

The Role of Encoding and Retrieval of Memories in PTSD: A Brief Overview

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Received: September 21, 2026; **Accepted:** September 25, 2026; **Published:** September 30, 2026

ABSTRACT

Post traumatic stress syndrome is described as a negative experience arising from injury, or being a witness to horrific acts violence, death or due to sexual violence just to name a few Keri. PTSD is not as common as other trauma and is likely to occur in about three percent of the population according to DSM-5 [1]. There are major flaws in processes of encoding, and retrieval of memories brought on by stress-fear response that changes the structure of the amygdala, hippocampus and influences the function of the prefrontal cortex [2]. These changes do not only affect the neurocritical structures but also of the astrocytes responsible for the structure and function of each memory [3].

Keywords: PTSD, Encoding, Retrieval of Memories, Engrams, Astrocytes

Memories are one of the greatest constructs of human cognition, connecting both our past relevant experiences to our present situation and guiding our prospects. However, not all memories are pleasant and as in the case of trauma and specifically PTSD, they can be both daunting and overwhelming. PTSD is based on a negative experience arising from injury, or being a witness to horrific acts violence, death or due to sexual violence just to name a few Keri. PTSD is not as common as other trauma and is likely to occur in about three percent of the population according to DSM-5 [1]. These experiences hinder normal functioning often exhibiting persistent recall and are often associated with intense autonomic reactions during intrusive yet emotional flashbacks and sleep disturbances such as nightmares [12]. Reoccurring flashbacks are activated by an external or internal cue and are based on a select number of scenes hewn together in explicit details [4,5]. Studies indicate that there is a great deal of variation in anecdotal evidence arising from their extremeness in many of the symptoms. This adds legitimacy to the controversy over the feasibility of existing definitions, interpretation and complicates our present understanding about its origin. Furthermore, the lack of an exact model and the limitations of existing models have failed to create significant success in treatment modalities. Stress plays a pivotal role in the many changes that occur in both engrams and cellular functions. To understand what occurs during the initial memory encoding and retrieval, one must take into view the various structures and their relationship with each

other and their overt expression of intrusive experiences. The understanding of these variations is likely to quell some of the dilemmas and to enhance our current understanding prevalent in more readily accepted conventional models.

PTSD can be described as a re-experiencing of a traumatic event whose intensity is significant enough to leave an imprint on the individual of such magnitude that he or she involuntarily becomes preoccupied with reliving this experience repeatedly. Generally, this leads to poor attention span, inability to remember specific details, minimization of specificity of existing autobiographical memory due to ruminations, lower executive functions, and functional avoidance [12]. The stress-related responses from the amygdala and dorsolateral cingulate cortex are not extinguished ventromedial prefrontal cortex. This leads to a diminished ability of safety engrams to inhibit engrams of a threat memory. The hippocampus is responsible for self-referential processes, autobiographical memories, emotional content related to those memories, and cues [6]. It is noted that due to diminished hippocampal volume, Neuroimaging suggests there is diminished and disrupted ability in recall tasks, greater reliance on beliefs to control activity, prediction errors due to avoidance and unwanted intrusions [12]. Furthermore, there is an inability of the dorsolateral prefrontal cortex to divert attention from threat related cues to more positive events resulting from decreased activity due less connectivity was detected in the DMN and the central executive network, while increased activity was noted in the salience network [7]. Recent engram (neural units of memory storage) research has expanded

Citation: Alexander J Shadikhan. The Role of Encoding and Retrieval of Memories in PTSD: A Brief Overview. Open Access J Clin Path Res. 2026. 2(3): 1-2.
DOI: doi.org/10.61440/OAJCPR.2026.v2.47

our detailed understanding of both encoding and retrieval of memories present in PTSD.

Human episodic memories rely on encoding, consolidation, and retrieval of experienced events. The process entails a dynamic interaction between the amygdala, the hippocampus, the prefrontal cortex, and to a certain degree the structures such as the striatum, thalamus, and dorsolateral prefrontal cortex [8]. However, when there is discord in any of the underlying structures, this interaction becomes less than harmonious. Most studies indicate a strong role of stress in retrieval and encoding of PTSD. Stress is a common variable that affects cued recall, whether external or internal, and is responsible for changes in brain structures such as the hippocampus and amygdala. Studies show that those neural networks responsible for encoding are responsible for retrieval [9]. Memory consolidation occurs when short term memory is converted to long term memories. The structural changes often involve a reorganization of the existing engram from a hippocampal to neocortical structures often taking a few days to weeks [2]. These changes also elicit a long-term structural change in the amygdala noted that during fear response results from a conditioned stimulus pathway sensory engram and can activate many of the downstream pathways that had been previously activated by an unconditioned pathway [8]. At the cellular level, plasticity plays a role in adaptation, modification, and retention of memories. There are indications that Plasticity is involved in increased density in dendritic spine as well as higher amplitude leading to a greater strengthening of engrams [8]. The strength of memories relies on plasticity of specific connections, as in CA1 to CA3 engram, as well as with higher AMPA to NMDA ratio in the hippocampus [2]. Most neuroimaging studies have suggested that the size of the hippocampus is smaller than usual in patients and seem to be associated with many sleep issues and declarative memory deficits associated with PTSD [4]. Current research has noted that these changes are associated specifically with glial cells.

Studies have noted anomalies in glial cells, specifically the Astrocytes [3]. Astrocytes are known to be more numerous in the CNS and are responsible for many functions including information processing and neural plasticity. Consequently, it uses cAMP to initiate and enhance the lactate shuttle, serving as energy for the process of neuroplasticity, memory consolidation, and along with LTP to elicit changes in the structure and function of memories. Lab research has found that there is a decrease in density GFAP-positive and vimentin-positive Astrocytes in both the hippocampus and prefrontal cortex, along with a change in synaptic transmission are likely to be responsible cognitive impairment [10]. Sleep disturbance and depression are major factors that play a role in PTSD. Studies also indicate that some of the can be attributed to changes in the hypothalamic-pituitary – adrenal axis (HPA), circadian genes in the brain, neurons and radial astrocytes in stem cells. Even though most research seems to be ongoing, it seems some of the factors involved in depression and mood disorders are closely linked to its pathogenesis of astrocytes.

PTSD remains elusive as far as finding the exact model for its functioning. Thus far it is known that most of the problems arise in the amygdala, hippocampus, and the prefrontal cortex. Driven by stress response, which seems to be the key driving factor responsible for most changes that occur in all three structures. The hippocampus is affected by many changes in the neocortical structures, arising from stress-fear response mechanisms that effect normal functioning and is more than likely responsible for its decreased volume [4]. Morphology of astrocytes indicates a dysregulation and transmission equated with fear-learning due to increased hippocampus interleukin 1-B and inhibition of IL-1 receptors [10]. These changes in turn affect the prefrontal cortex and give rise to intrusions in memory, flashbacks, sleep disturbance, and social function to say the least [11]. Despite these strides in research, many intervening variables remain elusive.

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