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The Neuropsychological Fundamentals of Stress Experiences in Military Personnel

Nataliia Sablina
Private Higher Education Institution "University of Modern Knowledge", Kyiv, Ukraine.
natashasablina@ukr.net,
https://orcid.org/0009-0006-2000-983X

Taras Kulakovskyi
Polissia National University, Zhytomyr, Ukraine.
taraskulakovsky@gmail.com,
https://orcid.org/0000-0001-8299-0279

Hanna Yavorska
Vasyl Stefanyk Carpathian National University, Ivano-Frankivsk, Ukraine.
adiza@ukr.net,
https://orcid.org/0009-0002-0685-849X

Maksym Yavorskyi
Cherkasy State Technological University, Cherkasy, Ukraine. jaworski@ukr.net,
https://orcid.org/0000-0002-3464-2453

Serhii Mordiushenko
National University of Civil Protection of Ukraine, Cherkasy, Ukraine.
semimor9@gmail.com,
https://orcid.org/0009-0003-8455-7323

Iryna Ievtushenko
National Technical University of Ukraine "Igor Sikorsky Kyiv Polytechnic Institute",
Kyiv, Ukraine.
eiv7308@gmail.com,
https://orcid.org/0000-0001-5145-7332

Abstract: *Combat operations are a comprehensive test for military personnel serving in regional and local armed conflicts. The tragic consequences of war extend beyond the high number of those killed, wounded, and maimed. Combat stress plays a significant role in operational losses, as it substantially reduces the combat effectiveness of troops. Factors in the combat environment have a strong psychotraumatic effect, disrupting the balance between adaptive mechanisms and the external world. This disruption explains the specific nature of the stress disorders that develop under such conditions. From a conceptual perspective, combat stress should be viewed not merely as a temporary reaction, but as a systemic neurobiological restructuring of the organism. Prolonged emotional pressure triggers a cascade of destructive processes, ranging from neuroendocrine imbalance to structural changes in cerebral vessels. It may increase the risk of strokes and heart attacks, even in individuals with a healthy diet. Emotional responses to stress vary widely among military personnel and are influenced by individual characteristics. Hormonal regulation plays a central role in this process. Stressful situations provoke an excessive release of cortisol, which may become neurotoxic with prolonged exposure. Increased cortisol levels can impact neurogenesis (the formation of new neurones), particularly in the hippocampus, thereby directly impairing a soldier's cognitive flexibility and capacity to learn under combat conditions.*

Keywords: *traumatic events; stressful situations; post-traumatic stress disorder; stress hormones; cortisol; neurogenesis.*

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1. Introduction

In combat situations, the human body undergoes a complex, multilevel process of non-specific adaptive responses. These responses occur within the demanding conditions of warfare, activating mechanisms of reactive self-regulation and leading to specific psychophysiological adaptations. The nature of combat stress, whether constructive or destructive and traumatic, depends on the intensity and duration of stressors, the warrior's characteristics, and resilience.

Constructive combat stress is a functional state in which cognitive, emotional, volitional, and psychomotor processes align to fulfil a combat mission. In this condition, a warrior operates at an optimal level of capability. However, prolonged exposure to intense combat stressors can cause adaptive stress to become damaging. Signs of this harmful stress may include shivering,profound fatigue, heart palpitations, rapid breathing, chest discomfort, shortness of breath, dizziness, irritability, impaired cognitive function, difficulty making decisions, and reduced self-confidence. Under these conditions, both individual and unit combat effectiveness decline, making warriors more vulnerable to enemy attacks and increasing the risk of injury and mortality.

Shchyruk (2024) uses a psychological-axiological approach to study the experiences of military personnel and individuals who have undergone combat stress and developed post-traumatic stress disorder (PTSD). Bryndikov (2019) notes that a warrior under severe combat stress may lose combat readiness, becoming unable to see, hear, understand, move, or remain present on the battlefield. Blinov (2017a, 2017b, 2020) emphasises the role of self-regulation in maintaining combat readiness. He highlights the importance of controlling various physiological functions within the psychophysiological structure of stress. These functions can be tracked internally through self-reflection and externally through careful observation. Restoring normal function is expected to benefit cognitive and psychomotor processes, motivation, and overall combat performance.

According to Parsons (1988), during combat, a soldier is required to evaluate the battlefield, act tactically, confront the enemy, and safeguard both personal safety and the well-being of fellow soldiers. Achieving these objectives requires intentional and focused effort, which directly impacts overall combat performance. However, both over-activation and under-activation of psychophysiological resources can impair performance, ultimately decreasing operational efficiency in the field.

