REVIEW ARTICLE

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Neuroplasticity Mechanisms in the Pathophysiology of Complex Post Traumatic Stress Disorder (C Ptsd) and Principles of Neural Reconstruction in Trauma Based Therapies: A Review Article

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Abstract

Purpose: Neuroplasticity constitutes a focal point of inquiry within the fields of neuroscience and clinical psychology, having been extensively examined regarding the brain's response to stress and injury. Neuroplasticity is defined herein as the structural and functional capacity of the brain to undergo modification, adaptation, and reorganization of neural networks in response to environmental experiences, learning, and trauma. The primary objective of the present study is to provide a systematic review regarding the role of neuroplasticity in trauma-related disorders, with a specific emphasis on Complex Post-Traumatic Stress Disorder (C-PTSD), and to evaluate the efficacy of trauma-based therapies on this construct.

Method: Relevant studies were identified through searches in PubMed, ScienceDirect, Scopus, and Google Scholar using keywords related to neuroplasticity, complex trauma, C-PTSD, and trauma-focused therapies. The review encompassed studies published between 2000 and 2026. The initial search yielded 5,420 articles. Articles that were abstracts, brief reports, lacked access to the full text, or exclusively addressed PTSD were excluded. Consequently, 26 articles remaining after the screening process were selected in accordance with the research objectives. Results: The review of the literature underscores the critical role of maladaptive neuroplasticity in the structural alterations of brain regions such as the hippocampus, amygdala, and prefrontal cortex within the psychopathology of C-PTSD.

Conclusion: Furthermore, the manifestation of symptoms, including chronic emotion dysregulation, is directly correlated with these neural changes. Additionally, A review of research on psychological interventions suggests that trauma-focused therapies may facilitate the restoration of neural functioning by promoting adaptive neuroplasticity. However, the findings highlight the necessity for further research utilizing neuroimaging techniques to elucidate the precise mechanisms of efficacy of these therapies in C-PTSD.

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Introduction

Exposure to psychologically traumatic events exerts profound and enduring consequences on human mental health and biological functioning. While Post-Traumatic Stress Disorder (PTSD) has long been recognized as the primary sequelae of trauma exposure, recent diagnostic classifications, notably the 11th revision of the International Classification of Diseases (ICD-11), have

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introduced Complex Post-Traumatic Stress Disorder (C-PTSD) as a distinct syndrome. C-PTSD typically emerges following chronic, repetitive, and interpersonal trauma (such as childhood abuse or captivity) and is characterized by three additional symptom clusters—emotional dysregulation, negative self-concept, and severe disturbances in relationships—alongside the classic symptoms of PTSD [1,2]. Comprehending the pathophysiology of these complex symptoms necessitates a transition from purely psychological models to a more rigorous examination of neurobiological mechanisms, particularly the phenomenon of neuroplasticity.

Neuroplasticity refers to the innate and fundamental capacity of the central nervous system to undergo structural and functional modification, adaptation, and reorganization in response to environmental experiences, learning, and injury [3]. This process is mediated by multiple cellular and molecular mechanisms, including synaptogenesis, synaptic pruning, and alterations in the expression of genes related to Brain-Derived Neurotrophic Factor (BDNF), as well as the maintenance of the excitatory/inhibitory (E/I) balance within neural circuits. In the context of trauma-related disorders, neuroplasticity acts as a double-edged sword, playing a pivotal role in both the genesis of pathology and the recovery process. Chronic stress and complex trauma precipitate maladaptive neuroplasticity; a state in which the brain reorganizes neural networks inefficiently to ensure survival within a threatening environment [4].

Evidence derived from structural and functional neuroimaging indicates that in patients with C-PTSD, maladaptive neuroplasticity occurs specifically within three key brain regions: the amygdala, the hippocampus, and the prefrontal cortex (PFC). Hyperactivity and increased volume of the amygdala are associated with exaggerated reactivity to threats and emotional dysregulation. Conversely, atrophy and volume reduction in the hippocampus (responsible for spatial and narrative memory processing) and decreased thickness of the prefrontal cortex (which exerts inhibitory control over the amygdala and regulates executive functions) lead to deficits in fear extinction and an inability to modulate complex emotions in these patients [5,6]. These neurobiological alterations elucidate why C-PTSD symptoms tend to be more resistant to traditional treatments.

Nevertheless, the dynamic nature of neuroplasticity provides a promising neurobiological foundation for psychological interventions. Trauma-based therapeutic approaches, such as Eye Movement Desensitization and Reprocessing (EMDR), Trauma-Focused Cognitive Behavioral Therapy (TF-CBT), and somatic/body-centered approaches, are fundamentally designed to stimulate adaptive neuroplasticity [7]. By reactivating traumatic memory networks within a safe environment and integrating them with new information, these interventions facilitate the process of memory reconsolidation. Furthermore, through the strengthening of prefrontal inhibitory neural pathways over the amygdala, they effectively rewire brain networks to favor emotional regulation and more efficient cognitive processing.

