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NEUROBIOLOGY OF TRAUMA: IMPACTS, DEFENSE MECHANISMS AND THE SEARCH FOR HEALING

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Abstract: This article explores the neurobiology of trauma, focusing on behavioral impacts, the brain's defense mechanisms and the search for healing. The analysis covers the function of the hypothalamic-pituitary-adrenal (HPA) axis and the limbic system, highlighting the role of the amygdala, hippocampus and prefrontal cortex in the response to trauma. In addition, the article discusses dysfunctions in neurotransmitter systems such as serotonin, dopamine and glutamate, which are central to the manifestation of symptoms such as anhedonia, anxiety and depression. The protective effect of unconscious mechanisms is also analyzed, as well as the importance of bringing traumatic memories to consciousness to promote healing. Fragmentation of consciousness and dependence on external validation are discussed as consequences of trauma that perpetuate psychological suffering.
Keywords: Trauma, neurobiology, HPA axis, limbic system, serotonin, dopamine, glutamate, defense mechanisms, fragmentation of consciousness.

INTRODUCTION

Trauma, an umbrella term that refers to experiences that have a significant negative impact on physical and mental health, can have lasting consequences on individuals' lives. These experiences, which range from one-off to repeated events, involve real or perceived threats to the individual's physical, emotional or psychological integrity. Trauma can result in intense and persistent stress reactions, affecting social, physical and emotional well-being, and manifesting as psychiatric disorders such as Post-Traumatic Stress Disorder (PTSD). The neurobiology of trauma involves a complex interaction between various brain regions and neurotransmitter systems. The limbic system, particularly the amygdala, hippocampus and prefrontal cortex, plays a central role in modulating the response to trauma. In addition, the hypothalamic-pituitary-adrenal (HPA) axis is one of the main mediators of the stress response, and its dysfunction is associated with the development of conditions such as PTSD. Understanding the defense mechanisms that the brain uses to deal with these experiences, and the importance of bringing traumatic memories to consciousness to promote healing, are crucial to the development of effective therapeutic strategies.

WHAT IS A TRAUMA?

Trauma is a very broad term that covers a variety of situations that have a strong negative impact on a person's physical and mental health and which last over time. These situations can be one-off or repeated, and involve a threat or real damage to the life, integrity or dignity of the individual or those close to them.

It can cause intense and persistent stress reactions, which affect a person's functioning and well-being in various aspects, such as social, physical and emotional. These reactions can manifest as symptoms of post-traumatic stress disorder (PTSD), or as normal adaptive processes aimed at survival, but which can become dysfunctional if they are not properly processed and integrated.

Trauma can also be understood in different ways, depending on each person's theoretical, cultural and individual perspective, but it generally involves a breakdown in the ability to deal with events that refer to a traumatic situation and a change in the view of the world and of oneself, as well as being understood as a defense mechanism to avoid constantly ruminating on these experiences.

The so-called complex traumas are situations in which the individual experiences the trauma repeatedly over a period of time or multiple types of trauma, which generates a demand for more extensive and intensive treatment and adaptations of the traditional treatments used for PTSD.

THE TRAUMA CIRCUIT

The article by Shin and Liberzon (2010) discusses the trauma circuit, which is the set of brain structures involved in the development and maintenance of Post-Traumatic Stress Disorder (PTSD). The predominant model suggests that the amygdala, responsible for processing fear, becomes overactive in PTSD, resulting in exaggerated fear responses and the persistence of traumatic memories. In contrast, the medial prefrontal cortex (mPFC), especially its rostral part (rACC), is hypoactive, failing to inhibit the amygdala. This dysfunction in the mPFC can lead to deficits in fear extinction, emotional regulation, attention and contextual processing. The hippocampus also plays an important role in the trauma circuit, with its abnormal function contributing to problems in contextual processing, memory and neuroendocrine dysregulation. The dorsal anterior cingulate cortex (dACC) and insular cortex also appear to be involved, with hyperactivity observed in PTSD. The dACC may be related to exacerbated fear learning, while the insular cortex may be associated with generalized anxiety and the monitoring of internal bodily states. In short, the trauma circuit is characterized by a complex interaction between brain structures, with the hyperactive amygdala and hypoactive mPFC playing central roles in the expression of PTSD symptoms