On the other hand, the current theoretical paradigm requires more in-depth analysis. PTSD should be considered not only as a mental disorder but also as a consequence of neural circuits becoming persistently engaged in survival mode. This state leads to a disruption of the functional connections between the prefrontal cortex, responsible for rational control, and the amygdala, which mediates fear responses. The study of this dissociation is key to understanding why a soldier loses the ability to adequately distinguish between combat-related threats and those encountered in peaceful life.

Accordingly, this article aims to examine the neuropsychological mechanisms underlying stress in military personnel and to analyse how PTSD develops during combat. It also seeks to substantiate the concept of neuropedagogical correction, which lies in restoring neuroplasticity and cognitive agency in veterans.

Neuropedagogical correction is regarded as a systematic process of targeted training and retraining for veterans. It is based on the mechanisms of guided neuroplasticity and integrates classical didactic approaches with Cognitive Remediation Therapy (CRT) techniques. Unlike purely clinical rehabilitation, this approach focuses on the deliberate restoration of impaired cognitive domains, including attention, memory, and cognitive flexibility. This is achieved through the development of new behavioural and cognitive strategies within a structured educational environment (Wykes et al., 2011). In this context, external and internal assistive tools that compensate for impaired brain functions (e.g., interactive digital organisers, stress de-escalation algorithms, or mnemonic strategies) function as a form of cognitive prosthesis. Within the framework of cognitive rehabilitation theory and the concept of the Extended Mind, such a prosthesis is understood as an artificially developed compensatory system that assumes the functions of impaired cognitive processes, thereby enabling veterans to adapt more effectively to the demands of civilian life (Clark & Chalmers, 1998; Wilson, 2003).The ultimate goal of this form of instrumental support is to restore cognitive subjectivity, defined as an individual's capacity to regulate their own cognitive processes, emotional responses, and behaviour in a proactive, autonomous, and conscious manner. This construct is closely related to the concept of locus of control in cognitive psychology and represents a fundamental prerequisite for the successful socio-psychological reintegration of military veterans (Bandura, 1999; Ben-Yishay & Diller, 2011).From a methodological perspective, this article is based on the examination of neuropsychological and neuroimaging studies. These include functional magnetic resonance imaging (fMRI) and positron emission tomography (PET).Furthermore, the article synthesises the neurobiological concepts of PTSD, with a focus on a systems approach to the study of stress disorders among combatants. These disorders are analysed across multiple levels, ranging from gene expression to changes in brain architecture.

2. Research Methodology

According to its scientific design, this study constitutes a narrative (descriptive) literature review combined with elements of conceptual and theoretical analysis. A clearly defined search strategy and explicit study selection criteria were employed to ensure scientific rigour, transparency, and objectivity throughout the source selection process.The literature search was conducted using the international bibliographic databases PubMed, Scopus, and Web of Science, as well as Google Scholar. The search strategy was developed using Boolean operators (AND, OR) and included the following search terms, used in various combinations: *"combat stress"*, *"amygdala hijack"*, *"cortisol hippocampal damage"*, *"neuropsychological resilience"*, *"PTSD military"*, and *"neuroscience of stress"*.The search covered publications published between 2016 and 2026, allowing the synthesis of findings from contemporary neuroimaging (fMRI and PET) and neuropsychological research. To provide theoretical and methodological context, seminal works in stress psychology, particularly those by Lazarus (2013), were also included.The inclusion criteria were as follows: (1) peer-reviewed publications reporting empirical findings on the neurobiological and neuropsychological mechanisms of stress; (2) studies investigating acute or chronic stress among combatants, military personnel, or individuals exposed to extreme conditions; and (3) articles describing brain plasticity and mechanisms of cognitive compensation and/or rehabilitation following stress-related injury.The exclusion criteria comprised conference abstracts, non-peer-reviewed publications, and articles of a purely journalistic nature. Studies examining the effects of stress exclusively within the clinical context of severe somatic disorders unrelated to military service were also excluded.The initial search identified more than 120 publications. Following screening and detailed content analysis based on the predefined eligibility criteria, the most relevant conceptual and empirical studies were selected for inclusion in the review, providing the basis for the development of a comprehensive neuropsychological model of combat stress.

3. Neuropsychological Aspects of Stress in Military Personnel

As noted by Lazos (2016), psychological support and timely professional help are important for enabling military personnel to manage stress and emotional strain during service. Short-term stress triggers the body's "fight or flight" response, an evolutionary survival mechanism that, although once used to escape predators, is still activated by today's stressors such as heavy traffic or interpersonal tension.

