTSD may make it impossible for the accused to appreciate the nature and quality or wrongfulness of her acts.[80] Yet, some courts refuse to admit PTSD testimony and insist that only battered woman syndrome testimony is admissible, even where the expert explains that PTSD—and not a syndrome—is responsible for the symptoms.[81] Thus, the use of PTSD testimony is controversial in meeting the insanity defense.[82] This controversy has little justification,

---

78. *See, e.g.*, Seidel v. Merkle, 146 F.3d 750 (9th Cir. 1998) (finding habeas petitioner was denied effective assistance of counsel because counsel failed to investigate the extent and ramifications of his client's PTSD); State v. Hunter, 695 N.E.2d 653, 657 (Mass. 1998) (finding PTSD testimony relevant to issue of responsibility but permitting prosecution expert to testify that the defendant did not exhibit the symptoms of PTSD); *cf.* Barrett v. State, 675 N.E.2d 1112, 1117 (Ind. Ct. App. 1997) (recognizing that "the presence of a self-defense issue is not the determinative factor in deciding the admissibility of battered woman syndrome and finding such testimony admissible to show lack of intent on the part of a mother accused of criminally neglecting her child, who was killed by the mother's batterer).

79. *See, e.g.*, *Seidel*, 146 F.3d at 750, 757 (granting habeas because trial counsel's failure to introduce defendant's PTSD diagnosis constituted ineffective assistance where, had such evidence been proffered, "the jury in all likelihood would have returned a verdict of manslaughter instead of murder"); Morgan v. Krenke, 72 F. Supp. 2d 980 (E.D. Wis. 1999) (granting petition for habeas relief because exclusion of PTSD testimony denied defendant her right to present a defense). In jurisdictions following the Model Penal Code, mitigation is permitted if a homicide is committed "under the influence of extreme mental or emotional disturbance for which there is reasonable explanation or excuse." MODEL PENAL CODE § 210(3)(1)(b) (1989).

80. 18 U.S.C. §17(a) provides an affirmative defense to federal prosecution when "the defendant, as a result of a severe mental disease or defect, was unable to appreciate the nature and quality or wrongfulness of his acts." 18 U.S.C. § 17(a) (1994). Many states provide similarly.

81. *See, e.g.*, Marley v. State, 729 N.E.2d 1011, 1014-15 (Ind. Ct. App. 2000) (finding PTSD testimony inadmissible unless presented under the aegis of battered woman syndrome testimony as an insanity defense to show that the accused suffered a dissociative state and was not conscious of what she was doing when she killed her abuser). Because the expert did not refer to battered woman syndrome and insisted that the reason the accused suffered from a dissociative state was that she suffered from PTSD as a result of abuse, the appellate court found the testimony deficient under the Indiana "effects of battery" statute. *Id.* at 1016.

82. *Compare* United States v. Rezaq, 918 F. Supp. 463 (D.D.C. 1996) (finding PTSD testimony relevant to the insanity defense under federal law) *with* United States v. Cartagena-Carrasquillo, 70 F.3d 706, 712 (1st Cir. 1995) (excluding PTSD testimony as irrelevant because the psychiatrist's report, while concluding that the defendant suffered from PTSD, did not conclude that as a result of his PTSD the defendant was "unable to appreciate the nature and quality or wrongfulness of the act") *and* United States v. Long Crow, 37 F.3d 1319, 1324 (8th Cir. 1994) (finding no insanity instruction was called for despite expert PTSD testimony because the expert whose testimony was admissible did not diagnose the accused, and the accused's diagnosis

however, as PTSD testimony ought to be freely admissible under such circumstances.

PTSD may also be relevant to duress.[83] Victims of domestic violence may be coerced into crimes against third parties, such as robbery and drug-related crimes, and in some of these, PTSD may be a factor.[84] Domestic violence, with its patterns of physical violence, coercion and control, may cause its victims to suffer PTSD, which may explain the behavior of a battered woman who commits crimes at the behest of her batterer.[85] Many courts—often the same courts that admit battered woman syndrome testimony to explain the reasonableness of an accused killer's conduct—find both battered woman syndrome testimony and PTSD testimony wholly irrelevant to coercion.[86] For example, battered women whose abusive partners have killed their children frequently claim duress as an excuse for not reporting the abuse to child protective agencies.[87] These claims, however, are rarely

---

was neither admitted nor was its exclusion appealed).