HYPOTHALAMIC-PITUITARY-ADRENAL (HPA) AXIS AND LIMBIC SYSTEM: NEUROENDOCRINE RESPONSES TO TRAUMA

Trauma provokes a series of complex and highly coordinated neurobiological responses involving various brain regions and neurotransmitter systems. The interactions between the limbic system and the hypothalamic-pituitary-adrenal (HPA) axis are crucial to understanding how the body responds to acute and chronic stress [McEwen, 2007].

LIMBIC SYSTEM AND RELATED STRUCTURES

Amygdala: The amygdala is central to the evaluation of threats and the activation of the fear response [Shin et al., 2006]. In trauma situations, its hyperactivity is related to an exaggerated perception of danger, even in safe contexts. This hyperactivity can be mediated by neurotransmitters such as **serotonin (5-HT)** and **norepinephrine**, which modulate alertness and anxiety [Arnsten, 2009].

Hippocampus: The hippocampus, which is essential for the consolidation of contextual and spatial memories, suffers atrophy when exposed to high levels of cortisol, as occurs in situations of chronic stress [Bremner, 2006]. This atrophy compromises the ability to form new memories and contributes to the difficulty in distinguishing between the safe present and traumatic memories. The regulation of **glutamate** and **GABA** in the hippocampus is critical for synaptic plasticity and neurogenesis, both of which are negatively affected by trauma [Teicher et al., 2012].

Pre-Frontal Cortex: The pre-frontal cortex (PFC) is divided into several sub-regions, each with specific functions in emotional regulation and executive control:

- **Dorsolateral Prefrontal Cortex (DPRFC):** It is involved in cognitive regulation and planning. Chronic stress can reduce the activity of the DFCL, compromising decision-making and executive control [Arnsten, 2009]. **Dopamine** plays a central role in modulating the function of the CPFDL.
- **Ventromedial Prefrontal Cortex (VPFC):** Regulates emotional decision-making and is closely linked to the amygdala. Dysfunction in the PFCVM, exacerbated by exposure to trauma, is associated with difficulty in regulating intense emotions. **Oxytocin** and **vasopressin** modulate the interactions between

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the CPFVM and the amygdala, influencing social and emotional responses.

- **Prefrontal Orbitofrontal Cortex (PFOC):** Involved in decision-making and reward regulation, the PFOC is sensitive to the imbalance in **serotonin** and **dopamine** levels often observed in individuals with PTSD [Heim et al., 2008].

Insula: The insula integrates emotional and sensory signals, contributing to emotional awareness and the processing of bodily sensations. The insula is highly responsive to neurotransmitters such as **histamine** and **glutamate**, and its hyperactivity is related to the altered interoception observed in traumatized individuals.

Anterior Cingulate Cortex (ACC): The ACC is involved in processing emotional conflicts and regulating mood. Alterations in the function of the ACC, mediated by neurotransmitters such as **dopamine** and **GABA**, are associated with symptoms of depression and anxiety in individuals exposed to trauma.

BRAINSTEM AND THALAMUS

- **Brainstem:** The locus coeruleus, the main source of norepinephrine in the brain, and the parabrachial nucleus, involved in the fear response and pain regulation, are crucial brainstem structures in the response to trauma.
- **Thalamus:** The thalamus acts as a relay center for sensory and emotional information to the cortex, playing a role in modulating attention and the fear response.

HYPOTHALAMIC-PITUITARY-ADRENAL (HPA) AXIS

The HPA axis coordinates the endocrine response to stress, with the **hypothalamus** releasing **corticotropin-releasing hormone (CRH)**, which stimulates the **pituitary gland** to secrete **adrenocorticotropic hormone (ACTH)**. ACTH, in turn, promotes the release of **cortisol** by the adrenal glands. In trauma situations, this system can become dysregulated, resulting in chronically high levels of cortisol, which exacerbates the dysfunction of the hippocampus and other brain areas involved in processing memories and emotions [Yehuda, 2002].

