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PTSD as Pathologically Anchored Flashbacks: A Mutant Form of Emotional Déjà Vu

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Abstract. This paper puts forward that the PTSD experience, which is often called a traumatic flashback, might be a pathological variant of emotional déjà vu. It is characterized by intrusive memories, which are represented in the form of non-linear, affectively charged, and incoherent with the individual's autobiographical narrative. Borrowing from neurocognitive ideas, including Emotionally Enhanced Memory (EEM) and the Multiple Trace Memory (MTM), we purport that a severe trauma results in the hyper-consolidation of the amygdala-activated fragments owing to the overstimulation of the amygdala and hippocampal dysregulation. These fractions refer to the pathological anchors that take place and the experience woven together like a distorted and high-arousal déjà vu. Additionally, we propose that the DRM paradigm, which has been modified, can produce such flashbacks in an experimental setting. Hence, it should be viewed as a tool to study and possibly reduce PTSD through memory retrieval and reconsolidation.

Keywords: flashbacks, déjà vu, pathological anchoring, emotional memory, DRM paradigm

1. Introduction

When survivors face traumatic experiences, they often experience sudden, intense psychological and physical responses. However, when reminders of the traumatic experience produce severe distress for some people—chronic normal-state responses to trauma—the social and psychological consequences can be devastating and thoroughly inhibit daily functioning [1]. After decades of clinical and scientific research, this group of symptoms was systematically synthesized and formally described in the DSM-5 as a pronounced psychiatric disorder [2]. Post-traumatic stress disorder (PTSD) is a major psychiatric condition, as a substantial part of people exposed to trauma suffer from it. According to the DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition) [2], "development of characteristic symptoms following exposure to one or more traumatic events" is the essential feature of PTSD. The criteria for PTSD can be separated into 8 kinds. To note, the second criterion, also known as criterion B, is Intrusion Symptoms (≥1). More specifically, PTSD is always accompanied by dissociative reactions (e.g., flashbacks).

According to this criterion, one core symptom of PTSD is intrusive flashbacks, which can also be described as a fragmented reproduction of visual, emotional, and somatic sensations [2]. For example, when motor vehicle accident (MVA) survivors with PTSD encounter traffic-related stimuli (e.g., watching traffic, hearing horn sounds) [3], their brains could experience accident-related scene

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results in primal emotions — terror, helplessness, and physiological arousal (tachycardia, sweating). In other words, these “reactions” are understood as reactivation of trauma, and patients touching the trauma-related clue may trigger intrusive memories of the accident and primal emotions. There is a key point that MVA survivors do not directly point to the survivors from the accident; it can also include accident onlookers [4]. The difference between the two may relate to symptom duration, time to resolution, and symptom severity. It could also be explained as the different severity of pathological anchoring. Viewing trauma as an anchor could also explain why the patient is unable to form a new memory circuit. Furthermore, if this anchor is divided into memory fragments, such as sound or heart rate. The trauma can be seen as a mutant kind of emotional déjà vu, which means that the familiar impression of déjà vu is covered with fear or strong emotion. Therefore, there may exist a similarity between déjà vu and PTSD flashbacks.

Déjà vu is a French term. It is used to describe a sense of “already seen” [5]. This term was first used to attempt to further refine the “dreamy states” in epilepsy. Therefore, this experience enables the removal of the hat of “pathological”. “Pathological anchoring” means that a specific sensory detail (e.g., a specific sound, object layout) or emotional state (e.g., terror, helplessness) from trauma becomes an abnormally effective “anchor point”. This anchor may be strongly associated with sensory and emotional fragments. Therefore, when the brain starts to progress in memory retrieval, this “anchor point” cannot be ignored. The “anchor point” could be like a strong magnet, preventing access to the broader, contextualized memory narrative. For example, when MVA survivors with PTSD are watching traffic, they may only recall trauma instead of a happy family period.