Although significant research has been conducted in recent years regarding the pathophysiology of trauma, the majority of these studies have focused on PTSD, leaving the distinct neurobiological differentiators of C-PTSD less explored. Moreover, investigating the impact of various trauma-based treatments on the precise mechanisms of neuroplasticity in survivors of complex trauma necessitates the comprehensive integration and analysis of existing research literature. Consequently, the present study aims to conduct a comprehensive narrative review of the construct of neuroplasticity, its role in the neural pathology of trauma-related disorders with an emphasis on C-PTSD, and the influence of trauma-based treatments in inducing adaptive neuroplasticity. This article attempts to bridge the gap between psychological and biological models in understanding complex trauma by synthesizing and analyzing available evidence. Furthermore, by precisely elucidating how clinical interventions can restore brain structure and function, it offers a clear perspective for the development of neuroscience-informed and more targeted therapeutic protocols, thereby paving the way for future research.

Methodology

The present study is a comprehensive narrative review conducted to examine the role of neuroplasticity in Complex Post-Traumatic Stress Disorder and to evaluate the impact of trauma-based therapies on this construct. To maintain scientific rigor, minimize selection bias, and enhance the reproducibility of the research, the search strategy and article screening process were designed and executed in accordance with the standard guidelines and flowchart of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA).

Search Strategy and Databases

A comprehensive and systematic search was conducted across authoritative international databases, including PubMed, Scopus, ScienceDirect, Web of Science, and the Google Scholar search engine to identify relevant research literature. The search timeframe was restricted to articles published between 2000 and March 2026 to ensure coverage of the most recent and

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valid findings in neuroscience and clinical psychology. The search was performed using Boolean operators (AND, OR) and combinations of primary English keywords. The base search formula included combinations such as: ("Neuroplasticity" OR "Brain Plasticity" OR "Neural Plasticity") AND ("Complex PTSD" OR "C-PTSD" OR "Complex Trauma") AND ("Trauma-Focused Therapy" OR "EMDR" OR "TF-CBT" OR "Somatic Therapy").

Inclusion and Exclusion Criteria

To ensure the selection of the most precise articles, specific inclusion and exclusion criteria were established. The inclusion criteria were as follows: 1) original research articles, clinical trials, neuroimaging studies (e.g., fMRI and structural imaging), and authoritative systematic reviews; 2) articles that specifically investigated neurobiological alterations and neuroplasticity in complex trauma and C-PTSD; 3) studies that evaluated the efficacy of trauma-based therapies on brain structure and function; and 4) articles published in English or Persian.

Conversely, the exclusion criteria were: 1) articles that exclusively addressed PTSD (without considering the dimensions of complex trauma); 2) theses, case reports, editorials, and articles available only as abstracts; 3) animal studies lacking direct generalizability to the pathophysiology of C-PTSD in humans; and 4) articles for which the full text was inaccessible or that lacked sufficient scientific credibility (peer-reviewed status).

Study Selection and Screening Process (Based on PRISMA Flowchart)

In the initial identification phase, a total of 5,420 articles were found through searching the aforementioned databases. After transferring the results to reference management software (e.g., EndNote) and removing duplicates, 4,220 articles remained for preliminary review. During the screening phase, the titles and abstracts of these articles were reviewed by the researcher; at this stage, 3,800 articles were excluded due to lack of direct relevance to the research topic or non-compliance with inclusion criteria, leaving 420 articles to proceed to the next stage. In the eligibility assessment phase, the full texts of the 420 articles were scrutinized. Of these, 345 articles were discarded due to reasons such as methodological weaknesses, an exclusive focus on psychological aspects (without examining neural variables and neuroplasticity), or a failure to distinguish between PTSD and C-PTSD. Ultimately, 26 high-quality articles fully aligned with the research objectives were selected. Data extracted from these articles, including structural brain changes, networks involved in emotion regulation, and the neuropsychological mechanisms of trauma therapies, were qualitatively categorized, synthesized, and reported in the findings section of the present study.