- **Cortisol:** Cortisol has complex effects on the brain, including modulating memory circuits in the hippocampus and regulating emotions in the amygdala and prefrontal cortex. Chronically high levels of cortisol are associated with atrophy of the hippocampus and dysregulation of the function of the prefrontal cortex.
- **Neurotransmitters Involved:** The interaction between the HPA axis and neurotransmitters is critical for the stress response. **Serotonin** and **norepinephrine** modulate the release of CRH and the system's sensitivity to stress. **GABA** and **glutamate** are fundamental for regulating the negative feedback that normally limits the stress response, preventing system overload.
- **Genetic and Epigenetic Influence:** Genetic polymorphisms in the genes that code for glucocorticoid and serotonin receptors can influence the reactivity of the HPA axis and predisposition to developing PTSD [Binder et al., 2008; Kilpatrick et al., 2007]. In addition, traumatic experiences can lead to epigenetic changes, modifying gene expression without altering the DNA sequence, and contributing to vulnerability to trauma and its long-term effects.

OTHER MOLECULES AND HORMONES

- Anandamide**:** This endocannabinoid modulates the stress response and plays a neuroprotective role in trauma situations, regulating the function of the amygdala and hippocampus.
- **Nitric Oxide (NO):** NO acts as a neuromodulator, influencing synaptic plasticity in the hippocampus and cerebral blood flow, both of which are essential for the adaptive response to stress.
- **Substance P:** Associated with the transmission of pain, substance P is also involved in the mediation of stress responses, influencing the activation of the amygdala and the modulation of autonomic responses.
- **Endogenous Opioids:** Involved in the modulation of pain and emotions, endogenous opioids such as endorphins play a role in regulating the stress response, acting mainly in the limbic system.

NEUROTRANSMITTERS AND TRAUMA GENOMICS

The dysfunctional regulation of neurotransmitters is a central aspect in the neurobiology of trauma, directly impacting the symptoms observed in traumatized individuals. Serotonin (5-HT), dopamine and glutamate are some of the neurotransmitters most involved, playing crucial roles in modulating mood, motivation and emotional responses.

- **Serotonin (5-HT):** Serotonin is fundamental in regulating mood and anxiety. Dysfunctions in its signaling are associated with anhedonia, depression and other emotional symptoms often observed in people who have suffered trauma. Variants in the serotonin transporter gene (5-HTTLPR), particularly the low expression «s" variant, have been associated with greater vulnerability to the development of Post-Traumatic Stress Disorder (PTSD) after exposure to trauma [(Caspi et al., 2003)].

- **Dopamine:** This neurotransmitter is crucial for the reward system and pleasure regulation. Dysfunction in dopaminergic signaling, especially in the mesolimbic circuit, can lead to symptoms of anhedonia and motivational dysfunction. Studies suggest that polymorphisms in the catechol-O-methyltransferase (COMT) gene, which degrades dopamine, may influence the response to stress and susceptibility to PTSD [(Zubieta et al., 2003)].

- **Glutamate:** As the brain's main excitatory neurotransmitter, glutamate plays an essential role in synaptic plasticity and memory formation. Trauma can lead to glutamate-mediated excitotoxicity, resulting in neuronal damage, especially in areas such as the hippocampus, which is associated with memory deficits and cognitive difficulties [(Krystal et al., 2017)].

GENOMIC ASPECTS

In addition to neurotransmitters, genetics plays a vital role in predisposing people to developing PTSD. Genome wide association studies (GWAS) have identified several genetic variants that influence vulnerability to trauma, many of which are related to the regulation of the hypothalamic-pituitary-adrenal (HPA) axis and stress response systems.

