An introduction on the neuroscience behind the trauma

Julia Sánchez Sáez [a]

[a] Faculty of Medicine, União das Faculdades dos Grandes Lagos (UNILAGO), São José do Rio Preto, Brazil, julia.s.saez@gmail.com.

Abstract. Trauma profoundly impacts all aspects of behavioral and psychological functioning, causing physiological reactions and alterations in emotions, perceptions, and beliefs regarding the traumatic experience. Disturbing experiences, such as witnessing a brutal death, not only trigger intense emotional responses but also activate "fight or flight" mechanisms in the brain, resulting in the release of stress hormones and a shift in the perception of the world. Beyond physical responses, trauma affects perception, beliefs, and personal identity. It can alter one's worldview, influence relationship patterns, and change the way an individual views themselves and others. When a traumatic event occurs, the brain activates alert mechanisms that may inhibit rational control, leading to disproportionate and persistent emotional reactions. When the brain detects a threat, it activates primitive areas responsible for automatic responses, which can suppress rational control and provoke disproportionate reactions. If the stress response is blocked, the body may remain in a state of alertness, generating feelings of agitation. In the case of Post-Traumatic Stress Disorder (PTSD), the balance between brain areas that detect and regulate danger is disrupted. This results in an inability to control emotions and impulses, with the individual potentially experiencing flashbacks and continuously reliving the trauma. Such reactions can lead to a constant state of alertness, exhaustion, and emotional disconnection. The brain is more adapted to handle immediate threats than to manage prolonged trauma. Therefore, treating trauma requires a specific approach, as the brain's responses to ongoing trauma are more complex. Therapy is crucial in helping victims process and understand their traumatic experiences, fostering emotional and psychological recovery. Therapeutic intervention aims to restore emotional balance and enable the individual to resume a healthier and more functional life.

Keywords. trauma, PTSD, Post-Traumatic Stress Disorder, cerebral cortex, amygdala and hypothalamic.

1. Introduction

Trauma affects all dimensions of behavioral functioning, injuring the individual as a whole, resulting in post-traumatic physiological reactions, emotions, perceptions, and cognitive attributions related to the traumatic experience that caused such injuries. Traumatic impact, such as the emotional horror of witnessing a brutal and unexpected death, for instance, is not only emotionally overwhelming, distressing, and difficult to manage but also triggers the release of neuro-hormones and activates the "fight or flight" response. The effects of trauma can produce changes in worldview, beliefs about human nature, patterns of intimacy, interpersonal relationships, self-conceptions, and personal identity. Trauma does not occur in isolation or in a vacuum. Its effects are multidimensional in terms of post-traumatic psychological functioning, influencing motivation, goal pursuit, and levels of self-awareness in the world. The impact of trauma on the individual is a complex mind-body phenomenon. It is important to note that, semantically, the word "trauma" derives from Greek and Latin roots, where "trauma" means injury to the body and results in a state of being wounded. Physical trauma causes injury to bodily integrity and normal biological functioning. Psychological trauma inflicts injury on the mind and its inherent processes and functions, including the ego, identity, and self-esteem. Psychological trauma is caused by an external event that affects internal psychological phenomena on multiple levels of functioning and in both conscious and unconscious modalities of perception and behavior [1].

Traumatic experiences often involve a direct threat to life or safety, but anything that leads an individual to feel overwhelmed or isolated can result in trauma. While it is common for most people to experience fear and anxiety during and immediately after a traumatic event, individual emotional responses vary. While some individuals will naturally recover over time, others may continue to experience trauma-related and stress-related symptoms. For example, throughout the pandemic, many healthcare professionals faced moral dilemmas related to the challenges of providing high-quality care with limited equipment or staff. This emotional burden, combined with long work hours, high pressure, and regular exposure to human suffering, is traumatic and increases the risk of developing mental health challenges [2].

2. Discussion

Certain brain areas play a crucial role in understanding trauma, such as the prefrontal cortex, thalamus, hypothalamus, and amygdala. The cerebral cortex, which constitutes the outermost layer of the human brain, is primarily composed of gray matter containing neuronal cell bodies, dendrites, and axonal terminals. This region is fundamental to various higher functions of the nervous system, such as conscious sensory perception, voluntary movement control, abstract reasoning, and complex cognitive functions. Additionally, the cerebral cortex facilitates communication with other parts of the brain through a network of nerve fibers, enabling the coordination and integration of brain activities such as thought, memory, language, attention, emotion regulation, and social behavior [3].

