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PTSD

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Abstract

Introduction and Definition: Post-Traumatic Stress Disorder (PTSD) is a psychiatric condition arising from exposure to traumatic events. The World Health Organization (WHO) defines PTSD as: *"a mental health condition that develops in some people who have experienced or witnessed a traumatic or frightening event such as a natural disaster, a serious accident or assault, a terrorist act or military combat, or those who have been threatened with death, sexual violence or injury"*. The most common symptoms of PTSD include sleep disturbances, recurrent nightmares, and intense distress over reminders. The condition's first recognition in the third edition of Diagnostic and Statistical Manual of Mental Disorders (DSM-III) in 1980 revolutionized the clinical understanding of trauma's long-term psychological and physiological effects. Nowadays, PTSD's relevance continues to grow in clinical neuroscience as research reveals how trauma reshapes the brain's architecture and function.

Body: This encyclopedia entry explores the neurobiology of PTSD, beginning with an overview of its diagnostic criteria under DSM-5, which include symptoms across four clusters: intrusion, avoidance, negative alterations in cognition and mood, and hyperarousal. The entry focuses on several key brain regions consistently implicated in PTSD. The amygdala becomes hyperactive, which is associated with excessive threat detection and fear responses. The hippocampus often exhibits reduced volume, contributing to impaired memory processing and difficulties in distinguishing safe contexts from traumatic memories. The prefrontal cortex (PFC) shows reduced activity, limiting top-down emotional regulation and exacerbating reactivity. These alterations are further compounded by dysregulation in the Hypothalamic-Pituitary-Adrenal (HPA) axis, which affects cortisol levels and perpetuates chronic physiological stress. Neuroimaging and structural imaging studies confirm these findings, while research into developmental trauma highlights how early adversity affects brain maturation. The entry also explores how these insights inform treatment, including CBT and pharmacological approaches. Future directions include examining the environmental, genetic, and epigenetic factors that support resilience to trauma, as well as developing novel interventions such as psychedelic-assisted therapy and decoded neurofeedback therapy.

keywords: trauma; disorder; post-traumatic stress; mental health; neuroscience; neurobiology; neurodevelopment; brain changes; resilience; DSM

Introduction and Definition

Post-Traumatic Stress Disorder (PTSD) is a psychiatric disorder that emerges following exposure to a traumatic event involving actual or threatened death, serious injury, or sexual violence [1]. While distress is a normative response to trauma, PTSD reflects a chronic and maladaptive disruption in psychological and physiological functioning that persists beyond the initial stressor [2]. Characterized by intrusive re-experiencing, emotional dysregulation, hyperarousal, and cognitive alterations, PTSD is associated with significant impairments in daily functioning and quality of life [3].

PTSD was first formally recognized in 1980 with its inclusion in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III), following decades of accumulated observations in military and clinical settings [4]. Its conceptualization has evolved significantly, culminating in its current classification as a trauma-related disorder in the DSM-5 [4]. According to DSM-5 criteria, PTSD is diagnosed when traumatic experiences lead to persistent symptoms across four clusters: intrusive re-experiencing, avoidance of trauma-related stimuli, negative alterations in cognition and mood, and marked alterations in arousal and reactivity [4].

Contemporary neuroscience has shifted the understanding of PTSD from a purely psychological construct to one grounded in neurobiology [5]. Trauma alters brain function and structure, disrupts neuroendocrine systems, and reshapes emotional and cognitive circuits [5].

These alterations are shaped by a range of factors, including genetic predisposition and developmental timing of trauma [2]. This entry examines PTSD's clinical criteria, underlying mechanisms, affected brain regions, and emerging treatment directions [6].

Body

Diagnostic Criteria and Symptomatology

The DSM-5 outlines four primary symptom clusters for PTSD: intrusive symptoms, avoidance behaviors, negative alterations in cognition and mood, and alterations in arousal and reactivity [6]. These symptoms must persist for more than one month following trauma exposure and must cause significant distress or impairment in functioning [7]. The trauma must involve exposure to actual or threatened death, serious injury, or sexual violence—whether directly, as a witness, or through vicarious experience (e.g., first responders) [7].