The amygdala, a key structure involved in recognising threats and producing emotions such as fear and anger, becomes more active during stress. This increased activation prepares the body for potential danger by raising heart rate, slowing digestion, and causing the skin to pale as blood is redirected to vital organs. At the same time, visual and auditory sensitivity may diminish, and attention narrows sharply towards the stress.

According to Lazarus and Folkman (1984), short-term stress can enhance cognitive performance in military personnel. When faced with a stressful situation, individuals concentrate on overcoming challenges, which can improve composure and focus. This beneficial effect occurs only when the stress arises from a clear and specific challenge. In the face of short-term stress, the prefrontal cortex, i.e., the region of the brain responsible for memory, planning, and thinking, reduces its activity. This area facilitates routine operations and decision-making when conditions are typical. During the "fight or flight" response, however, only the basic mental processes needed to deal with the immediate threat remain active.

While short-term stress can offer temporary cognitive benefits, extended stress carries serious risks. Stress triggers increased cortisol production, which, over time, harms the hippocampus, namely, the brain's centre for memory, information processing, and neurogenesis (the formation of new neurones). The hippocampus manages the flow of information between short-term and long-term memory, deciding what to retain or discard.

Long-term exposure to cortisol can harm the brain and cause memory problems. It initially makes learning new information more difficult and, over time, can also interfere with remembering things already learned. The loss of neurones and reduced brain cell growth further slow thinking and learning (Lazarus and Launier, 1978).

A critical aspect of combat stress is the phenomenon of "amygdala hijack". When the amygdala detects a threat, it temporarily suppresses neural pathways to the prefrontal cortex. This is a valid biological reaction in combat situations since a brief hesitation can be fatal. In cases of chronic PTSD, this leads to a loss of cognitive control over emotions. Recent functional magnetic resonance imaging (fMRI) studies consistently demonstrate that the development of post-traumatic stress disorder (PTSD) is characterised not only by dysfunction within individual brain structures but also by profound disruption of functional connectivity across the amygdala–hippocampus–prefrontal cortex network. Contemporary neuroimaging evidence indicates that the retrieval of trauma-related memories involves widely distributed neural representations. Specifically, impaired top-down regulation by the prefrontal cortex compromises fear extinction, resulting in the persistence of conditioned fear responses. Consequently, trauma-related memories remain embedded within maladaptive neural circuits, contributing to the ongoing maintenance of PTSD symptoms (Rauch, 2021).

Chronic stress and increased cortisol levels may damage dendritic connections that facilitate communication between neurones. The number and integrity of dendrites are also associated with memory capacity and the speed of information processing. A reduction in dendritic complexity is linked to slower and less efficient cognitive functioning. As a result, neural networks become less branched, and brain architecture is simplified. At the molecular level, this structural simplification is largely driven by stress-induced suppression of **brain-derived neurotrophic factor (BDNF)**. Contemporary neurobiological evidence indicates that chronic elevation of cortisol downregulates BDNF expression, thereby impairing synaptic plasticity and reducing hippocampal neurogenesis. This deficit in neurotrophic support contributes to structural alterations within the neural circuits involved in fear regulation. Overall, the pathophysiology of post-traumatic stress disorder (PTSD) is closely associated with these molecular and cellular changes. Clinically, this may manifest as perceptual narrowing (commonly referred to as *tunnel vision*) and a diminished capacity to generate adaptive responses in complex situations (Uno et al., 2024).

In cases of chronic stress, the amygdala is persistently overstimulated and may identify potential risks in every environment despite the absence of objective danger. This results in hyperreactivity, a condition in which even neutral stimuli, such as loud sounds or sudden movements, are interpreted as potential threats. At this stage, a functional dissociation may occur: the prefrontal cortex may recognise the absence of danger, while the amygdala has already initiated a cascade of neuroendocrine responses.

More critically, this state is maintained by sustained amygdala-driven attentional bias towards threat-related stimuli, which reinforces continuous monitoring of the stressor. Khatsaiuk et al. (2021) believe that individuals may find it increasingly difficult to disengage from threat processing and shift attention to everyday activities.

In a healthy state, neural connections are typically protected by the myelin sheath. It provides protection and facilitates the efficient transmission of signals between neurones during thinking and decision-making. Chronic exposure to cortisol may negatively affect myelin integrity and neural efficiency, thereby reducing the effectiveness of information processing. This explains the psychomotor inhibition observed in military personnel under conditions of severe stress.

Emphasis must also be placed on the epigenetic aspect, given that prolonged stress may alter gene expression associated with stress resilience. Therefore the effects of stress may become stabilised at the molecular level without proper rehabilitation.