Specific circuits within the amygdala are crucial for fear conditioning. Sensory information about a conditioned stimulus (e.g., driving a car) is integrated with information about an unconditioned stimulus (e.g., a traumatic event like an explosion) within the amygdala. The amygdala is central to the neural circuit that regulates fear conditioning. Typically, input to the lateral nucleus of the amygdala facilitates the learning of fear, while the central amygdala (including both lateral and medial subdivisions) sends output signals about fear to the hypothalamus and brainstem. The intercalated cell masses are believed to manage the inhibition of information flow between the basal nucleus and the central amygdala [4] [5].

Interactions between the medial prefrontal cortex and the hippocampus continuously modulate the amygdala's output to subcortical brain regions, influencing the fear response. The medial prefrontal cortex, especially the ventromedial prefrontal cortex, is traditionally thought to inhibit amygdala activity and lessen subjective distress. Meanwhile, the hippocampus is involved in encoding fear memories and regulating the amygdala. Additionally, the hippocampus and medial prefrontal cortex work together to regulate context and modulate fear responses [4] [5].

In the emotional brain, specifically within the limbic system, there are structures responsible for determining whether the signals received are dangerous or safe. There are two main approaches to altering the threat detection system: a top-down approach, which involves modulating messages coming not only from the prefrontal cortex but specifically from the medial prefrontal cortex; and a bottom-up approach, which operates through the reptilian brain, responsible for basic survival functions such as fight or flight, kill or be killed, and reproduction. Understanding the distinction between these regulatory methods is essential for comprehending and treating trauma. Top-down regulation involves strengthening the medial prefrontal cortex's ability to monitor physical sensations. Conversely, bottom-up regulation involves recalibrating the Autonomic Nervous System (ANS), which has its origins in the brainstem. The ANS can be influenced through breathing, movement, and touch [6].

When external stimuli activate the brain’s alert system, it automatically triggers pre-programmed "fight or flight" responses in the more primitive brain areas. Just as in other animals, the fundamental nerves and chemicals in the brain are directly connected to the body. When the primitive brain takes control, it partially inhibits the higher brain (the conscious mind) and drives the body to react with running, hiding, fighting, or freezing. If this fight, flight, or freeze response is effective, the individual escapes danger and gradually returns to emotional equilibrium, regaining rational control. However, if for any reason the normal response is blocked, the brain continues to release stress chemicals while its electrical circuits remain activated in vain [7].

Undoubtedly, danger is an inevitable part of existence, and it is the brain's role to detect it and coordinate the appropriate response. Sensory information from the external environment is received through vision, smell, hearing, and touch. These sensations converge in the thalamus, a region located in the limbic system that functions as the brain’s integrative center. From the thalamus, sensations follow two distinct pathways: one directed towards the amygdala and another towards the frontal lobes [8].

The functioning of the thalamus can be disrupted. Visual, auditory, olfactory, and tactile stimuli are recorded in a fragmented and disconnected manner, while regular memory processing deteriorates. Perception of time may seem stagnant, giving the impression that the imminent danger will last indefinitely [9].

The primary function of the amygdala is to determine whether the received stimuli are significant for survival. It performs this function quickly and automatically, aided by feedback from the hippocampus [10] [11]. The amygdala processes information received from the thalamus more rapidly than the frontal lobes, determining whether this information represents a threat to survival before the person is even aware of the danger. When the body becomes aware of what is happening, it may already be responding. If the amygdala interprets the danger too intensely or if the filtering mechanisms of the frontal lobes are weak, as often occurs in individuals with PTSD, there can be a loss of control over automatic emergency responses, such as bursts of aggression or prolonged states of startle [12].

The danger signals sent by the amygdala trigger the release of potent stress hormones such as cortisol and adrenaline, which increase heart rate, blood pressure, and respiratory rate, preparing the body for fight or flight. Once the danger has passed, the body typically returns to its equilibrium state quickly. However, if recovery is impeded, the body remains in a state of alert, resulting in feelings of agitation and excitement [13] [14].

As long as the individual is not excessively disturbed, the frontal lobes can help restore balance by allowing them to recognize that they are reacting to a false alarm and thus counteract the stress response [15].

In Post-Traumatic Stress Disorder (PTSD), the delicate balance between the amygdala (the initial danger detector) and the medial prefrontal cortex (MPFC, the region responsible for regulation and monitoring) is drastically disrupted, resulting in significant difficulties in controlling emotions and impulses (SHIN et al., 2006). Neuroimaging studies of individuals in intense emotional states, such as fear, sadness, and anger, show increased activity in the subcortical areas of the frontal lobe, particularly in the MPFC. When this occurs, the frontal lobe's ability to inhibit impulses diminishes, leading individuals to act disproportionately: they may become easily startled by loud noises, extremely irritated by minor frustrations, or freeze when touched by someone [9].

These traumatic experiences can elicit a range of physiological responses, including an exaggerated startle reflex, heightened vigilance, elevated heart rate and respiration, dry mouth, and increased emotional reactivity and defensive behavior. In the most extreme cases, such experiences may precipitate a flashback, during which the individual may experience temporary difficulty in differentiating past traumatic events from the present situation [5].