83. *See* United States v. Waddell, No. 93-3982, 1994 WL 279390, at * 9 (6th Cir. June 22, 1994).

> [T]o establish a prima facie case of duress, a defendant must make a showing that "there was an immediate threat of death or serious bodily injury to the defendant[,] . . . [she] had a reasonable fear that the threat of death or serious bodily injury would be carried out . . . [and that she] had no reasonable opportunity to escape the threatened harm.

Id. The courts' approach to battered woman syndrome testimony (or PTSD) in defense cases ranges from calling it irrelevant to relevant only as a mitigating factor, to admitting it as part of a complete defense.

84. *See, e.g.*, United States v. Brown, 891 F. Supp. 1501, 1508 (D. Kan. 1995) (finding battered woman syndrome testimony admissible to "assist the jury in determining [the accused's] state of mind and whether she acted as she did because [her abuser] threatened or abused her"). PTSD testimony would offer more scientifically sound evidence of state of mind. *See also* Kelly Grace Monacella, Comment, *Supporting a Defense of Duress: The Admissibility of Battered Woman Syndrome*, 70 TEMP. L. REV. 699 (1997) (chronicling the inconsistent results reached by courts when battered woman syndrome is proffered for duress rather than self-defense).

85. *See Waddell*, 1994 WL 279390, at *8-9 (affirming conviction of bank robber despite introduction of expert testimony that defendant suffered from PTSD and finding that although it was questionable whether the jury should have been given a duress instruction on the basis of her suffering PTSD, the instruction given was sufficient); Susan D. Appel, Note, *Beyond Self-Defense: The Use of Battered Woman Syndrome in Duress Defenses*, 1994 U. ILL. L. REV. 955, 969 (1994) (discussing the "murky" status of battered woman syndrome testimony in duress cases).

86. *See, e.g.*, State v. Riker, 869 P.2d 43 (Wash. 1994) (finding expert testimony about battered woman syndrome relevant only to explaining actions within a battering relationship and not to explain actions outside the relationship).

87. *See, e.g.*, Barrett v. State, 675 N.E.2d 1112, 1116 (Ind. Ct. App. 1996) (providing that evidence of battered woman syndrome was admissible in criminal neglect proceedings to determine the accused mother's mental state and intent when her

successful.[88] Because duress and self-defense both require proof of similar elements, it is illogical to permit PTSD testimony in one but not the other.[89]

Battered woman syndrome testimony is often used by the prosecution to explain inconsistencies in witness testimony,[90] and delays in reporting rape or domestic violence.[91] PTSD testimony, with its stronger empirical foundation, would be a better choice to explain these complex behaviors.

2. *Civil Cases*

PTSD testimony may be relevant and helpful not only in criminal cases, but in civil cases, too.[92] As damages in torts claims, PTSD

---

child died at the hands of her batterer); State v. Wyatt, 482 S.E.2d 147 (W. Va. 1996) (reversing woman's murder conviction for the death of her partner's son and remanding for new trial where trial court excluded testimony of battered woman syndrome as irrelevant in a case not involving self-defense, and ordering testimony about accused's state of mind be reconsidered in light of *Daubert*).

88. *See* Heather R. Skinazi, Comment, *Not Just a "Conjured Afterthought": Using Duress as a Defense for Battered Women Who "Fail to Protect"*, 85 CAL. L. REV. 993 (1997) (arguing that battered women charged with failing to protect their children are seldom afforded the defense of duress despite evidence of overwhelming fear of imminent death or serious bodily injury and absence of avenues for escape).

89. *See* People v. Romero, 13 Cal. Rptr. 2d 332 (Cal. Ct. App. 1992), *rev'd on other grounds*, 883 P.2d 388 (Cal. 1994) (extending the use of evidence about past abuse from self-defense to duress claims by finding counsel ineffective for failing to proffer battered woman syndrome testimony in a duress case).