“Mutant Emotional Déjà vu” means that while déjà vu is characterized by an intense, irrational feeling of familiarity ("I've experienced this exact moment before"), the “familiarity” may be replaced or covered with strong, severe emotions such as fear and stress. This replacement may turn déjà vu into a mutant kind, that is, emotional flashback. Furthermore, emotional flashback may be accompanied by a pronounced physical response, such as body tremors or a rising heart rate [6]. It should be noted that somatic response may also stimulate emotional response, explained by Dual Representation Theory [7]. DRT points out that Verbally Accessible Memory (VAM) and Situationally Accessible Memory (SAM) are two kinds of memory systems of traumatic experiences. Based on this theory, while emotional flashbacks are triggered, the accompanying somatic responses could further enhance the sense of fear, thereby establishing a vicious cycle of somatic-emotional reinforcement.

2. Challenges in the field

The study of déjà vu began in patients with epilepsy, starting from pathology, but lacking the exploration of transient déjà vu. Though Urquhart et al. [8] simulated transient déjà vu in a laboratory setting by implanting a modified Deese-Roediger-McDermott (DRM) paradigm under fMRI scanning, extant research remains on theoretical explanations about transient déjà vu and lacks explanation about the difference between pathology and transient. The Deese-Roediger-McDermott paradigm is a classic model for investigating repressed memory in which participants have lists of semantically related words presented to them (for example: bed, rest, awake, tired, dream) and are very likely to mistake a non-included critical lure (for example: sleep) for being included in the studied list. This takes place because of the automatic triggering of related ideas within the semantic network. According to Brown [9], approximately 60% population has experienced transient déjà vu. However, this prevalent phenomenon can only be regarded as a clinical manifestation of epileptic pathology.

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This article posits that PTSD is a pathological anchored flashback and can also be a mutant kind of emotional déjà vu, which is that the patient repeatedly experiences a strong but distorted “sense of the past” that they have failed to integrate. This article proposes that PTSD flashbacks are the result of pathological memory anchoring, which is an uncontrolled manifestation of the Emotionally Enhanced Memory (EEM) mechanism under extreme trauma, resulting in the inability of memories to be converted into Multi Trace Memory (MTM) normally, forming a "mutated" emotional déjà vu.

This article tries to connect PTSD and déjà vu in both biological and theoretical sections, then suggests a possible healing method for PTSD based on modified DRM.

3. Neurological similarities

Neuroimaging studies of PTSD patients have been widely discussed. According to Hinojosa et al. [10] and Bremner [11], three sections of the brain have been recognized that have strong relationships with PTSD: the amygdala, the hippocampus, and the prefrontal lobe.

3.1. Amygdala: overactivation in PTSD

Exposed trauma-related clues, such as fearful faces, amygdala activation shows increasing [11] and consistently show heightened amygdala reactivity during threat–related tasks [10]. According to Bremner [11], stronger amygdala activation correlated with more severe PTSD symptoms (e.g., intrusive memories, hyperarousal). The amygdala is generally responsible for emotion control, especially in response to fear or terror. The overactivation in PTSD could be explained by disability of emotion regulation.

3.2. Hippocampus: structural atrophy and functional hypoactivity

As Bremner [11] argues, PTSD patients often show smaller hippocampal volumes (8–12% reductions in Vietnam veterans, childhood abuse survivors, and combat PTSD). PTSD patients display reduced hippocampal activation during fear inhibition, memory, and emotion regulation tasks [10].

Hippocampus is typically referred to as the memory integration responsible for consolidating multisensory memory components. This reaction is seen in that memory fragments failed to integrate.

3.3. Prefrontal lobe: decreases the connection with the amygdala and the hippocampus

Under trauma stress, the prefrontal lobe (particularly the mPFC area) shows decreasing activation. This reaction also appears in the cortex that connects the prefrontal lobe with the hippocampus and amygdala. The prefrontal lobe is generally regarded as the central hub for memory encoding, which determines whether the memory has occurred before, and it could further emotion (connected to the amygdala). The weakened connection between areas is seen as a regulatory failure.

In other words, if déjà vu is related to these brain regions, it could partly suggest that déjà vu is an aggregate of memories and emotions and is a mutant kind of flashback.

Current research lacks direct neuroimaging of transient déjà vu; two research avenues are being proposed: one investigates eliciting déjà vu experiences through electrical stimulation of specific brain regions in epileptic patients, while the other implants a modified DRM paradigm to experimentally simulate déjà vu experience in laboratory settings.