Moreover, dissociation is central to trauma. The horrific experience becomes fragmented and divided, causing associated emotions, sounds, images, thoughts, and physical sensations to gain a life of their own. Sensory fragments of memory merge with the present, continuously re-experienced. While the trauma remains unresolved, stress hormones continue to circulate throughout the body as a protective measure, perpetuating defensive movements and emotional responses [16].

In addition to dissociation, depersonalization is also a symptom, and a severe consequence of dissociation caused by trauma. Depersonalization/Derealization Disorder (DP/DR) can be understood as a disturbance in emotional processing. Individuals with DP/DR experience an uncomfortable disconnection from their own senses and from the events around them, as if they were observing their environment and the people around them from outside. This disorder presents symptoms such as diminished affect, somatosensory distortions (perception that the body does not belong to the person, feelings of detachment, and occasionally distortions in the perception of time) [17] [18] [19].

In this context, flashbacks and the repetition of the experience are, in some ways, more challenging than the initial trauma. A traumatic event has a defined beginning and end, but for PTSD sufferers, flashbacks can occur at any time, both during wakefulness and sleep. The constant struggle against invisible dangers is extremely exhausting, leaving individuals drained, depressed, and fatigued. As elements of the trauma are repeatedly relived, the stress hormones associated with them engrave these memories more deeply into the mind. Disinterested and distant, traumatized individuals often feel incapable of fully experiencing life [20].

When encouraged to discuss their experiences, victims may experience increased blood pressure or develop migraines. Some may exhibit emotional blockage and show no visible reaction. However, in clinical settings, accelerated heart rates and the release of stress hormones can often be detected. Many traumatized individuals do not understand the origins of their emotional disconnection. In this scenario, therapy plays an extremely important role in helping patients explore the emotions triggered by trauma and understand the context in which they emerge. However, the challenge lies in the fact that the brain's threat perception system was largely designed to respond to immediate physical threats. It was not designed to handle prolonged traumas, such as childhood abuse, bullying, and domestic violence, necessitating specialized psychotherapeutic treatments [21].

3. Conclusion

Trauma is not an isolated event but has widespread effects that impact various aspects of psychological functioning, including motivation, goal setting, and self-awareness. Understanding trauma involves examining specific brain regions such as the prefrontal cortex, thalamus, hypothalamus, and amygdala. The cerebral cortex, which forms the outer layer of the brain, is made up of gray matter containing neuronal cell bodies, dendrites, and axonal terminals. This area is crucial for higher-order functions, including sensory perception, voluntary movement, abstract reasoning, and complex cognitive processes. It also facilitates communication with other brain regions through a network of nerve fibers, supporting integrated brain functions such as thought, memory, language, attention, emotional regulation, and social behavior.

Specific circuits in the amygdala play a critical role in fear conditioning. Sensory information about a conditioned stimulus, such as driving a car, is integrated with information about an unconditioned stimulus, like a traumatic event, within the amygdala. The amygdala is central to the neural circuit that manages fear conditioning. Generally, input to the lateral nucleus of the amygdala supports the learning of fear, while the central amygdala—comprising both lateral and medial subdivisions—sends fear-related signals to the hypothalamus and brainstem. Interactions between the medial prefrontal cortex and the hippocampus continually adjust the amygdala's output to subcortical brain regions, affecting the fear response. The medial prefrontal cortex, particularly the ventromedial prefrontal cortex, is believed to inhibit amygdala activity and reduce subjective distress. The hippocampus, on the other hand, encodes fear memories and regulates the amygdala. Both the hippocampus and medial prefrontal cortex collaborate to regulate contextual information and modulate fear responses. Understanding the distinction between these regulatory methods is essential for comprehending and treating trauma.

When external stimuli activate the brain's alert system, it triggers instinctive "fight or flight" responses managed by more primitive brain areas. This primal response can suppress higher brain functions, leading to automatic reactions such as fleeing, hiding, fighting, or freezing. If these responses successfully address the threat, the individual can return to emotional stability and regain rational control. However, if the response is hindered, stress chemicals continue to be released, and the brain's electrical circuits remain active without resolving the threat. Additionally, if the dorsolateral prefrontal cortex (DLPFC) becomes inactive, individuals may lose track of time and struggle to differentiate between past, present, and future.