90. *See* Arcoren v. United States, 929 F.2d 1235 (8th Cir. 1991) (allowing prosecution to use battered woman syndrome testimony to explain a rape victim's recanting her testimony). Although the court admitted expert testimony on battered woman syndrome to explain the witness's diametrically opposed sworn statements, PTSD evidence would have been more scientific and would have achieved the same goal. The Eighth Circuit found the syndrome testimony admissible to explain "mental aberrations in human behavior, when such knowledge will help the jury to understand relevant issues in the case." *Id.* at 1240.

91. *See, e.g.*, United States v. Gowen, 32 F.3d 1466 (10th Cir. 1994) (admitting PTSD testimony to explain rape victim's actions); State v. Grecinger, 569 N.W.2d 189, 195 (Minn. 1997) (permitting prosecution to use battered woman syndrome testimony to explain victim's delay in reporting domestic violence, her conflicting testimony, and why she returned to a relationship with her batterer as "necessary to explain the complexity of [the victim's] behavior"); People v. Yates, 637 N.Y.S.2d 625 (N.Y. Sup. Ct. 1995) (admitting testimony to explain delay in reporting homosexual rape); State v. Martens, 629 N.E.2d 462 (Ohio App. 1993) (allowing admission of PTSD testimony admissible to explain delay in reporting rape).

92. Richard L. Newman & Rachel Yehuda, *PTSD in Civil Litigation: Recent Scientific and Legal Development*, 37 JURIMETRICS J. 257, 259 (1997) (observing that "PTSD can explain the psychological consequences of a trauma and legitimatize the demand for compensation for these injuries").

evidence has been widely used in airline disasters,[93] medical malpractice,[94] and workers' compensation claims, among others. Only slowly are the courts beginning to recognize, however, that PTSD is a physical injury.[95] But the evidence of physical changes as a result of the disease is strong, and the torts courts will undoubtedly follow the science.[96]

Sometimes PTSD claims have been used in troubling ways. For example, there appears to be a growing use of PTSD testimony against battered women in custody cases. This is a particularly disturbing trend because custody awards already penalize victims of domestic violence.[97] Courts also appear unwilling to permit PTSD to excuse delay in filing claims outside the limited purview of criminal rape, child sexual abuse, and domestic violence charges. That is, PTSD is seldom permitted to explain a delay in coming forward with civil claims pertaining to the same matters.[98]

---

93. *See, e.g.*, Ben-Shimol v. TWA, No. C-93-1586 MHP, 1996 WL 724782 (N.D. Cal. Nov. 22, 1996) (finding that not only was PTSD testimony admissible in determination of damages from plane crash, but defense testimony was also admissible to show that plaintiff had suffered from PTSD prior to being injured in plane crash); Weaver v. Delta Airlines, 56 F. Supp. 2d 1190 (D. Mont. 1999) (recognizing PTSD that resulted from emergency landing as "bodily injury" for purposes of Warsaw Convention). *But see* Alvarez v. Am. Airlines, No. 98 Civ. 1027 (MBM), 2000 WL 145746, at *1 (S.D.N.Y. Feb. 8, 2000) (dismissing claim for PTSD damages as psychological or emotional injury which must be proximately caused by physical injuries).

94. *See, e.g.*, Luecke v. Bitterman, 658 N.Y.S.2d 34 (N.Y. App. Div. 1997) (holding that damages for medical malpractice included two and one-half years of PTSD).

95. PTSD is accompanied by demonstrable physical changes in the neurochemical, endocrine, and physiological makeup of the brain. Roger K. Pitman et al., *Psychophysiologic Assessment of Posttraumatic Stress Disorder Imagery in Vietnam Combat Veterans*, 44 ARCHIVES GEN. PSYCHIATRY 970, 973 (1987); Rachel Yehuda et al., *Dose-Response Changes in Plasma Cortisol and Lymphocyte Glucocorticoid Receptors Following Dexamethasone Administration in Combat Veterans With and Without Posttraumatic Stress Disorder*, 52 ARCHIVES GEN. PSYCHIATRY 583, 583 (1987). Although these physical changes cannot yet be used for diagnosis, they have been repeatedly correlated with PTSD. *See* Newman & Yehuda, *supra* note 92, at 265 (suggesting that "the presence of biological changes provides corroborative evidence that PTSD is present").