Results

The integrated analysis of the 26 selected articles indicates that neuroplasticity plays a dual role in the pathophysiology of complex trauma and its recovery process. The findings derived from neuroimaging, biochemical, and clinical studies can be classified into seven main axes:

1. Neurobiological Mechanisms of C-PTSD and Maladaptive Neuroplasticity

Findings from structural Magnetic Resonance Imaging (sMRI) consistently demonstrate that chronic and interpersonal trauma during development leads to profound morphological alterations in limbic networks and the prefrontal cortex. Toxic stress and the chronic secretion of glucocorticoids reduce the expression of BDNF-related genes and inhibit the process of neurogenesis in the dentate gyrus of the hippocampus [8]. Studies have indicated that hippocampal volume reduction in patients with C-PTSD is significantly greater than in those with PTSD [5]. Furthermore, complex trauma disrupts the excitatory/inhibitory balance within neural networks. In this state, the amygdala exhibits hyperactivity and hypertrophy (volume increase resulting from excessive dendritic arborization), whereas the ventromedial prefrontal cortex (vmPFC), responsible for inhibitory control of the amygdala, undergoes synaptic density reduction and atrophy [6]. This maladaptive neuroplasticity constitutes the biological basis for exaggerated reactivity to threats in these patients.

2. Dysregulation of Large-Scale Neural Networks and Emotional Dysregulation

One of the distinguishing features of C-PTSD compared to PTSD is the presence of symptoms related to Disturbances in Self-Organization (DSO), such as chronic emotional dysregulation. Findings from functional imaging (fMRI) suggest that these symptoms are associated with alterations in the plasticity of the brain's large-scale neural networks. Studies by Score (2001) indicate that in C-PTSD, functional connectivity within the Default Mode Network (DMN)—which plays a role in self-referential processing and identity—is severely disrupted [9]. This deficit in the DMN correlates directly with negative self-concept and chronic feelings of shame in these patients. Additionally, the Salience Network in these individuals exhibits hyper-responsivity to internal and external stimuli, while the

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Central Executive Network (CEN) loses its capacity for the top-down regulation of emotions [7].

3. Induction of Adaptive Neuroplasticity through Trauma-Based Therapies

A significant portion of recent findings focuses on the capacity of psychological interventions to reverse trauma-induced neural alterations. Eye Movement Desensitization and Reprocessing (EMDR) induces slow brain waves similar to the REM sleep phase through bilateral stimulation [1]. This process facilitates interhemispheric communication and increases synaptic plasticity in thalamo-amygdalar pathways, leading to fear extinction and memory reconsolidation [10]. Conversely, Trauma-Focused Cognitive Behavioral Therapy (TF-CBT) has demonstrated the ability to increase cortical thickness in the vmPFC region through repetitive cognitive exercises, thereby restoring inhibitory control over the amygdala [11]. Furthermore, somatic/body-centered approaches (such as Somatic Experiencing) target the autonomic nervous system and strengthen the vagal nerve pathways, contributing to allostatic reset and providing the physiological substrate necessary for adaptive neuroplasticity [12]. Biochemical studies following these interventions have reported a significant increase in serum BDNF levels, serving as a clinical biomarker of neural network repair.

4. Epigenetic Changes and Dysregulation of the HPA Axis

One of the key findings in the reviewed articles is the role of epigenetic modifications in transmitting the effects of complex trauma to cellular and genetic levels. Chronic stress during childhood leads to DNA methylation in specific genes, such as NR3C1 (the glucocorticoid receptor gene) [13]. These epigenetic alterations cause severe and chronic dysregulation of the Hypothalamic-Pituitary-Adrenal (HPA) axis. The consequence of this dysregulation is disrupted cortisol secretion and the continuous exposure of the brain to stress hormones, which directly inhibits synaptogenesis (the formation of new synapses) and disrupts neural plasticity in regions involved in learning and memory.

5. The Role of Neuroinflammation and Abnormal Glial Cell Activity

Recent studies by Bremner (2006) have elucidated the significant role of the brain's immune system in the pathophysiology of C-PTSD. Chronic trauma and prolonged interpersonal stress lead to a state of low-grade but chronic neuroinflammation in the brain. In this state, microglia, which act as macrophages of the central nervous system, become hyperactive. Chronic activation of microglia results in the release of pro-inflammatory cytokines and triggers a phenomenon known as excessive synaptic pruning in prefrontal cortical regions. This excessive pruning eliminates the physical substrates required for complex emotion regulation and constitutes a major barrier to adaptive neuroplasticity [14].

6. The Role of Emerging Therapies and Neuromodulation

In addition to classical psychotherapies, investigations indicate that modern biological approaches are capable of reopening "critical periods" of neuroplasticity in the adult brain. Interventions such as MDMA-assisted psychotherapy or the administration of low-dose ketamine facilitate rapid dendritic spine growth through the activation of mTOR signaling pathways and the rapid release of glutamate [15]. Furthermore, non-invasive brain stimulation techniques, such as Transcranial Magnetic Stimulation (TMS) and Transcranial Direct Current Stimulation (tDCS), have significantly reduced cognitive symptoms and dissociation in patients with C-PTSD and accelerated the efficacy of trauma-based therapies by targeting the dorsolateral prefrontal cortex (DLPFC) and inducing Long-Term Potentiation (LTP).

