Source: NATIONAL JUDICIAL EDUCATION PROGRAM, UNDERSTANDING SEXUAL VIOLENCE: PROSECUTING ADULT RAPE AND SEXUAL ASSAULT CASES (2000).

The Neurobiology of Trauma[1]

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The human brain is the product of millions of years of evolution. Much of its anatomy and physiology is the legacy of our common ancestry with the other animals, and even with the earlier life forms from which mammals evolved. One very significant legacy of this evolutionary heritage is a highly developed capacity to experience fear.

Although 21st century humans look very much like the planet's number one predator, the vast majority of our time on earth has been spent as a prey species: mid-sized meals for true predators, such as lions, leopards and saber-tooth tigers. Our survival depended on our capacity to react instantaneously to the threat of attack from these formidable animals. When a leopard makes its lunge, life or death may be decided in a millisecond.

Successful adaptation demanded the evolution of a system of hormones and neurochemicals capable of instantaneously altering the body's functioning, preparing it for life-saving flight or savage fight, and for the capacity to freeze completely until either flight or fight was chosen. This cascade of chemicals is the biological equivalent of setting off an atomic bomb inside the body. Within a fraction of a second, heart rate, blood pressure, breathing rate, blood distribution and pupil dilation are all fundamentally altered.

This "fight or flight" response is triggered without any conscious intervention. Conscious thought, although subjectively fast, actually is far too slow to beat the leopard. Evolution demanded a system that by-passes the cortex – the brain's center of higher and integrative functioning. It demanded a system that is wired directly into the amygdala, the brain's "fear center." So, when we see a threatening stimulus, like a gun for example, our amygdala has received the information and triggered the fight or flight response long before we say to ourselves, "there's a gun!"

Not only can we react without the intervention of our cortex, we can also store a great deal of information without it. In fact, once again, it has been crucial to our survival that we be capable of encoding in memory particular stimuli, the recognition of which might determine life or death, and to do so without cortical involvement. Imagine a zebra bending down to drink at a water hole, every sense keenly alert for danger. Suddenly, from out of the grass nearby, a blur of brown hide lunges out. The zebra instantly spins, lunges and gallops away, alive for another hour. But thirst demands that it return to the water hole. When it does, the particular stimuli that preceded the attack will now have been etched into memory – not at the cortical level — but rather at far simpler, sub-cortical levels. At the zebra's second visit to the water hole, should a gust of wind happen to sway the grass and cause a similar sound to that of the lion's lunge, the poor animal will flee with the same experience of terror as before. If zebras think, it may say to itself, "that was just the wind," but it will be 50 yards away when it does so.

Being creatures with complex brains, humans actually have multiple pathways to the experience of fear. The amygdala route, the "low road" to fear, is the fastest. However, we may also perceive something in the environment that is not instantly recognizable as a threat. In such a case we rely on the "high road" to fear, the route that takes the information into the cortical regions of the brain. There, more thorough analysis of the stimulus is possible, and we can make a more deliberative determination of the nature of the threat.

The human capacity to experience fear, so crucial to our species' survival, is also the cornerstone of our capacity to become traumatized. A human being who has not been traumatized is capable of using both the "low" and "high" roads to fear. A moving car that suddenly appears in the corner of one's eye will activate the low road and allow us to jump back onto the curb before being struck. The sight of a lion at the zoo will activate the high road, allowing us to recognize that although its canines would surely rip us to shreds, we are safely beyond its reach. The sound of a loud bang may well activate both low and high roads, giving us a sudden start until we identify the sound as the backfire of a car engine. A human being who has been traumatized, however, will have far less flexibility in the activation of their fear system. For the traumatized human, the low road to fear predominates.

Human beings who have been raped will, just like the zebra, carry with them a network of neurons forever prepared to respond to the perception of any of the cues that were present during the rape. It might be the sound of a man's voice; the feel of hands on a particular part of the body; or the look of anger in another's eyes. The possibilities are literally infinite. At the sound, touch or sight of those cues, the rape victim will experience the same cascade of neurochemicals that were triggered during the actual rape. Their heart will begin racing, their blood pressure will spike, their breathing will accelerate. They may find themselves fleeing in terror from a supermarket because someone unexpectedly touched them on the arm. They may find themselves frozen in terror because a man said something hostile to them in a parking lot. Their reactions are not conscious choices, not "hysterical" over-reactions, any more than is the zebra's flight from the sound of windblown grass. Both are reactions governed not by the cortex, not by conscious thought, but rather by the "low road" of amygdala-based fear networks.

Traumatic Memory

The memory of a traumatic experience is not encoded in the same way as is a normal experience. The powerful neurochemicals that trigger the fight or flight response have far-reaching effects, including dramatic effects on the manner in which memories are encoded. Often, a traumatized person cannot generate the kind of narrative memory that we can normally muster for an important experience. Their memories are often fragmented, out of sequence, and filled with gaps. They may recall very specific details for particular aspects of the experience, and recall little or nothing for others. It is for this reason – the neurobiology of traumatic memory – that great care must be taken in interviewing trauma survivors. The fact that a traumatized person recalls a detail which

they earlier had not is not prima facie evidence of fabrication; it is the characteristic way in which these types of memories are stored and recalled. The fact that they can recall the texture of the rapist's shirt, but cannot recall whether he was wearing a hat, is not evidence that something is being hidden; it is a product of how the brain encodes information during a trauma.

Once again, these characteristics of traumatic memory are not the consequence of conscious choice or resistance. Rather, they are the consequence of the radically altered neurochemical environment in which the memories were encoded.

To summarize, the rape victim, like any traumatized human, is left with a permanently altered brain. As part of its legacy, trauma leaves its victims with fear networks etched into the amygdala, networks that can be triggered by a multitude of cues that would ordinarily not evoke fear. Trauma also leaves its victims with fragmented and discontinuous memories of what happened to them. As a consequence of these legacies, the rape victim faces enormous challenges in the judicial process. To participate in that process – to endlessly recount their trauma, to appear in the court room where the rapist sits – is equivalent to the zebra consciously choosing to return to the water hole where the lion attacked. In both cases, a confrontation with the biological legacy of trauma is inevitable. The zebra only does so out of absolute necessity; the rape victim's choice must be more conscious, and more deliberate.

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[1] For further information about the neurobiology of fear, see Joseph Ledoux, *The Emotional Brain*, 1996, New York, Simon & Shuster. For detailed information about the neurobiology of trauma, see Rachel Yehuda and Alexander C. McFarlane, *Psychobiology of Posttraumatic Stress Disorder*, 1997, New York, Annals of the New York Academy of Sciences, Volume 821.