

# [The neurobiological impacts of trauma](https://assignbuster.com/the-neurobiological-impacts-of-trauma/)

No human in this world is or has ever been immune to feeling stress and anxiety. They are necessary evils that are an unfortunate, inherent thread of fabric that are woven into daily life of societies around the world. For aggressive cultures that can be found in countries such as the United States, stress is essentially an expected component almost necessary to attain success and prestige. To breakdown under pressure brings shame; to admit to struggling against anxiety invites side glances and external perceptions of weakness. Yet, to dismiss any level of angst and tension is a grave mistake as it can have a profound and lasting impact on person’s quality of life, especially when the repercussions have the capacity to alter one’s neurological functioning indefinitely. The impacts of trauma have the capacity to forever alter the way one’s brain operates. A devastating divorce, rape, witnessing a loved one die, dealing with a soul-sucking boss, being a recipient of gaslighting, getting incessantly bullied — these are all examples that can potentially hijack the way a person’s brain functions. Whether accepted or not, traumatic and stressful experiences can seep into someone’s upper story and throw it out of whack on an abysmal level. This paper will demonstrate just how much a trauma can spin a person’s neurobiological hormones, chemicals, and the fundamental nuts and bolts of their core mentality into a twister of disarray.

Experiences that result in psychological trauma are essentially ones in which an individual feels as if his or her life and well-being are in danger. The aftermath of these experiences often involves a slew of negative and chronic emotions such as intense fear, helplessness, and anxiety. Given the magnitude and impact of the experience, individuals can go on to develop post-traumatic stress disorder (PTSD), a debilitating disorder characterized by characteristics and symptoms that can be grouped into three main domains: reminders of exposure, activation, and deactivation. Reminders of exposure can include flashbacks, intrusive thoughts, and nightmares. In activation, an PTSD victim may experience hyperarousal, insomnia, agitation, irritability, impulsivity, and anger. On the flip side, deactivation involves numbing, avoidance, withdrawal, confusion, derealization, dissociation, and depression. A PTSD diagnosis must include certain symptoms that are intense and extreme enough that an individual’s social, professional, and interpersonal spheres of life are debilitated for one month or longer.

More importantly, these chronic and prolonged symptoms clearly show that experiencing severe trauma is indicative of pervasive, abnormal alterations of an individual’s neurobiological systems. Simply put, serious and persistent psychological trauma alters the way one’s brain functions in a profound and irreversible way as it meddles with various neural control centers that regulate stress responses. This includes endocrine and neurotransmitter pathways and brain regions such as the brain stem, limbic system, and cortex — all of which are essential in regulating fear responses at both the conscious and unconscious levels.

There has been much research that confirms the neuroendocrine system is impacted by traumatic experiences. There has been consistent evidence that indicates that features of PTSD include a regulation impairment of cortisol and thyroid hormones. The main component of the endocrine system is the hypothalamic-pituitary-adrenal (HPA) axis, which is the central coordinator of the neuroendocrine stress response system that influences and interacts with the hypothalamus, pituitary, and adrenal cortex. One of its primary functions is in regulating responses to stress through the release of glucocorticoids, or human stress hormones. The glucocorticoid cortisol is particularly important as it directly impacts the metabolism, immunity, and norepinephrine releases from the adrenal medulla. The result is an extensive and direct repercussion on numerous essential brain functions such as memory.

Upon experiencing a threatening situation, the hypothalamus is triggered to release chemicals such as the corticotropin releasing factor (CRF), which then activates the pituitary gland to release the adrenocorticotropic hormone (ACTH). This results in glucocorticoids, namely cortisol, to be released from the adrenal gland as means to process and cease the stress response. However, chronic and severe stress has been shown to over-activate the HPA axis and result in inadequate cortisol levels. As Sherin and Nemeroff (2011) pointed out, the “ decreased availability of cortisol, as a result of or in combination with abnormal regulation of the HPA axis, may promote abnormal stress reactivity and perhaps fear processing in general” (p. 267). A lack of cortisol can also have detrimental impacts on learning, memory, and other cognitive functions — all of which have been shown to suffer impairment in individuals with PTSD.

Additionally, anxiety and other similar behaviors have been linked to the over-activation of CRH-containing neurons in the amygdala. In fact, there have been several animal studies that have shown that increased CRH activity can promote certain features of PTSD such as the following: learned fear responses; increased startle reactivity; hypersensitivity to stressors; and hyperarousal. It is also important to note that a lack in neuropeptide Y (NPY), which has stress-buffering properties, may promote maladaptive stress responses and contribute to the development of PTSD.