- **Cortisol and the HPA axis:** Variants in genes such as FKBP5, which regulates the sensitivity of glucocorticoid receptors, have been associated with altered responses to stress and an increased risk of PTSD. FKBP5 influences the negative feedback of the HPA axis, and specific variants can lead to greater reactivity to stress [(Binder et al., 2008)].

- **Noradrenergic systems:** Polymorphisms in genes related to the noradrenergic system, such as the dopamine beta-hydroxylase (DBH) gene, which converts dopa-

mine into norepinephrine, are associated with amplified responses to stress and an increased risk of PTSD. The locus coeruleus, the main source of norepinephrine in the brain, is crucial in the «fight or flight" response and its hyperactivity is associated with symptoms such as hypervigilance and exacerbated reactivity to stress [(Liberzon et al., 1999)].

- **Neuropeptide Y (NPY):** NPY acts as a neuromodulator that attenuates the stress response, promoting resilience. Genetic variants that affect the expression of NPY can alter this ability to attenuate stress, influencing vulnerability to PTSD [(Yehuda et al., 2006)].

- **Cannabinoid Receptors (CNR1):** Variants in the gene encoding the cannabinoid receptor type 1 (CNR1) are associated with stress modulation and PTSD risk, suggesting that the endocannabinoid system plays a role in trauma response and emotional regulation [(Hauer et al., 2013)].

These genomic studies highlight the complexity of the molecular pathways involved in the response to trauma, showing that vulnerability to PTSD is influenced by a dynamic interaction between genetic and environmental factors. Understanding these interactions is crucial for the development of personalized therapeutic interventions.

BEHAVIORAL EFFECTS OF TRAUMA

Psychological trauma can trigger a series of profound impacts on an individual's mental health and behavior. Among the most common conditions associated with traumatic experiences are Post-Traumatic Stress Disorder (PTSD) and Acute Stress Disorder (ASD). These disorders reflect the brain's response to extreme stress, which involves a complex combination of neurobiological, psychological and environmental factors.

POST-TRAUMATIC STRESS DISORDER (PTSD)

PTSD is a serious psychiatric condition that can arise after exposure to traumatic events, such as violence, abuse, serious accidents or natural disasters. Individuals with PTSD often relive the traumatic experience through nightmares, flashbacks and intrusive thoughts that cause great suffering. The amygdala, a brain structure central to threat detection, becomes overactive in individuals with PTSD, contributing to exaggerated reactivity to stimuli reminiscent of the trauma. The noradrenaline system (norepinephrine) plays a crucial role in modulating this hyperactivity, exacerbating the fight-or-flight response that characterizes PTSD.

In addition, the prefrontal cortex, particularly the ventromedial and dorsolateral regions, which are responsible for executive control and emotional regulation, show impaired functioning in individuals with PTSD. This dysfunction leads to difficulties in managing fear and anxiety, as well as inhibiting maladaptive emotional responses. Neurotransmitters such as serotonin and GABA are critical in regulating these regions and, when dysregulated, contribute to symptoms of hypervigilance, irritability and concentration difficulties [(Yehuda, 2002); (McEwen, 2007)].

The behavioral symptoms of PTSD also include avoidance of situations or memories associated with the trauma, which can lead to social isolation and reduced quality of life. Dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis in response to stress, with altered cortisol levels, is associated with the development and maintenance of PTSD, further complicating recovery without appropriate medical or psychological intervention [(Heim et al., 2008)].

ACUTE STRESS DISORDER (ASD)

Acute Stress Disorder (ASD) is an immediate response to trauma, typically occurring within a month of the traumatic event. Like PTSD, ASD is characterized by symptoms of dissociation, negative mood, and intrusive thoughts that can be intensely disturbing. ASD can be considered an early form of PTSD, because if symptoms persist beyond a month, the diagnosis can evolve into PTSD.

The neurobiological mechanisms of ASD involve the acute activation of the HPA axis, resulting in an exacerbated stress response that includes the massive release of cortisol and adrenaline. This response is mediated by neurotransmitters such as glutamate, which increases neuronal excitability, contributing to the feeling of hypervigilance and intrusive symptoms. Activation of the amygdala and inhibition of the function of the prefrontal cortex are also common, compromising the ability to process and integrate the traumatic experience in a healthy way.