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Research shows that the Medial Temporal Lobe (MTL) is strongly associated with the simulation of the déjà vu experience. The entorhinal cortex is regarded as a part of the hippocampus that integrates memory fragments. According to Bancaud et al. [12], the entorhinal cortex exhibits a high susceptibility to electrical stimulation in experiments simulating déjà vu experiences. Moreover, the hippocampus demonstrates lower susceptibility, while the amygdala exhibits an even lesser degree. The entorhinal cortex always serves as the primary interface for both input to and output from the hippocampus [13]. In this situation, experiences have led to highly sensitive cellular mechanisms for information processing, as explained by Grid Cell Theory. As the amygdala is often responsible for fear or other strong emotions, familiarity impression, the crucial component of déjà vu, may not stimulate a strong response in it.

According to Urquhart et al. [8], after modified DRM, functional magnetic resonance imaging(fMRI) neuroscanning demonstrates that déjà vu shows a relationship with the prefrontal lobe. This relationship suggests that memory conflict is a necessary component of déjà vu.

In summary, both PTSD and déjà vu are related to MTL and the prefrontal lobe. The difference is the response strength of each region.

4. EEM in overdrive: the genesis of pathological anchors

Emotionally Enhanced Memory (EEM) describes the storage prioritization of emotional memories. According to Nadarevic [14], people remember negative words better than neutral words. Under extreme trauma, however, this memory storage mechanism may be overactivated.

4.1. Traumatic EEM dynamics

The amygdala overactivation may highly bundle multisensory memory fragments (e.g., the sound of car honking in MVAs) with highlighted emotions (e.g., fear or helplessness). According to Lupien et al. [15], acute stress alters the functional connections of the prefrontal, amygdala, and hippocampus, causing the amygdala to occupy the body of thought patterns rather than the hippocampus. Therefore, the spatial coding capacity of the hippocampus is restrained under extreme trauma pressure.

4.2. Birth of the anchor

A part of these or one of these salient memory fragments may become a pathological attracting point. Due to their salience and lack of context binding, these points operate as individual anchors and are readily activated by associated clues [15].

As for déjà vu, EEM could explain why experiencers often cannot track the origin of memory fragments. The emotions associated with déjà vu are neural or low-severity. Lupien et al [15]. propose that the inverted-U-shaped relationship governs the connection between glucocorticoids and memory. As the key brain regions influenced by glucocorticoids, the hippocampus and the amygdala may exhibit the same inverted-U-shaped relationship in regulating memory and emotion. Therefore, in a low-glucocorticoid emotional state, memory encoding may lack context binding and result in a memory fragment that lacks spatial context. Through the different emotion memory storage processes, both PTSD and déjà vu are related to wandering memory fragments.

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5. MTM collapse: the failure to transcend fragmentation

The Multiple Trace Theory (MTT) posits that the encoding and extraction of memory are all reliable to the hippocampus. The hippocampus's role is to transform temporary memory into long-term cortical memory. PTSD is a pathological arrest in this locus.

According to Nadel & Moscovitch [16], because traumatic fragments lack contextual integration, they remain hippocampally tethered. Each reactivation keeps the raw fragments rather than an updated representation. As for déjà vu, ordinary déjà vu can be caused by a temporary malfunction of the hippocampal system, which fails to adequately retrieve the spatio-temporal context of the memory trace that is only partially activated by the current cue. Consequently, individuals experience a strong sense of familiarity without knowing the source of the stimulus that triggered this sense of familiarity. The following table describes the comparison between ordinary déjà vu and PTSD flashbacks.

Table 1. Comparison of Neurocognitive Profiles Between Ordinary Déjà Vu and PTSD Flashbacks

Feature	Ordinary Déjà Vu (within MTT Framework)	PTSD Flashback (within the Pathological Déjà Vu Framework)
Trigger Mechanism	Partial feature matching	Pathological anchoring
Hippocampal Function	Temporary, minor binding failure	Persistent, severe functional inhibition
Amygdala Involvement	Minimal or mild involvement	Hyperactivation
Memory Content	Vague sense of familiarity	Specific sensory-emotional fragments
Temporal Perception	Awareness of present reality	Temporal collapse (“nowness”)
Prefrontal Function	The monitoring system corrects the error	Dysregulated function (reduced vmPFC activity)
Nature	Harmless, transient cognitive glitch	Distressing, intrusive pathological state

Note. MTT = Memory Trace Theory; vmPFC = ventromedial prefrontal cortex.