Intrusion symptoms include recurrent involuntary memories, nightmares, flashbacks, and physiological distress in response to trauma reminders [6]. Exaggerated fear responses are contributed to by amygdala hyperactivity in PTSD patients [8]. The hippocampus, playing a central role in contextual memory, often exhibits structural and functional deficits, contributing to disorganized and intrusive recollections [9].

Avoidance symptoms involve efforts to avoid trauma-related thoughts, feelings, or external reminders [10]. Neuroimaging studies indicate that these symptoms are associated with reduced activity in the medial prefrontal cortex (mPFC), which plays a key role in emotional regulation and fear extinction [8].

Negative alterations in cognition and mood encompass distorted self-blame, persistent negative emotions, diminished interest in activities, and feelings of detachment or estrangement from others [10]. These features suggest broader dysfunction in limbic-prefrontal connectivity [11]. Decreased connectivity between the ventromedial and dorsolateral prefrontal cortices (vmPFC and dlPFC, respectively) and limbic regions, including the amygdala and anterior cingulate cortex, is associated with impaired cognitive reappraisal and emotional regulation [11].

Arousal and reactivity symptoms include irritability, hypervigilance, exaggerated startle response, concentration difficulties, and sleep disturbances [10]. These symptoms are linked to heightened activity in the sympathetic nervous system and noradrenergic pathways, particularly within the locus coeruleus [12]. This overactivation is compounded by reduced inhibitory control from the prefrontal cortex, contributing to impulsivity and impaired stress regulation [12].

Together, the symptom clusters reflect complex dysfunction across multiple brain systems [15]. Understanding these patterns helps guide individualized assessment and supports the development of treatments aimed at restoring disrupted neural processes [7, 14].

Etiology and Risk Factors

PTSD arises from a convergence of traumatic exposure and individual vulnerability factors that shape the neurobiological response to trauma [16]. While trauma is a necessary condition, it is not sufficient: most individuals exposed to trauma do not develop PTSD [16]. This discrepancy points to the importance of examining genetic, epigenetic, neurobiological, and psychosocial risk factors [16].

The type and intensity of trauma strongly influence PTSD risk [17]. Interpersonal traumas—such as sexual assault, torture, and child abuse—carry higher PTSD rates than natural disasters or accidents, suggesting that trauma involving direct personal violation may be particularly pathogenic [18]. Repeated or chronic trauma is also especially potent, often resulting in more severe and persistent neurobiological alterations, including hippocampal atrophy and amygdala sensitization [19].

Genetic predispositions also contribute to PTSD vulnerability [20]. Variants in genes regulating the stress response—such as *FKBP5*, *COMT*, and *SLC6A4*—modulate cortisol sensitivity, emotional regulation, and serotonergic tone, respectively [19]. Trauma exposure has been associated with epigenetic changes in glucocorticoid receptor genes (e.g., *NR3C1*), which may persist into adulthood and contribute to altered HPA axis function and heightened stress reactivity [19].

In sum, PTSD results from an interplay between trauma exposure and individual vulnerabilities that collectively shape the brain's response to stress [16]. These factors influence the onset, expression, and chronicity of PTSD, highlighting the importance of personalized assessment and intervention strategies [16].

Key Brain Structures

Amygdala

The amygdala detects and processes emotional stimuli, especially fear and threat [19]. In PTSD, amygdala hyperactivity has been observed in response to trauma-related cues, which is associated with exaggerated fear responses, hypervigilance, and intrusive memories [8]. Functional MRI (fMRI) studies showed heightened amygdala activation even to neutral stimuli [8], contributing to the overgeneralization of fear and difficulty distinguishing between safe and threatening contexts [19].

Hippocampus

The hippocampus is critical for declarative memory, contextual learning, and the modulation of fear responses [9]. Structural imaging studies have consistently reported reduced hippocampal volume in individuals with PTSD, likely reflecting a combination of neurotoxic effects of chronic stress, glucocorticoid exposure, and developmental disruption [19]. This atrophy contributes to flashbacks and dissociation, where past and present experiences are not properly differentiated [9].