96. *See, e.g.*, Newman & Yehuda, *supra* note 92, at 266 (suggesting that courts will be better able to evaluate PTSD claims post-*Daubert* and post-DSM-IV).

97. *See* Jane H. Aiken & Jane C. Murphy, *Evidence Issues in Domestic Violence Civil Cases*, 34 FAM. L. Q. 43, 51 (2000) (noting that "approximately 70 percent of contested custody cases that involve a history of domestic violence result in an award of sole or joint custody to the abuser") (citing DOMESTIC VIOLENCE AND THE COURTROOM: UNDERSTANDING THE PROBLEM, KNOWING THE PROBLEM (1995)).

98. *See* Florez v. Sargeant, 917 P.2d 250, 255 (Ariz. 1996) (refusing to toll the statute of limitations for claims of child sexual abuse based on PTSD claims and noting that "no state has found that a diagnosis of posttraumatic stress disorder alone, is sufficient to constitute insanity or unsound mind within the meaning of the relevant

Too often, moreover, lawyers are uninformed about PTSD and its effects. Numerous cases find that counsel rendered ineffective assistance by failing to investigate the extent or ramifications of the accused's impairment.[99] Educating lawyers, judges, and juries about the ramifications of severe trauma is vitally important in a significant number of cases. PTSD, which has solid empirical support, can and should pass *Daubert* muster. Syndrome testimony, on the other hand, should be relegated to the dustbin of unsupported speculation.[100]

C. Social Context Evidence Distinguished

Although syndrome testimony has little scientific merit as psychological testimony, the impetus for its widespread acceptability is the insight that the social context of a person's actions is crucial to assessing the reasonableness of her conduct. It is important to distinguish between two kinds of evidence here. Explaining why a defendant reasonably believed she was in danger of death or great bodily harm does not necessarily require testimony about PTSD. Instead, it may require expert testimony about the social context, including the woman's history of being abused. Social context evidence—if there is empirical support for it—should be admissible, but it should not be lumped together with state of mind testimony under the guise of battered woman syndrome.

For example, many juries and judges have difficulty believing a woman's claims of abuse, because they fail to understand why, if she was abused, she didn't just leave. The inference is that she must be exaggerating the claims of abuse in retrospect because no reasonable

---

[tolling] statute").

99. *See, e.g.*, Seidel v. Merkle, 146 F.3d 750 (9th Cir. 1998) (finding ineffective assistance where attorney failed to investigate client's mental condition).

100. Indeed, some courts are already recognizing the importance of PTSD evidence in self-defense claims. *See, e.g.*, State v. Hines, 696 A.2d 780 (N.J. Super. Ct. App. Div. 1997) (reversing conviction for failure to admit expert testimony on PTSD). In *Hines*, the accused had killed her father and claimed that she acted in self-defense because her father had sexually abused her since she was nine and was again attempting to rape her at the time of the incident. *Id.* at 781. PTSD evidence had been proffered to explain the accused's failure to tell anyone about her childhood abuse and to explain why the accused believed her elderly father was about to rape her. *Id.* The trial court excluded the testimony because it found the jury competent to evaluate reasonableness without expert assistance. *Id.* at 783, 785. The appellate court reversed, finding that the expert testimony would have "lent additional credibility to defendant's allegations regarding the victim's past sexual abuse and would have been probative of the honesty and reasonableness of her belief that she had to resort to deadly force to prevent him from raping her again." *Id.* at 788.

person would subject themselves to repeated violence. In addition, self-defense (and duress) requires the defendant to make use of available legal protection (why did she stay in an abusive relationship; why did she fail to call the police before resorting to violence?). The answers to these questions may have much more to do with the social realities of her alternatives—which also must be demonstrated empirically—as it has to do with any impairment to her cognitive functions from the trauma.