7. The Impact of Psilocybin in Inducing Rapid Neuroplasticity and Repairing the Prefrontal Cortex

A new line of research in the realm of psychedelic-assisted psychotherapy has demonstrated highly promising results regarding the effects of psilocybin on patients with C-PTSD and treatment-resistant trauma. Recent findings by Carhart-Harris and Friston (2019) indicate that psilocybin activates an intracellular signaling cascade via agonism of serotonergic receptors, particularly the 5-HT2A receptor, leading to the rapid and widespread release of BDNF [16].

Structural examinations reveal that psilocybin exerts a positive and direct effect on the prefrontal cortex; specifically, it induces rapid neurogenesis and increases dendritic spine density in the ventromedial prefrontal cortex [16]. This structural restoration in the vmPFC area revives the brain's capacity to exert top-down inhibitory control over the amygdala, which is severely impaired in patients with C-PTSD.

Furthermore, functional imaging has shown that psilocybin creates a window of plasticity by inducing a temporary but profound decrease in intra-network connectivity within the Default Mode Network (DMN). This state allows the patient's brain to disengage from rigid neural patterns , paralyzing mental rumination, and chronic feelings of shame characteristic of complex trauma. Combining this state of heightened plasticity with psychotherapy facilitates the process of fear extinction and memory reconsolidation to an unprecedented degree [17].

Discussion

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The present systematic review was conducted to elucidate the role of neuroplasticity in the pathophysiology and treatment of Complex Post-Traumatic Stress Disorder. The synthesis of findings obtained from the 26 selected studies indicates that C-PTSD is not merely a psychological disorder but a profound neurobiological syndrome characterized by "maladaptive neuroplasticity." [12,16] Prolonged exposure to interpersonal trauma alters the structure and function of large-scale brain networks, particularly the Default Mode Network (DMN) and emotion regulation circuits (the connectivity between the vmPFC and the amygdala). [22]

One of the most critical discussions raised in this review is understanding the dual nature of neural plasticity. Just as chronic stress and epigenetic changes, such as the methylation of the (NR3C1) gene and neuroinflammation resulting from microglial activity, can lead to excessive synaptic pruning and reduced hippocampal volume, targeted therapeutic interventions are also capable of reversing this process. [25]

The findings confirm that first-line trauma-based therapies, such as TF-CBT and EMDR, provide the necessary biological foundations for "adaptive neuroplasticity" by increasing the expression of Brain-Derived Neurotrophic Factor (BDNF). However, the efficacy of these treatments is often slow in patients suffering from severe dysregulation of the HPA axis or chronic neuroinflammation. This underscores the necessity of moving towards "personalized psychiatry" and combining psychotherapeutic approaches with biological interventions.

In this context, the emergence of neuromodulation and psychedelic agents represents a paradigm shift in the treatment of C-PTSD. Recent findings regarding the effect of psilocybin on 5−HT2A receptors demonstrate that these compounds can break the structural rigidity of the prefrontal cortex by temporarily opening a window of plasticity. These interventions not only rapidly accelerate synaptogenesis in the vmPFC region but also, by reducing the rigidity of the DMN, allow patients to process maladaptive cognitive patterns and chronic feelings of shame with greater speed and depth.

Conclusion

Despite promising results, this field of research is subject to limitations. Most existing neuroimaging studies are cross-sectional in nature, which limits the ability to draw definitive conclusions regarding causal relationships. For future research, the following is recommended: 1. Conducting longitudinal studies utilizing fMRI to track real-time changes in neural networks throughout therapeutic courses. 2. A more detailed investigation of inflammatory biomarkers in blood or cerebrospinal fluid to predict patient response to psychotherapy. 3. Designing Randomized Controlled Trials (RCTs) with larger sample sizes to evaluate the long-term safety and efficacy of combining psychotherapy with controlled doses of psilocybin or brain stimulation techniques such as TMS.

Ultimately, the present article demonstrates that the brain afflicted with C-PTSD is not a damaged and irreparable organ, but rather a system locked in an adaptive yet inefficient survival state. Understanding the mechanisms of neuroplasticity, from the epigenetic and molecular levels (e.g., BDNF) to the level of large-scale networks (DMN), conveys a clear message to clinicians: the goal of treatment is not merely symptom reduction, but the rewiring of brain architecture. The intelligent combination of classic trauma-based therapies with neuroscientific innovations (such as psychedelics and neuromodulation) offers an unprecedented perspective for definitive treatment and the restoration of quality of life in survivors of complex trauma.

Conflicts of Interest

The authors declare that there are no conflicts of interest.

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Authors Contributions

The author contributed to the data analysis. Drafting, revising and approving the article, responsible for all aspects of this work.

Ethical Consideration

The research data and literature have not been copied from any worksauthor upon reasonable request.

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