If left untreated, ASD can progress to PTSD, increasing the risk of long-term complications, including depression, chronic anxiety, and significant functional impairment. Early interventions aimed at restoring neurochemical balance, such as GABA-based therapies and psychotherapeutic approaches, are essential for preventing the progression of ASD to PTSD [(Bryant, 2007); (Shin et al., 2004)].

PROTECTIVE EFFECT AND THE IMPORTANCE OF AWARENESS

The protective effect in relation to traumas, especially those deeply rooted in the unconscious, acts in a similar way to psychological defense mechanisms such as repression and dissociation. These mechanisms are ways in which the brain tries to protect the individual from constant suffering, preventing traumatic memories from continually invading consciousness. However, this protection can come at a cost, as repressed traumatic memories can continue to exert an insidious influence on the psyche, manifesting as feelings of seemingly inexplicable unease, sadness or anxiety.

NEUROBIOLOGICAL MECHANISMS OF TRAUMA AND CONSCIOUSNESS

Although consciousness does not fully retain these traumatic memories, neurobiological evidence suggests that these experiences are located in regions of the brain such as the prefrontal cortex and hippocampus, where they exert a depressive and anxiogenic effect on the individual. When these traumatic memories remain unprocessed, they can contribute to chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis and hyperactivity of the amygdala, both of which are associated with an increased risk of developing disorders such as depression and chronic anxiety [(Yehuda, 2002); (McEwen, 2007)].

The process of bringing these traumatic memories to consciousness, often facilitated by psychological therapies such as cognitive--behavioral therapy (CBT) and eye movement desensitization and reprocessing (EMDR), can result in significant relief. By bringing these memories to the prefrontal cortex, especially the dorsolateral prefrontal cortex (DLPFC), the individual can reprocess the experience, transferring it from the unconscious through the hippocampus. This allows these memories to be "relocated" from the unconscious to the conscious, where they can be integrated and rationalized, reducing their negative emotional impact [(Shin et al., 2006); (Foa et al., 2006)].

This process is not just a rationalization of a negative experience, but a fundamental modification in the "residence" of these memories within the brain. The successful integration of these memories into the prefrontal

cortex can alleviate the associated emotional burden, preventing the constant activation of stress and anxiety neural circuits.

FRAGMENTATION OF CONSCIOUSNESS AND THE SEARCH FOR THE SELF

In addition, it is believed that trauma can cause a fragmentation of self-consciousness, leading to a dissociation between different aspects of personal identity. This fragmentation can result in a pathological dependence on external validation, in an attempt to fill the void created by the trauma. This incessant search for validation in others can generate small dopaminergic rewards which, although temporary, perpetuate the distorted logic of suffering and make healing difficult.

Dopamine, a crucial neurotransmitter in the brain's reward system, is involved in this process, reinforcing behaviors that seek external acceptance but fail to resolve the internal distress caused by the fragmentation of the self. This perpetuation of suffering can be understood as an unsuccessful attempt to restore identity cohesion, leading to repetitive cycles of seeking validation and subsequent frustration [(Van der Kolk, 2014); (Panksepp, 1998)]. By integrating traumatic memories and repairing the fragmentation of consciousness, the individual can rebuild a more cohesive sense of self, which is essential for recovery from trauma and reducing the symptoms of associated disorders such as depression and anxiety.

DISCUSSION AND SUMMARY

Behavioral effects of trauma: Trauma can have various impacts on an individual's health, generating disorders such as post-traumatic stress disorder (PTSD), a condition that arises after a person experiences or witnesses a traumatic event, such as violence, abuse, an accident or a natural disaster. PTSD causes the person to relive the trauma in the form of nightmares, flashbacks or intrusive thoughts, as well as causing anxiety, fear, isolation, irritability and changes in sleep and concentration, damaging the individual's quality of life and well-being and requiring psychological treatment and sometimes medication. Another common disorder caused by trauma is acute stress disorder (ASD), an intense psychological reaction that occurs about a month after a traumatic situation, causing negative mood, dissociation and intrusive thoughts. ASD can also develop into PTSD when the symptoms last for more than a month after the event.