A dysregulated HPA axis can also result in increases and decreases in sympathetic and parasympathetic tone and the release of ACTH, respectively. It can also prompt increases in catecholamines from the pituitary, adrenal cortex, and adrenal medulla. The catecholamine family of neurotransmitters includes dopamine and norepinephrine — both which play important roles in fear conditioning and mediating autonomic stress responses. Increases in catecholamines are linked to a hallmark characteristic of PTSD: prolonged hyperactivity of the autonomic sympathetic part of the autonomic nervous system. This has been shown through psychophysiological symptoms such as heightened heart rate, blood pressure, and skin conductance. Research has consistently shown that patients with PTSD feature increased urinary excretion of catecholamines as well as increased heart rate, blood pressure, and norepinephrine responses to traumatic reminders. This is indicative that the catecholamine norepinephrine is directly linked to traditional aspects of PTSD symptoms.

In addition to peptides and catecholamines, other neurochemical factors such as serotonin and amino acids also play significant roles in stress responses and are altered when faced with the impact of trauma. Serotonin helps to regulate sleep, appetite, sexual behavior, aggression, motor function, and neuroendocrine functions. It also works with the endocrine system and norepinephrine to coordinate stress responses. When impaired, serotonin transmission can contribute to symptoms of PTSD including hypervigilance, increased startle, impulsivity, and intrusive memories.

In the realm of amino acids, there are two primary neurotransmitters that are linked to stress response mechanisms: GABA and glutamate. GABA is the main inhibitory transmitter that helps to regulate the expression and extinction of fear conditioning. Fear conditioning caused by trauma can cause a reduction in GABA signaling in the amygdala, hippocampus, and prefrontal cortex regions resulting in the impairment of key conditioned fear mechanisms. Indeed, as Mahan and Ressler (2012) pointed out, there is evidence that “ GABAergic inhibitory microcircuits might regulate acquisition and expression of fear memories in the central nucleus of the amygdala” (p. 29).

Meanwhile, glutamate is the primary excitatory neurotransmitter in the brain and is essential for fear extinction. Glutamate is activated by stressors and the release of glucocorticoids mentioned earlier. It also binds to the receptors in the NMDA receptor system, which has been linked to learning and memory and is considered to contribute to trauma memories in PTSD (Sherin & Nemeroff, 2011). An overexposure to glutamate has been shown to damage neurons and is thought to lead to the reduction of neurons in the hippocampus and prefrontal cortex of those who suffer from PTSD. Furthermore, overexposure to glutamate induced by trauma can result in the loss of metabotropic receptors in the limbic system. This has been shown to impair both consolidation and extinction of fear conditioning. Concurrently, the activation of these receptors in the amygdala has been shown to enhance fear learning (Mahan & Ressler, 2012).

The HPA axis, neurotransmitters, peptides, and key stress hormones are also heavily intertwined with primary brain regions, highlighting the complexity of the cascade of trauma-induced effects. Functional brain imaging studies have consistently confirmed that the brainstem, limbic system, and cortex are altered in individuals who suffer from chronic stress or PTSD. Specifically, much evidence has shown that trauma creates significant changes in the brainstem, hippocampus, amygdala, and prefrontal cortex, which are tantamount to the adaptation to stress and fear conditioning.

The brainstem helps shape the energy levels the limbic and cortical regions by directly controlling states of arousal and responses to threats. More specifically, the brainstem works in tandem with the limbic and the higher cortical regions and serves as the arbiter of whether a person responds to threat by fighting, fighting, or freezing. As Siegel (2013) put it, feeling a deep drive to respond in a certain way indicates that the brainstem is collaborating with the limbic area to prompt action.

The limbic system, which drives basic needs and emotions, is arguably one of the most important cogs in the stress response system. Traumatic experiences can chronically spike cortisol levels and sensitize limbic reactions, which can interfere with the proper growth and function of neural tissue. The limbic system is comprised of two main components: the hippocampus and amygdala. Together with the cortex regions, these areas are most clearly altered in PTSD (Mahan & Ressler, 2012).