Prefrontal Cortex

The prefrontal cortex (PFC) is essential for executive functioning, impulse control, and emotional regulation [10]. In PTSD, multiple subregions of the PFC are often implicated, such as the vmPFC, which has been shown to inhibit amygdala activity and support fear extinction. It shows reduced activity, contributing to impaired top-down regulation of emotional responses [10]. The dlPFC, involved in working memory and cognitive control, also shows decreased function, contributing to poor regulation of intrusive thoughts and negative beliefs [10]. This prefrontal hypoactivity has been associated with the domination of limbic structures, enabling emotional dysregulation and maladaptive responses to trauma reminders [9].

HPA Axis and Neuroendocrinology

The hypothalamic-pituitary-adrenal (HPA) axis is the central neuroendocrine system contributing to coordinating the body's response to stress [17]. Its dysregulation has been consistently associated with PTSD, with alterations observed in both acute and chronic phases of the disorder [21]. Through the release of glucocorticoids, primarily cortisol, the HPA axis has been found to modulate arousal, memory consolidation, immune responses, and emotional regulation [17]. In PTSD, persistent disruption of HPA axis activity contributes to both psychological symptoms and somatic dysfunction, including chronic pain, gastrointestinal issues, and cardiovascular dysregulation [17].

Neurodevelopment and Childhood Trauma

Sensitive Periods and Brain Maturation

The brain undergoes regionally specific growth during childhood and adolescence [22]. For instance, the amygdala matures early, making young children especially sensitive to emotionally charged experiences [22]. In contrast, the dlPFC and vmPFC that support executive function and emotional regulation, develops more slowly and is not fully mature until the mid-20s [22]. The hippocampus also continues to develop into adolescence and is highly sensitive to glucocorticoid exposure [22].

When trauma occurs during these sensitive windows, it may lead to accelerated amygdala growth, delayed or stunted prefrontal maturation, and hippocampal atrophy, thereby altering the functional balance between emotion and regulation systems [22]. These alterations increase susceptibility to PTSD, particularly in the form of intrusive memories, emotional lability, and poor impulse control [22].

Structural and Functional Brain Changes

Neuroimaging studies of individuals with histories of childhood maltreatment have revealed reduced hippocampal volume, associated with memory deficits and vulnerability to intrusive trauma recollections [19]. Increased amygdala reactivity has also been observed, consistent with patterns of hypervigilance and threat sensitivity [8]. In addition, cortical thinning in PFC, including the dlPFC, has been reported, which has been linked with impaired emotion regulation and decision-making [8]. Finally, disrupted connectivity between the amygdala and prefrontal regions has been noted, associated with reduced efficiency of top-down control of fear responses [8]. Importantly, these neurobiological alterations are not uniform and can differ based on trauma type, duration, and developmental timing [22].

Implications for Lifespan Risk and Resilience

Childhood trauma confers a dose-dependent risk for PTSD, with the likelihood and severity of symptoms increasing with the number of Adverse Childhood Experiences (ACEs) [17]. These early insults can also sensitize individuals to later trauma, where developmental vulnerabilities interact with adult stressors to produce chronic pathology [17]. However, protective factors—such as secure attachment, supportive caregiving, and early intervention—can buffer against neurobiological dysregulation and promote resilience [23]. This highlights the importance of developmental timing for prevention and intervention [17].

Treatment Implications

Psychological Therapies

Trauma-focused cognitive behavioral therapy (TF-CBT) remains the gold-standard psychological treatment for PTSD [17]. TF-CBT incorporates exposure-based techniques, cognitive restructuring, and emotion regulation training [17]. Neuroimaging studies show that successful CBT is associated with increased activity in the prefrontal cortex, particularly the dlPFC and vmPFC, supporting improved top-down regulation of emotion [24]. At the same time, reduced amygdala activation has been observed, which may reflect decreased fear responses to trauma-related stimuli [24]. Additionally, improvements in hippocampal functioning have been reported, aiding contextual memory integration and fear extinction [24].