Neuroanatomy of trauma: Some specific regions of the brain also undergo changes in people with PTSD, such as an increase in the amygdala, reduced activity in the medial prefrontal cortex and a decrease in the volume of both the hippocampal and anterior cingulate cortex. In addition, studies have also identified neurochemical changes in the brains of people who suffer from trauma, such as increased levels of dopamine, glutamate, plasma levels of neuropeptide Y, serotonin concentration in some parts of the brain, noradrenaline levels or activity and beta-endorphin levels in the cerebrospinal fluid.

Genetic factors related to trauma: Despite the lack of an in-depth GWAS analysis of the impact of trauma and PTSD, some studies have so far linked some genetic polymorphisms in alternative neurobiological pathways to the problem, such as components of the locus coeruleus and noradrenergic systems (DBH, GAABRA2, NPY and COMT), and some markers of the hypothalamic-pituitary-adrenal axis (CNR1, GCCR and FKBP5). Some studies also raise a relationship between an «s" variant with low inferred expression in the 5-HTTLPR and the increased risk of developing PTSD after exposure to stress from traumatic situations.

Protective effect and the importance of awareness: The protective effect in relation to traumas, particularly when they are deeply rooted in the unconscious, acts in a similar way to a defense mechanism to avoid a constant rehashing of these experiences. Although consciousness does not retain full awareness of these traumas, evidence indicates that they are located in the cortex, exerting a depressive influence on the psyche, and even without understanding why, the person carries a weight, feeling bad. It is by bringing the memory of the trauma to consciousness, that the trauma is relieved, as if it were being removed from where it is parked, but when it is unable to be found, anxiety then fulfills its role as a hangover, but when it can't find the memory, this hangover becomes constant, installed, thus promoting disorders and illnesses such as depression. When this memory is brought to the prefrontal cortex, we sweep it out of the unconscious by transferring it through the hippocampus. In other words, it can be said that rather than rationalizing a negative experience, we are dealing with a change in the "residence" of these painful experiences and intrusive thoughts. Currently, it is also thought that the presence of a trauma generates a fragmentation of the consciousness of the self, producing a kind of dependency in the search for this self in the other, generating small rewards that do not fulfill their initial purpose, but facilitate an acceptance of the wrong logic that generates a perpetuation of suffering.

Personality traits and temperament play a significant role in an individual's vulnerability to developing trauma-related disorders, such as Post-Traumatic Stress Disorder (PTSD). The interaction between these traits, genetic and environmental factors, and exposure to traumatic events can increase the likelihood of a dysfunctional response to trauma.

PERSONALITY AND TEMPERAMENTAL TRAITS ASSOCIATED WITH VULNERABILITY TO PTSD

NEUROTICISM:

- **Description:** Neuroticism is a personality trait characterized by a predisposition to experience negative emotions such as anxiety, fear, sadness and irritability. Individuals with high levels of neuroticism tend to have increased emotional reactivity and greater sensitivity to stress.
- **Relationship with PTSD:** High levels of neuroticism are strongly associated with greater vulnerability to PTSD. The tendency of these individuals to perceive the world as threatening and to experience intense negative emotions can exacerbate fear and anxiety responses after a traumatic event.

EXTROVERSION (LOW):

- **Description:** Extroversion refers to an individual's tendency to be social, assertive and seek external stimuli. Low levels of extroversion, or introversion, are associated with a tendency towards social isolation and less seeking of social support.
- **Relationship with PTSD**: Individuals with low extroversion may have a more limited social support network, which can aggravate the impact of the trauma and make recovery more difficult. Lack of social support is a known risk factor for the development and maintenance of PTSD.