This may function as a "cognitive prosthesis", whereby the formation of new neural connections around functionally impaired areas supports the restoration of psychological integrity. In addition, sustained intellectual activity may enhance prefrontal regulatory control, gradually reducing amygdala hyperactivity. Mapping these network alterations and BDNF deficits has contributed to a shift in the contemporary clinical paradigm towards targeted therapeutic interventions. Understanding the distributed neural representations underlying PTSD opens new avenues for precision medicine, including non-invasive neurostimulation techniques such as repetitive transcranial magnetic stimulation (rTMS), as well as neurobiologically informed cognitive interventions designed to promote neuroplasticity and support dendritic remodelling (Leuchter & Corlier, 2018).

Consequently, understanding these neuropsychological effects highlights the close link between physiological and psychological responses to stress and offers a foundation for strategies to manage its impact.

4. Key Features of PTSD Development in Military Personnel

Many individuals experience acute stress reactions in response to unusual or intense stimuli. When resources are limited, the natural stress response can become harmful, particularly if it disrupts the hypothalamic–pituitary–adrenal axis and the sympathetic–adrenal system. This disruption can result in PTSD symptoms, increased anxiety, heightened physiological arousal, and hypervigilance.

In cognitive models of PTSD, now supported by neuroimaging studies, the pathogenesis of PTSD is closely linked to disruptions in information processing and the integration of traumatic experiences into one's personal memory (Hinojosa et al., 2024). Conceptually, PTSD can be defined as a failure of adequate information processing. The brain cannot transform chaotic sensory impressions (e.g., sounds of explosions, flashes, smells) into an organised narrative within declarative memory.

Traumatic memories are associated with reduced activity in the left inferior frontal cortex. This indicates that sensory and emotional factors are more prominent than verbal connections. Traumatic experiences are "trapped" within the brain's right hemisphere as visual and somatic sensations without any verbal framework.

Information about the trauma may persist in working memory until it is adequately processed and integrated. Intrusive memories tend to appear and fade gradually over time. Conflicts between attempts to make sense of the experience and pre-existing cognitive schemas can cause avoidance and emotional numbing, increasing the risk of comorbid depression. In such cases, intense emotions may be perceived as threats to the stability of the individual's psychological equilibrium.

This phenomenon is known as "emotional numbing". Affect loses its signalling function, and the brain may begin to interpret any strong emotion, including positive emotions such as joy, as a potential signal of danger. This can contribute to social withdrawal and depressive symptoms.

Persistent avoidance of trauma-related stimuli, along with blocked emotional responses and newly developed numbness (not present before the trauma), may appear in the forms shown in Table 1.

Table 1. Manifestations involving persistent avoidance of trauma-related stimuli, suppressed emotional reactions, and trauma-induced numbness

No.	Manifestation type
1.	Avoidance of thoughts, feelings, or discussions linked to the traumatic event.
2.	Difficulty recalling important details about the trauma.
3.	Lowered emotional responsiveness, for example, finding it hard to feel emotions such as love.
4.	Losing interest in activities that used to feel important or enjoyable.
5.	A sense of having no future perspective, such as a lack of expectations for career, marriage, or long life.

Source: authors' own elaboration

Particular attention should be paid to the transition from a psychogenic disorder to somatic and organic changes. Chronic PTSD in combatants is not a static condition but a dynamic process involving the disruption of neural connectivity. Longitudinal studies of veterans indicate an increased prevalence of organic neurological alterations (Marcolini et al., 2023). Persistent high levels of cortisol are associated with a reduction in hippocampal volume, a brain region responsible for contextual memory. For this reason, PTSD symptoms gradually develop into memory loss and cognitive deterioration, similar to the initial phases of dementia.

In general, PTSD leads to significant emotional disturbances or disruptions in social, professional, and other important aspects of life. Combatants, as a high-risk group for PTSD, often experience changes in the brain due to trauma, which can influence the disorder's progression and long-term outcomes (Nerubasska and Maksymchuk, 2020).

Prospective and retrospective studies of veterans reveal an increasing prevalence of organic disorders within the clinical profile of PTSD. Conditions initially diagnosed in the acute phase as "acute stress reactions", "adjustment disorders", and "post-traumatic stress disorder" may, over time, present with symptom profiles more characteristic of organic pathology. These include personality changes of psychopathic-like features and cognitive decline (Chambliss et al., 2024).

The most common comorbidities of PTSD include depressive episodes, recurrent depressive disorder, panic disorder, generalised anxiety disorder,substance dependence, psychoactive substance (PAS) dependence, social phobia, specific phobia, dissociative disorders, bipolar affective disorder, residual-organic brain diseases, exogenous-organic brain diseases, and schizophrenia.