6. Modified DRM

Based on the theory that explains the correlation between déjà vu and PTSD, one possible method that could be used to simulate PTSD flashbacks is. According to Urquhart et al. (2021), modified DRM could partly simulate transient déjà vu in laboratory settings. This paper also discusses the relationship between déjà vu and PTSD. The following are the corresponding reactions in the theoretical experiment explanation:

1) The DRM task semantic network, comprised of the words used, is similar to the traumatic memory network, which is organized around a harsh, forbidden anchor.

2) The critical lure is the anchor point declared traumatic, which is the very salient, undeclared thought at the core of the network.

3) The critical lure, which is falsely recognized with a high rate, reflects the intrusive and irresistible nature of the pathological anchor, which emerges into consciousness regardless of the current situation (that is, it was not included in the list).

4) The accuracy and vividness of memory regarding false recognition match the experience of the patient during his or her flashbacks.

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Therefore, after implementing trauma-related lures. Modified DRM could also partly simulate the PTSD flashback experience in a controlled environment. This method allows for the translation of findings from laboratory simulation to pathological simulation. Moreover, compared to modified DRM, Urquhart et al [8]. suggested that this method may enhance the correlation between lures and participants. This correlation could better simulate the familiarity impression of déjà vu, instead of focusing on creating memory conflict. However, it should be noted that it is a broad range of trauma-related lures, which is difficult to specify for each patient's trauma. There may be two methods to solve this problem. One is recruiting a similar kind of trauma patient (e.g., MVAs). In this way, the trauma-related word could effectively be determined and incorporated into the lures. However, if trauma is highly idiosyncratic or detailed, this approach may not be optimally effective. Considering this limitation, another method is the construction of personalized word lists for each identified anchor (e.g., crushing). This experiment could be processed under an fMRI scanner to capture the neural signature of anchor activation. As a result, I could predict that the prefrontal lobe and the hippocampus may show the same activation as transient déjà vu. But compared to transient déjà vu, the amygdala may demonstrate a more pronounced response.

Through improving the modified DRM, it may enhance the metamemory monitoring ability of the prefrontal lobe to trauma-related clues. Based on MTM, this method may create a new memory circuit and propose a possible method to alleviate PTSD symptoms.

7. Conclusion

The essay has suggested a framework of conceptual and neurocognitive relationships between PTSD flashbacks and déjà vu, putting the former as a disease condition having a modified form of the latter. While both refer to the presence of impaired and involuntary memory inputs, which are lacking contextual and comprehensive integration, they are opposite in terms of valence, intensity, and impact, respectively. The conjunction of medial temporal lobe anatomy, notably comprising the hippocampus and entorhinal area, along with the prefrontal regions responsible for their control, provides evidence that the cases of déjà vu are more or less similar to flashbacks and failures of memory retrieval. Yet, in PTSD, that means of hyperactivity of the amygdala and the prominent dysregulation of the prefrontal cortex push a normal mental error into a traumatic, painful recurrence of the past. If the adapted DRM paradigm can be sufficiently developed to allow research into these intrusions in a controlled setting, it has the potential to close the gap between theoretical laboratory models and actual clinical practice. Future studies should use functional neuroimaging to determine whether the memory recall associated with transient déjà vu and PTSD flashbacks shows distinct neural correlates and/or involves different cognitive processes. Quizzically, such research might use personalized trauma cues that present as stressors within the scanning protocols. Such studies will help to verify the proposed conceptual model and offer new avenues for curiosity that would help the development of therapeutic strategies that would work on the recontextualization of distressing memory anchoring points.