CONSCIENTIOUSNESS (LOW):

- **Description:** Conscientiousness is a personality trait that involves self-discipline, organization and fulfilling responsibilities. Low conscientiousness can be

associated with impulsive behavior and difficulty in dealing with stressful situations in a planned manner.

- **Relationship with PTSD:** Individuals with low conscientiousness may find it more difficult to implement effective coping strategies and proactively seek help, which may increase the risk of developing PTSD.

SENSITIVITY TO REJECTION:

- **Description:** Rejection sensitivity is a person's tendency to react strongly to signs of social rejection, interpreting even neutral interactions as rejecting.
- **Relationship with PTSD:** People with a high sensitivity to rejection can experience the impact of trauma more intensely, especially if the traumatic event involves elements of rejection or social exclusion. This can intensify feelings of insecurity and fear, increasing the risk of PTSD.

RESILIENCE (LOW):

- **Description:** Resilience is the ability of an individual to adapt and recover from adverse situations. Individuals with low resilience find it more difficult to overcome challenges and are more vulnerable to stress.
- **Relationship with PTSD:** Low resilience is associated with a greater likelihood of developing PTSD, since these individuals are less able to cope with the psychological impact of the trauma and to recover after the event.

In addition to the personality traits mentioned, other factors such as previous mental health history, the intensity and proximity of the traumatic event, and the social support received after the trauma also play a crucial role in the development of PTSD. More rigid personalities, who find it difficult to cope with change and adaptation, may also find it harder to process the trauma in a healthy way.

Identifying these traits can be useful for targeting preventive interventions and personalized therapies focused on increasing resilience and coping skills, as well as reducing vulnerability to the development of trauma--related anxiety disorders.

If you need more information or more in--depth information on any of these topics, I'm happy to help.

FINAL CONSIDERATIONS

The neurobiology of trauma reveals the complexity of brain and body responses to traumatic events. Alterations in the HPA axis and limbic system, as well as dysfunctions in neurotransmitter systems such as serotonin, dopamine and glutamate, are central to understanding the symptoms associated with PTSD and other trauma-related conditions. In addition, the unconscious defense mechanisms that protect the individual from constant suffering can paradoxically perpetuate psychological suffering if traumatic memories are not processed and integrated into consciousness. Fragmentation of consciousness and reliance on external validation are important aspects that need to be addressed in the treatment of trauma in order to promote a full and sustainable recovery. Advances in understanding these mechanisms will allow for the development of more effective and personalized interventions for those suffering from the consequences of trauma

Dysregulation in the trauma circuit, which contributes to the development and maintenance of anxiety disorders such as PTSD, is a complex and multifactorial phenomenon. Hyperactivity of the amygdala, hypoactivity of the medial prefrontal cortex (mPFC) and dysfunction of the hippocampus are key alterations in this circuit. The amygdala, crucial in fear processing, shows an exacerbated res-

ponse to trauma-related stimuli, resulting in exaggerated fear responses and persistence of traumatic memories. The mPFC, particularly its rostral portion (rACC), exhibits reduced activity, impairing its ability to inhibit the amygdala and modulate emotional responses, which can lead to deficits in fear extinction, emotional regulation, attention and contextual processing. The hippocampus, which is essential for memory and contextual processing, also functions abnormally, contributing to deficits in contextual processing, memory difficulties and neuroendocrine dysregulation. The causes of this dysregulation are diverse, including genetic vulnerability, traumatic experiences, childhood stress and alterations in neurochemistry. The interaction between genes and environment plays a crucial role, with specific genetic variations increasing susceptibility to developing anxiety disorders after exposure to traumatic events. Early stress can generate long-term alterations in the stress response and in key neurotransmitter systems, such as GABA and serotonin, which can also contribute to dysregulation. Epigenetic factors, such as modifications in gene expression induced by traumatic experiences, can increase vulnerability to anxiety disorders. In short, the combination of genetic, environmental and neurochemical factors, along with others such as life experiences, personality traits and social support, contributes to dysregulation in the trauma circuit, which in turn influences the development and maintenance of anxiety disorders.

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