The hippocampus is linked to the control of stress responses, explicit memory processes, and contextual aspects of fear conditioning. Those who suffer from PTSD have consistently been shown to have reduced hippocampal volume, which can result in a variety of adverse effects such as: activation of and failure to terminate stress responses; impaired extinction of conditioned fear; deficits in discriminating between threatening and non-threatening stimuli; verbal memory deficits; dissociative symptom severity, depression severity; and PTSD symptom severity. Additionally, hippocampal deficits are linked to the inability to distinguish between safe and dangerous situations. Yehuda and LeDoux (2007) explained that this could explain why patients with PTSD have exaggerated responses to trauma-related triggers (Yehuda & LeDoux, 2007). Furthermore, reduced hippocampal volume’s negative impact on cognitive capacity is likely to make it “ more difficult for persons to contextualize and reinterpret the experience of trauma in a way that can facilitate recovery” (Yehuda & LeDoux, 2007, p. 21).

The amygdala, on the other hand, is like the brain’s fear response master system that plays a heavy hand in emotional processing and fear responses. Functional brain imaging studies have revealed that chronic, traumatic experiences can put the amygdala into overdrive when confronted with both stressful and neutral stimuli. According to Yehuda and LeDoux (2007), threat processing by the amygdala is the key step in the circuitry through which catecholamines, ACTH, and cortisol are released into the circulation. When trauma-induced damage occurs to the amygdala, fear conditioning is prevented and exposure to fear stimuli can result in a hyperactive amygdala that goes off for a prolonged period even when a person is in a situation he or she once considered safe. In other words, those with a hyperactive amygdala “ may be more likely to process neutral, unconscious, or implicit threats, which would serve to even further weaken the ability of the prefrontal cortex to regulate these responses” (Yehuda & LeDoux, 2011, p. 22).

It is important to refer to the low levels of cortisol and other glucocorticoids seen in patients with PTSD mentioned earlier. Glucocorticoid activity in the amygdala generally promotes cognitive functions such as arousal, attention, and memory formation — all of which are pertinent in allowing a person to survive in threatening situations. Keeping this mind, reduced exposure to glucocorticoids in the amygdala could potentially explain how a person with PTSD is unable to adapt and process trauma. Also noteworthy is the fact that the combination of a chronic, hyperactive amygdala and hippocampal deficits are linked to not only PTSD, but other anxiety disorders such as panic, specific phobia, and generalized anxiety disorder. This implies that “ enhanced activation of the amygdala in response to provocation may be a general consequence of experiencing fear or anxiety regardless of whether the anxiety is anticipatory, based on a real threat, or the product of a disorder” (Yehuda & LeDoux, 2007, p. 23).

There is a lot of interplay between the amygdala and the prefrontal cortex. The prefrontal cortex’s link to the amygdala prompts it to exert inhibitory control over stress responses and emotional reactivity. Traumatic experiences can cause prefrontal cortex deficits that hinder fear extinction and prolong fear responses. In their primate study, Shin, Rauch, and Pitman (2006) concluded that extinction does not occur normally when the prefrontal cortex is damaged. Their findings suggested that PTSD victims often have damaged prefrontal cortexes, prompting them to “ exhibit persistent inappropriate fear responses in daily life and diminished extinction of conditioned fear responses” (p. 68). It is important to note that deficits of the anterior cingulate cortex (ACC) component of the prefrontal cortex is also implicated in PTSD symptoms. For example, findings have shown that traumatic experiences have been linked to a reduction in ACC volume, which in turn has been correlated with PTSD symptom severity. Taken altogether, there is an abundance of research and evidence that suggests reduced cortical volumes and prefrontal cortex deficits are linked to hyperactive amygdala reactions and hippocampal volume deficits — a roster that is consistently found in those with PTSD.

Advancements in technology and progressive theories have paved the way to demonstrating the importance of the neuroscience perspective when addressing PTSD and other trauma-related disorders. Brain imaging studies and literature reviews alike have revealed key links and associations between traumatic experiences and their impacts on the brain’s structure, chemicals, and functions. This growing amount of data maps out pathways and ideas for future studies that may lead to more effective treatments as well as prevention methods. For example, future studies may reveal certain biomarkers that might help determine and prevent at-risk individuals from developing PTSD. Furthermore, a better understanding how trauma affects the neural chemicals and hormones may lead to more effective pharmaceutical treatments. It is certainly possible that a neurobiological approach to trauma may be the key that unlocks groundbreaking solutions that have the potential to improve a multitude of lives that would otherwise be victim to life-long fear and anxiety.

## References

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