References

[1] Yehuda, R., Hoge, C. W., McFarlane, A. C., Vermetten, E., Lanius, R. A., Nievergelt, C. M., Hobfoll, S. E., Koenen, K. C., Neylan, T. C., & Hyman, S. E. (2015). Post-traumatic stress disorder. Nature Reviews. Disease Primers, 1(1), 15057-. https: //doi.org/10.1038/nrdp.2015.57

[2] American Psychiatric Association. (2022). Diagnostic and statistical manual of mental disorders (5th ed., text rev.). https: //doi.org/10.1176/appi.books.9780890425787

DOI: 10.54254/2753-7064/2025.HT28851

[3] Baker, A. S., Litwack, S. D., Clapp, J. D., Gayle Beck, J., & Sloan, D. M. (2014). The Driving Behavior Survey as a Measure of Behavioral Stress Responses to MVA-Related PTSD. Behavior Therapy, 45(3), 444–453. https: //doi.org/10.1016/j.beth.2014.02.005
[4] Injury Matters. (2021). Witnessing road trauma. Retrieved August 17, 2025, from https: //www.injurymatters.org.au/wp-content/uploads/2021/05/Witnessing-Road-Trauma-2pp.pdf
[5] O'Connor, A. R., Wells, C., & Moulin, C. J. A. (2021). Déjà vu and other dissociative states in memory. Memory, 29(7), 835–842. https: //doi.org/10.1080/09658211.2021.1911197
[6] Kearney, B. E., & Lanius, R. A. (2022). The brain-body disconnect: A somatic sensory basis for trauma-related disorders. Frontiers in Neuroscience, 16, 1015749. https: //doi.org/10.3389/fnins.2022.1015749
[7] Brewin, C. R., Dalgleish, T., & Joseph, S. (1996). A dual representation theory of posttraumatic stress disorder. Psychological Review, 103(4), 670–686. https: //doi.org/10.1037/0033-295X.103.4.670
[8] Urquhart, J. A., Sivakumaran, M. H., Macfarlane, J. A., & O'Connor, A. R. (2021). fMRI evidence supporting the role of memory conflict in the déjà vu experience. Memory, 29(7), 921–932. https: //doi.org/10.1080/09658211.2018.1524496
[9] Brown, A. S. (2003). A review of the déjà vu experience. Psychological Bulletin, 129(3), 394–413. https: //doi.org/10.1037/0033-2909.129.3.394
[10] Hinojosa, C. A., George, G. C., & Ben-Zion, Z. (2024). Neuroimaging of posttraumatic stress disorder in adults and youth: Progress over the last decade on three leading questions of the field. Molecular Psychiatry, 29(10), 3223–3244. https: //doi.org/10.1038/s41380-024-02558-w
[11] Bremner, J. D. (2007). Neuroimaging in Posttraumatic Stress Disorder and Other Stress-related Disorders. Neuroimaging Clinics of North America, 17(4), 523–ix. https: //doi.org/10.1016/j.nic.2007.07.003
[12] Bancaud, J., Brunet-Bourgin, F., Chauvel, P., & Halgren, E. (1994). Anatomical origin of déjà vu and vivid 'memories' in human temporal lobe epilepsy. Brain (London, England : 1878), 117(1), 71–90. https: //doi.org/10.1093/brain/117.1.71
[13] Hafting, T., Fyhn, M., Molden, S., Moser, M.-B., & Moser, E. I. (2005). Microstructure of a spatial map in the entorhinal cortex. Nature (London), 436(7052), 801–806. https: //doi.org/10.1038/nature03721
[14] Nadarevic, L. (2017). Emotionally enhanced memory for negatively arousing words: Storage or retrieval advantage? Cognition and Emotion, 31(8), 1557–1570. https: //doi.org/10.1080/02699931.2016.1242477
[15] Lupien, S. J., Maheu, F., Tu, M., Fiocco, A., & Schramek, T. E. (2007). The effects of stress and stress hormones on human cognition: Implications for the field of brain and cognition. Brain and Cognition, 65(3), 209–237. https: //doi.org/10.1016/j.bandc.2007.02.007
[16] Nadel, L., & Moscovitch, M. (1997). Memory consolidation, retrograde amnesia, and the hippocampal complex. Current Opinion in Neurobiology, 7(2), 217–227. https: //doi.org/10.1016/S0959-4388(97)80010-4