In other words, for the jury to evaluate the reasonableness of the defendant's actions, some chilling facts may need to be brought to the court's attention in a battered spouse case: although domestic violence constitutes the largest category of calls police receive, police are notoriously reluctant to respond to such calls, and when they do, only ten to eighteen of the abusers are arrested, despite grounds for arrest in fifty percent of the cases, and often despite mandatory arrest requirements.[101] Arrest of the abuser frequently results in increased violence toward the woman.[102] Shelters are often either unavailable or unsafe.[103] Police, district attorneys, and judges all frequently attempt to dissuade victims from proceeding with criminal charges.[104] Judges often blame the victim, presuming provocation, and often fail to believe women without visible injuries.[105] Moreover, women who kill a spouse are much more likely to be charged with murder than men who kill their wives—who are usually charged with manslaughter.[106] Furthermore, women's charges are much less likely to be reduced (eighteen versus forty-seven percent).[107] Batterers are rarely charged with felonies, and sentences tend to be lenient.[108] In other words, she needn't be suffering from PTSD, and may have been quite rational about staying in light of

101. *See* Parrish, *supra* note 71, at 128. Perhaps because of this reluctance on the part of the police to respond to calls involving domestic violence, and the paucity of arrests, studies estimate that only ten percent of domestic violence incidents are ever reported to the police. *See* Mary Ann Dutton, *Understanding Women's Responses to Domestic Violence: A Redefinition of Battered Woman Syndrome*, 21 HOFSTRA L. REV. 1191, 1213 (1993).

102. Parrish, *supra* note 71 at 128.

103. *See* Gretchen P. Mullins, *The Battered Woman and Homelessness*, 3 J.L. & POL'Y 237, 249-50 (1994).

104. *See id.*

105. *See* Parrish, *supra* note 71; *see also* Lynn H. Schafran, *There's No Accounting for Judges*, 58 ALB. L. REV. 1063, 1063-67 (1995) (describing attitudes of judges toward male domestic violence towards women).

106. *See* Parrish, *supra* note 71, at 141.

107. *See id.*

108. *See id.*

the high percentage of women who are murdered by their former partners after they leave abusive relationships, economic pressures, and the ineffectiveness of police intervention. This kind of evidence may require an expert to testify about the social context, but it will not require testimony of PTSD.

Similarly, in explaining why a rape was not reported promptly, social context evidence may have far more relevance than evidence of PTSD. The number of rape cases that are dropped is shockingly high, and few rapists are convicted. Overbearing, hostile police and vicious attacks on the victim's character at trial are the norm (coming in despite rape shield laws as motive for a false accusation or as context for the defendant's consent defense). Empirically—and for good reason—a certain percentage of rape victims may delay reporting the crime, even without suffering from PTSD. Exposing yourself and your actions to often brutal mental scrutiny after having been brutalized physically is not a pleasant prospect, and reluctance to report has a rational basis. An expert should be able to testify to the social context evidence (and the underlying studies will have to meet *Daubert* muster as sociological evidence) without any mention of PTSD. On the other hand, evidence that a woman is suffering from PTSD as a result of the trauma of abuse may indeed be relevant to her conduct, explaining why she reacted to a particular situation the way she did (the increased fight-or-flight response helping to explain why she used more force than appears necessary, for example, or the re-experiencing phenomenon to explain why she killed during a lull in the fighting), and may go far in explaining the way she appears to jurors and judges in court (the lack of affect is related to "hypofrontality," the decreased blood flow to the frontal lobes). Not all abused killers suffer from PTSD, but if the rape statistics (showing over one half of rape victims suffering from it) are anywhere close for battered women, a large percentage of them do. PTSD testimony should be available to them to explain their state of mind. In addition, a rape victim, who may have good reason to avoid reporting the rape because of the social context, may also be suffering from PTSD, which makes her avoidance behavior even more acute. In that case, the PTSD evidence also should be admissible.

IV. CONCLUSION

In sum, PTSD testimony is based on solid science and should be admissible under *Daubert*. Whether the case is criminal or civil, PTSD testimony should meet the fit requirements whenever mental state is at

issue. Syndrome evidence—whether battered woman, battered child, or urban rage—is not empirically supported outside of the PTSD dimension. It should be excluded because it cannot meet the *Daubert* standards. When mental state is not at issue, but social context is, then the testimony must also meet *Daubert* standards with respect to its empirical basis. A great deal of information that would be helpful to juries and judges is routinely excluded and some that is misleading is admitted because lawyers and judges fail to examine its scientific basis. They should, and under *Daubert*, they must.