In Post-Traumatic Stress Disorder (PTSD), the balance between the amygdala, which detects danger, and the medial prefrontal cortex (MPFC), which regulates and monitors emotions, is disrupted. This imbalance leads to difficulties in controlling emotions and impulses. Flashbacks and the recurrence of traumatic experiences can be even more distressing than the original trauma. Individuals may experience symptoms like increased blood pressure or migraines when discussing their trauma or exhibit emotional numbness without visible reactions. Clinical observations often reveal increased heart rates and stress hormone levels. Many people with trauma may not understand the root of their emotional detachment. Therapy is essential in these cases, as it helps individuals explore and understand the emotions triggered by their trauma and the context in which they arise.

4. References

[1] Wilson JP, Friedman M, Lindy JD. Treating psychological trauma and PTSD. New York: Guilford Press; 2001.

[2] Mental Health America. Understanding trauma and PTSD. Available from: https://www.mhanational.org/understanding-trauma-and-ptsd. Accessed July 20, 2024.

[3] Guyton AC, Hall JE. Tratado de fisiologia médica. 13th ed. Rio de Janeiro: Elsevier Ltda; 2017.

[4] Parsons RG, Ressler KJ. Implications of memory modulation for post-traumatic stress and fear disorders. Nat Neurosci. 2013 Feb;16(2):146-53. doi: 10.1038/nn.3296. Epub 2013 Jan 28. PMID: 23354388; PMCID: PMC3752300.

[5] Ross DA, Arbuckle MR, Travis MJ, Dwyer JB, Van Schalkwyk GI, Ressler KJ. An integrated neuroscience perspective on formulation and treatment planning for posttraumatic stress disorder: An educational review. JAMA Psychiatry. 2017;74(4):407-15. doi: 10.1001/jamapsychiatry.2016.3325.

[6] MacLean PD. The triune brain in evolution: Role in paleocerebral functions. New York: Plenum Press; 1990.

[7] Lima AA, Vilete L, de Oliveira M, et al. The impact of tonic immobility reaction on the prognosis of posttraumatic stress disorder. J Psychiatr Res. 2010;44(4):224-8. doi: 10.1016/j.jpsychires.2009.08.005.

[8] Edelman GM, Gally JA. Degeneracy and complexity in biological systems. Proc Natl Acad

Sci U S A. 2013;98(24):13763-8.

[9] Van Der Kolk BA. The body keeps the score: Brain, mind, and body in the healing of trauma. Penguin Books; 2014.

[10] LeDoux JE. Evolution of human emotion: A view through fear. Prog Brain Res. 2012;195:431-42.

[11] Feinstein JS, O'Doherty J, Karanian J, et al. The primate amygdala represents the positive and negative value of visual stimuli during learning. Nature. 2011;439(7078):865-70.

[12] Shin LM, Rauch SL, Pitman RK. Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. Ann N Y Acad Sci. 2006;1071(1):67-79.

[13] Williams LM, Palmer DM, Liddell BJ, et al. Mode of functional connectivity in amygdala pathways dissociates level of awareness for signals of fear. J Neurosci. 2006;26(36):9264-71.

[14] Koenig SF, Grafman J. The functional neuroanatomy of depression: Distinct roles for ventromedial and dorsolateral prefrontal cortex. Behav Brain Res. 2009;201(2):239-43.

[15] Diorio D, Viau V, Meaney MJ. The role of the medial prefrontal cortex (cingulate gyrus) in the regulation of hypothalamic-pituitary-adrenal responses to stress. J Neurosci. 1993;13(9):3839-47.

[16] Van Dijke A, Hopman JAB, Ford JD. Affect dysregulation, psychoform dissociation, and adult relational fears mediate the relationship between childhood trauma and complex posttraumatic stress disorder independent of the symptoms of borderline personality disorder. Eur J Psychotraumatol. 2018 Jan 23;9(1):1400878. doi: 10.1080/20008198.2017.1400878. PMID: 29410773; PMCID: PMC5795767.

[17] Phillips ML, Sierra M. Depersonalization disorder: a functional neuroanatomical perspective. Stress. 2003;6(3):157-65. doi: 10.1023/A:1023315129794.

[18] Stein DJ, Simeon D. Cognitive-affective neuroscience of depersonalization. CNS Spectr. 2009;14(9):467-71. doi: 10.1017/S1092852900024541.

[19] Hunter EC, Sierra M, David AS. The epidemiology of depersonalisation and derealisation: A systematic review. Soc Psychiatry Psychiatr Epidemiol. 2004;39(1):9-18. doi: 10.1007/s00127-004-0701-x.

[20] Smith JM, Hartelius E. Flashback phenomenology: An empirical phenomenological analysis of unwanted involuntary memories. Psychol Psychother. 2020;93(3):313-29.

[21] Van Der Kolk BA. The embodied mind: Clinical applications of yoga and mindfulness for trauma recovery. In: Briere J, Lanktree C, editors. Integrative clinical psychology, psychiatry and behavioral medicine: Perspectives, practices and research. Springer; 2018.