The high level of comorbidity between PTSD and depressive and anxiety disorders is explained by a shared neurobiological substrate. This includes the dysfunction of monoaminergic systems (serotonin and noradrenaline) and the dysregulation of glutamate. Chronic amygdala hyperactivity depletes the reward system (mesolimbic pathway), leading to depression. This is associated with anhedonia and depressive symptomatology. At the same time, reductions in grey matter volume in the anterior cingulate cortex (ACC) may impair the brain's ability to regulate affect effectively. This contributes to a self-reinforcing cycle of pathological emotional states.

To distinguish between these specific conditions, the most critical biomarker is the ratio of DHEA to cortisol. During constructive stress (eustress), a balanced release of DHEA is observed. This exerts a neuroprotective effect and helps protect hippocampal neurones from the toxic effects of cortisol. During destructive stress (distress), this balance is disrupted. Reduced DHEA levels leave the brain more vulnerable to cortisol-related effects, including dendritic atrophy and cellular apoptosis.

Another marker is the level of brain-derived neurotrophic factor (BDNF). It tends to remain elevated during constructive stress, supporting neuroplasticity. However, it may decrease significantly during the transition to PTSD.

Borderline personality traits (BPT) may develop due to various factors, which are presented in Table 2.

Table 2. Key factors in the development of borderline personality traits (BPT)

No.	Factors leading to BPT development
1.	Direct threats to life: Those who experience combat or other life-threatening events can undergo severe psychological strain and traumatic experiences.
2.	Loss of comrades, friends, or relatives: Such losses can cause intense stress and play a role in the development of combat-related psychological trauma.
3.	Uncertainty and unpredictability: Military conflict situations are often marked by ambiguity and unpredictability, which may promote psychological trauma.
4.	Disruption of safety and trust: When key boundaries are broken, mental well-being suffers, increasing the risk of combat-related trauma.

Source: the authors' own conception

The conceptual approach to rehabilitation should be based on the principle of "reintegration". It is necessary to facilitate the transfer of traumatic experiences from active working memory to long-term autobiographical memory. This process is achieved not only through psychotherapy but also through the restoration of neuroplasticity via the development of new cognitive skills. These skills support the re-establishment of prefrontal regulatory control over amygdala-driven emotional responses.

It is also important to recognise and address issues related to combat-related psychological trauma in a timely manner to ensure proper support and assistance for those affected.

5. Conclusion

This article examines the relevance of studying the cognitive mechanisms underlying combat stress. This need is driven by the requirement to develop methods and technologies to support internal troops, law enforcement officers, and security service personnel involved in military conflicts, as well as civilians who have become victims of combat stress.

Combat stress is a specific type of stress caused by psychological factors in the combat environment. These include prolonged, highly unpredictable, and immediate threats to the life and well-being of the individual and their loved ones, which far exceed everyday challenges. Together, these factors drain psychological resources, cause characteristic temporary or lasting mental changes, and impair social adaptation. Essentially, a new personal narrative emerges (a new biography) with altered frameworks for evaluating the past, present, and future. In response to traumatic events, individuals are forced to adapt to changes that induce stress.

Psychological manifestations of combat stress largely depend on the nature of combat operations, the duration of exposure, and the intensity of fighting. Due to extreme demands, combat stress occurs in most individuals exposed to a combat situation. Factors that heighten the risk of developing combat stress disorder include personal vulnerabilities and low psychological resilience. Under constant tension and alertness, individuals may experience strong mood swings, intense emotional reactions, impulsive anger outbursts, and a reliance on stereotyped behaviours common in combat situations.

A review of current literature on combat stress indicates that research often focuses less on the stress itself and more on its consequences. There is broad agreement regarding the definition of "combat stress": it is a particular form of stress that follows the general principles of the adaptation syndrome. The key distinction is that combat stress is usually a form of distress and is characterised by extremely high, often physiologically significant, intensity. As a result, it is consistently accompanied by various psychological disorders, both pre-pathological and pathological in nature.

Prospects for further research include the study of molecular markers of neuroplasticity, in particular BDNF, in veterans undergoing cognitive rehabilitation. The evaluation of the effectiveness of neuropedagogical methods, such as intensive language learning, is of particular interest. These methods may stimulate neurogenesis and support the functional restoration of connections between the prefrontal cortex and the limbic system during long-term follow-up.

6. Statement on the Use of Artificial Intelligence

The authors state that artificial intelligence tools were employed solely to assist with language refinement and to enhance the academic presentation of the manuscript. Full responsibility for the accuracy of the content, as well as for the interpretations and conclusions, rests entirely with the authors.

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