SECTION 1. GENERAL PSYCHOLOGY AND PSYCHOLOGY OF PERSONALITY

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A THEORETICAL MODEL OF MODIFYING TRAUMATIC MEMORIES VIA VIRTUAL REALITY THROUGH THE MEMORY RECONSOLIDATION PROCESS

ТЕОРЕТИЧНА МОДЕЛЬ МЕХАНІЗМУ МОДИФІКАЦІЇ ТРАВМАТИЧНИХ СПОГАДІВ ЗА ДОПОМОГОЮ ВІРТУАЛЬНОЇ РЕАЛЬНОСТІ ЧЕРЕЗ ПРОЦЕС РЕКОНСОЛІДАЦІЇ

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With the rising prevalence of post-traumatic stress disorder (PTSD) among military personnel, refugees, and survivors of violence, scientifically grounded approaches to modifying traumatic memories are urgently needed. Cognitive-behavioral therapy (CBT) and eye movement desensitization and reprocessing (EMDR) are insufficient for a subset of patients due to their limited impact on neuroplastic mechanisms, which, in turn, strengthens interest in virtual reality (VR) as an adaptive, personalized, and neurobiologically grounded tool. A particularly promising mechanism is reconsolidation-the return of a stabilized memory to a labile state following reactivation, creating an opportunity for change.

Theory and experimental work support the efficacy of targeted interventions during this time-limited window for weakening pathological memories, including in PTSD. Clinical constraints are linked to the difficulty of precisely controlling the reactivation context and the conditions required for destabilization. VR can create controlled, sensorily rich environments for reactivation and destabilization, followed by targeted influence on the structure of the memory trace, thereby motivating the development of an

integrated model that modifies its affective, sensory, and cognitive-components.

In mice, pairing a familiar object with a novel one destabilizes memory via NF-κB signaling in the hippocampus, confirming the role of a mismatch between expectations and new information as a trigger [1]. The process depends on plasticity involving NMDA receptors, β-adrenergic pathways, and protein synthesis. Blocking GluN2B-NMDA receptors in CA1 after reactivation completely disrupts fear reconsolidation. This effect is abolished by D-serine. Inhibition of Wnt/β-catenin signaling with DKK1 likewise disrupts the process, which is restored by NMDA-receptor activation [5]. Activation of ASIC1a increases fear lability after reactivation-evidence of synapse-specific modulation in the amygdala [3]. At the protein-synthetic level, FTO-mediatedmRNA demethylation in the hippocampus regulates BDNF expression during reconsolidation of object recognition, inhibiting FTO disrupts the process, and a TrkB agonist negates this effect, underscoring a critical epigenetic neurotrophic control of stabilizing the updated trace [2].

Reconsolidation is dynamic and temporally constrained, and it is sensitive to precise neuronal, synaptic, and molecular conditions, therefore, effective intervention requires knowing when and how to act. Traumatic memories are cognitive-affective structures characterized by fragmented sensory traces, intense emotions, and reactive response patterns. Their persistence is explained by the emotional-tag hypothesis, in which emotional valence-rather than cognitive richness-drives reactivation and is linked to impaired predictive control in PTSD [4]. The key to opening the labile window is prediction error-a mismatch between expected and actual outcomes-which triggers plasticity in the amygdala and updating of the stabilized trace [6].

Owing to its controlled context, controlled cue set, and sensory dynamics, VR is an optimal platform for inducing prediction error and affective interference. We present a VR-intervention model that deliberately rewrites the traumatic trace via reconsolidation (see Fig. 1).

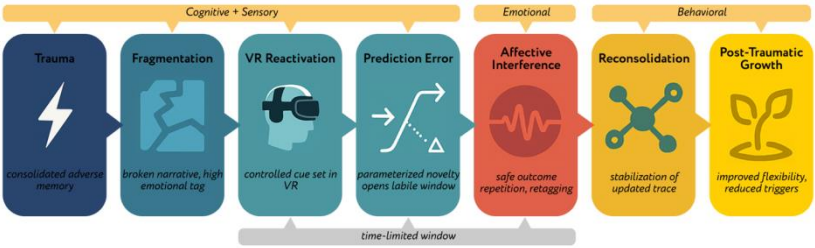

Fig. 1. Model of the mechanism for modifying traumatic memories with VR through the reconsolidation process

VR precisely parameterizes reactivation and elicits prediction error by combining familiar elements with dosed novelty, thereby amplifying attention and expectation incongruence. The transition from a stabilized to a labile state is determined by the magnitude and temporal profile of the error. The anticipated threat is substituted with a safe or neutral-positive outcome, and replacement of key objects shifts representations within hippocampo-amygdalar and fronto-parietal networks. The reconsolidation «gates» are supported by GluN2B-NMDA signaling in CA1, β-adrenergicmodulation, and Wnt/β-catenin pathways, their blockade narrows the plasticity window, whereas restoration of NMDAtransmission returns the trace to a state amenable to rewriting.

The affective component is modulated by the amygdala, ASIC1a activation enhances labilization, the dorsal raphe nucleus influences the stability of contextual traces. The epigenetic FTO→BDNF/TrkB contour provides protein-synthetic support for consolidating the changes. Operationally, within the active window, the VR intervention combines affective interference and cognitive reconstruction. The former repeatedly disconfirms catastrophic expectations under safe conditions, the latter retunes the semantic and causal links of the episode, with correspondences at the level of the posterior hippocampus and prefrontal control systems. The traumatic trace is viewed as a cognitive-affective composition with an emotional tag serving as a rapid-access index. The aim of the intervention is not amnesia but reducing the strength of the tag and retuning the predictive threat model, thereby lowering the probability and vividness of unwanted reactivations and increasing behavioral flexibility.

The model explicitly respects strict constraints of time and intensity. Interference delivered before a sufficient prediction error has formed, or outside the reconsolidation window, does not yield a durable rewrite, excessive sensory load in VR reduces the likelihood of labilization. Accordingly, the operative contour is: reactivation → prediction error → molecular-synaptic labilization → VR interference → consignation (stabilization) supported by NMDA transmission, β-adrenergic modulation, and Wnt/β-catenin signaling, together with the epigenetic FTO→BDNF/TrkB axis. VR serves as a tool for fine-tuning the parameters that open and maintain the reconsolidation «gates» purposefully weakening the emotional tag and re-specifying the associated behavioral responses.

Reconsolidation is a reliable basis for modifying traumatic memories, yet it requires precise entry into a narrow window of neuroplasticity. The critical condition for opening this window is prediction error, which shifts the stabilized trace into a labile state via GluN2B-NMDA, β-adrenergic modulation, Wnt/β-catenin signaling, and the epigenetic FTO→BDNF/TrkB contour. The proposed VR model combines controlled reactivation, dosed

induction of prediction error, and subsequent affective and cognitive interference, ensuring precise tuning of context, sensory dynamics, and scenario logic, the central target is the emotional tag. The model opens a path to personalized VR protocols, priorities for future work include empirical verification of the stages, optimization of VR parameters, and integration of individual neuropsychological profiles into intervention design.

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