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Pharmacological Implications of Modifying Abnormal Fear Memory: Aiming to Treat Post-traumatic Stress Disorder

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Editorial

Flashbacks, nightmares and uncontrolled thoughts about the incident that caused the patient's PTSD are the main symptoms of post-traumatic stress disorder (PTSD), a rare clinical mental abnormality. Establishing a thorough treatment plan for patients with PTSD is challenging and complicated since these patients may also struggle with comorbid sadness and anxiety over an extended period of time. The current study examines pharmaceutical interventions used to correct aberrant fear memories. On the basis of efforts to integrate recent clinical and preclinical basic studies, the functions of the central monoaminergic systems, including serotonin, norepinephrine and dopamine, within the fear circuit areas, as well as the involvement of the hypothalamic-pituitary-adrenal (HPA) axis and glucocorticoid receptor (GR), are explored.

Post-traumatic stress disorder (PTSD) is a relatively recent clinical psychiatric diagnosis, although the question of how people react to stressful events is an old one [1]. In an effort to restore combat neurosis into official nomenclature, the American Psychiatric Association (APA) first proposed the term PTSD in the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III) in 1980. Since then, the condition has been extensively studied and acknowledged in ways that go much beyond the definition of combat neurosis. People may display a range of psychological dysfunctions, such as anxiety, sadness, behavioural hyperarousal and intrusive memories of the traumatic experience, even after they are safe. Traumatic experiences have long-lasting effects that are difficult to recover from [2].

The primary approach to treating PTSD is to restore patients' cognitive function; nevertheless, the real-world effectiveness of pharmaceutical therapy is far from adequate, making PTSD one of the most challenging mental conditions to manage. For instance, monoaminergic receptors are the primary target of neurochemical treatments for PTSD symptoms, which have positive effects on reducing anxiety and depression. However, the majority of these treatments are ineffective at addressing the dysfunction of extinction retrieval, which is the pathogenesis of PTSD and contributes to the impairment of fear memory processing. There appears to be a knowledge gap between the effectiveness of pharmaceutical treatments and the underlying causes of PTSD [3].

Three different SSRIs, fluoxetine, paroxetine and sertraline, are suggested as the first-line treatments for PTSD patients in a meta-analysis research published in clinical psychiatry. For many years, SSRIs have been widely used to treat problems of the mood. Central 5-HT systems are therefore heavily responsible for the mood changes brought on by PTSD since the brain areas making up fear circuits (i.e., the prefrontal cortex, hippocampus and amygdala) have a lot of 5-hydroxytryptamine (5-HT) receptors. After stressful situations, this helps to lessen the likelihood of depression and anxiety symptoms.

As a diverse psychiatric condition, depression frequently co-occurs with other mental disorders, such as PTSD. Between 48 and 55 percent of PTSD sufferers either deal with depression right now or have in the past. The likelihood is that SSRIs only treat PTSD-associated depressed moods and not fear symptoms due to the high occurrence of comorbidity. Clarifying the connection between sadness and fear is required in order to investigate this possibility. Lin et al. separated the symptoms of sadness and terror using single prolonged stress (SPS) in a mouse model of PTSD [4].

The pathophysiology of PTSD is influenced by alterations in noradrenaline (or norepinephrine, NE) activity in both the autonomic nervous system (ANS) and the central nervous system (CNS). Patients with PTSD have increased sympathetic nervous system functional activity. Since the locus coeruleus (LC) is the primary location for norepinephrine synthesis in the CNS, the LC-NE system's activity is a possibility for mediating the connection between the ANS and CNS and providing insight into the hyperarousal characteristic of PTSD. Preclinical research has shown that the targets of LC projection and the amount of stress have a significant impact on the central NE's role in fear conditioning and extinction.

The use of antagonists, such propranolol, is thought to be beneficial for the treatment of PTSD. The blood-brain barrier is easily crossed by propranolol, which has positive effects both in the brain's core and on the periphery. The peripheral symptoms of anxiety disorders have long been treated with propranolol. Systematic meta-analyses have shown that propranolol's efficacy is dependent on the time of its delivery. Propranolol does not stop the onset of post-traumatic stress disorder (PTSD) whether given right away or soon after a distressing experience [5].

Conclusion

For NE, receptors aid in the consolidation of contextual traumatic memory and once the painful memories are solidified, it is challenging to get rid of them. B antagonists can't stop PTSD from happening, but they can help with its therapy if it's already happened. The central DA function should consequently be optimised since in individuals with PTSD, the loss of resilience affects the efficiency of fear memory extinction. Last but not least, PTSD develops over time and is influenced by anomalies of the HPA axis. Depending on the timing of the intervention, corticosterone helps people overcome their fears.

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Conflict of Interest

No potential conflict of interest was reported by the authors.

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