These changes correspond to clinical improvements in intrusive symptoms, hyperarousal, and cognitive distortions [24]. CBT also promotes the reconsolidation of trauma memories within safer frameworks, which may reverse some structural and functional brain abnormalities over time [24].

Pharmacological Treatments

Pharmacological interventions target neurochemical imbalances associated with PTSD, particularly within the serotonergic and noradrenergic systems [25]. Selective serotonin reuptake inhibitors (SSRIs), such as sertraline and paroxetine, are first-line medications approved for PTSD [6]. They modulate serotonergic tone and may normalize hyperactivity in the amygdala and insular cortex, reducing

anxiety and improving mood [25]. Moreover, Prazosin, an alpha-1 adrenergic antagonist, has been found effective in reducing nightmares and sleep disturbances by dampening noradrenergic hyperarousal [25].

Future Directions

Future research in the neuroscience of post-traumatic stress disorder (PTSD) is increasingly turning toward identifying the biological and environmental factors that contribute to resilience, rather than vulnerability, following trauma [26]. One promising avenue involves the investigation of environmental, genetic, and epigenetic mechanisms that underlie adaptive responses to trauma exposure [26].

In parallel, psychedelic-assisted therapy, particularly using MDMA or psilocybin, has shown potential in reducing PTSD symptoms by promoting emotional processing and fear extinction [27]. These substances appear to modulate activity in the amygdala, hippocampus, and prefrontal cortex, reducing hyperarousal and enhancing cognitive control during trauma recall [27]. Ongoing trials aim to clarify long-term efficacy, ideal dosing, and predictors of treatment response [27].

Another emerging approach involves decoded neurofeedback therapy, a technique that trains individuals to modulate their brain activity by providing real-time feedback, and has been used to recalibrate dysfunctional activity in regions such as the amygdala, vmPFC, and insula, which are central to threat detection and emotion regulation [28]. As the field advances, future research should focus on identifying neurobiological predictors of response, and integrating this method with other therapeutic modalities to enhance treatment precision and durability [28].

Conclusion

PTSD is a complex disorder that reflects trauma-induced changes in brain structure and function, stress hormone regulation, and memory processing [10]. The disorder is marked by hyperactivity in the amygdala, hypoactivity in the prefrontal cortex, hippocampal dysfunction, and HPA axis dysregulation [17]. These alterations underlie core symptoms of re-experiencing, avoidance, negative mood, and hyperarousal [14].

Trauma, particularly during sensitive developmental periods, leaves lasting imprints on neural circuits [8]. However, the brain's plasticity offers hope: evidence-based psychological therapies and emerging biological treatments can reverse or compensate for these changes [24–29].

As neuroscience continues to advance, a more nuanced and integrative understanding of PTSD will emerge—one that honors both the biological realities of trauma and the human capacity for recovery [26]. This knowledge will not only enhance treatment outcomes but also reduce stigma, promote resilience, and inform global approaches to mental health in trauma-affected populations [26].

List of Abbreviations

ACEs: adverse childhood experiences
DSM: Diagnostic and Statistical Manual of Mental Disorders
DSM-III: Diagnostic and Statistical Manual of Mental Disorders, Third Edition
DSM-5: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition
dlPFC: dorsolateral prefrontal cortex
fMRI: functional magnetic resonance imaging
HPA axis: hypothalamic-pituitary-adrenal axis
mPFC: medial prefrontal cortex
MDMA: 3,4-methylenedioxymethamphetamine (commonly known as ecstasy)
PFC: prefrontal cortex
PTSD: post-traumatic stress disorder
SSRIs: selective serotonin reuptake inhibitors
TF-CBT: trauma-focused cognitive behavioral therapy
vmPFC: ventromedial prefrontal cortex
WHO: World Health Organization

Conflicts of Interest

The author declares that they have no conflict of interest.

Authors' Contributions

HY: made substantial contributions to the design of the study, the collection of data as well as interpretation and analysis of the data, revised the manuscript critically, and gave final approval of the version to be published.

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