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Memory Reconsolidation and Fear Extinction as Treatment of Trauma and PTSD: A Review

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ABSTRACT

Intrusive and persistent fear memories are a defining feature of post-traumatic stress disorder (PTSD). Two processes have gained attention as alternatives to traditional therapies: memory reconsolidation, in which a recalled memory enters a labile state and can be modified, and fear extinction, in which new safety learning suppresses the original trace. This review examines eleven recent studies addressing both mechanisms and their clinical applications. Reconsolidation depends on protein synthesis, prediction error, and hippocampal-prefrontal coordination, and can be disrupted through pharmacological agents such as propranolol, rapamycin, and NMDA modulators, as well as neuromodulation. Extinction instead relies on building safety associations and is influenced by adrenergic state, sleep quality, and the salience of conditioned cues. Clinically, reconsolidation-based methods, including Reconsolidation of Traumatic Memories therapy and propranolol reactivation, show promise in weakening traumatic recall, while extinction-based approaches remain central to exposure therapies but often lead to relapse because patients must repeatedly face trauma. Together, the evidence suggests reconsolidation may allow more lasting change, whereas extinction provides reliable but fragile benefits. Future research should clarify boundary conditions and test integrated approaches to develop more durable, patient-centered PTSD treatments.

Keywords: *Memory Reconsolidation, Reconsolidation, Fear Extinction, Trauma, PTSD, Post-Traumatic Stress Disorder, Propranolol, Reconsolidation of Traumatic Memories (RTM) therapy, Exposure therapy*

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INTRODUCTION

Traumatic events like violence, abuse, or serious accidents can have a long-lasting impact on a person's thoughts, emotions, and behavior. When these effects persist over time and begin to interfere with daily functioning, they may develop into post-traumatic stress disorder (PTSD). In the United States, an estimated 6% of adults will experience PTSD at some point in their lives, and about 5% are affected in any given year. Veterans, particularly those who have served in combat zones, face a significantly higher risk, and women are more likely than men to develop PTSD due to increased exposure to certain traumatic events, such as sexual assault (U.S. Department of Veterans Affairs, 2025b).

Globally, approximately 5.6% of the world's population, around 400 million people, will experience a traumatic event and develop PTSD at some point, while an estimated 3.9% are currently living with the condition. Individuals with PTSD may experience intrusive recollections of the trauma, hyperarousal, emotional numbness, and avoidance behavior, all of which can cause significant distress and disrupt daily life (World Health Organization, 2024).

Sherin and Nemeroff (2011), using neuroimaging, showed that PTSD alters the fear and memory center structure and function. The amygdala overactivates, causing fear responses, while the prefrontal cortex underactivates, limiting its regulation. Hippocampal volume and function often decrease, causing fragmented memories and trouble distinguishing past threat from present safety. These changes explain PTSD symptoms and emphasize the need for memory-targeted treatments like reconsolidation and extinction.

Traditional treatments for PTSD include cognitive behavioral therapy (CBT), prolonged exposure (PE), and Eye Movement Desensitization and Reprocessing (EMDR). These treatments can significantly reduce symptom severity; however, they often require long-term commitment and repeated confrontation with traumatic memories, which can contribute to high drop-out rates and, in some cases, exacerbate distress (U.S. Department of Veterans Affairs, 2025a). Many patients experience only partial recovery or relapse following the cessation of treatment. These limitations have led to a growing interest in therapeutic strategies that directly target the traumatic memory itself, rather than focusing solely on symptom management. Two such methods, grounded in neuroscience, are fear extinction and memory reconsolidation, both of which offer potential pathways to reduce the emotional weight and accessibility of traumatic memories (Ecker & Vaz, 2022; Pace-Schott et al., 2023).

Chen et al. (2025) describe the two memory processes. Fear extinction is a form of inhibitory learning in which repeated exposure to trauma-related cues in a safe context weakens the conditioned fear response, facilitating the formation of new, non-threatening associations that compete with the original fear memory. Memory reconsolidation occurs when a previously consolidated memory is reactivated and temporarily enters a

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labile state, during which it can be disrupted and modified before being restored. This process provides a window for altering the traumatic memory itself, potentially leading to longer-lasting therapeutic effects.

This literature review examines fear extinction and memory reconsolidation as neuroscience-based interventions for trauma and PTSD. By analyzing their underlying mechanisms, exploring their augmentation through pharmacological agents, and evaluating their limitations, this paper aims to assess whether either process offers particular promise for improving trauma outcomes and reducing the lasting impact of traumatic experiences.

METHODOLOGY

To conduct this literature review, peer-reviewed research articles that discuss the neurological mechanisms and therapeutic applications of memory reconsolidation and fear extinction concerning trauma or post-traumatic stress disorder (PTSD) were collected from the academic databases PubMed and ScienceDirect. A range of search terms, which were "reconsolidation" or "memory reconsolidation", "trauma" or "traumatic", "PTSD" or "Post-Traumatic Stress Disorder", and "fear extinction", were used in various combinations depending on the focus of each search. The Boolean operator "AND" was used occasionally to refine results, especially when searching for articles focusing on the use of memory reconsolidation and fear extinction in treatment for trauma or PTSD. All papers were found through the "Open access & Open archive" search filter. The combinations of keywords used were "Memory Reconsolidation" with 3 articles found, "Reconsolidation AND PTSD" with 4 articles found, "'Fear Extinction' Trauma" with 2 found, "Reconsolidation" with one found, and "Reconsolidation Extinction PTSD" with one found. In total, 11 articles were found. Both foundational neuroscience papers and more recent studies on medical applications were included. The priority was to use articles published within the last 5 years (2020-2025), but one document from outside that time frame (2018) was included.

The selected articles were organized into mechanistic studies, which examine the molecular and neural bases of reconsolidation and extinction, and clinical studies, which test these processes as potential interventions for PTSD. Within the clinical studies, some also included pharmacological components (e.g., propranolol, rapamycin, NMDA modulators), which were noted as such. Each article was evaluated based on its relevance to the central research question of how reconsolidation-based and extinction-based approaches compare as possible treatments for traumatic memories, with priority given to studies that addressed neural pathways, clinical feasibility, and treatment outcomes.

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RESULTS

This review synthesizes findings from eleven primary research articles that address both the mechanisms and clinical applications of memory reconsolidation and fear extinction. Neuromodulation research in humans, clinical trials in PTSD patients, and rodent molecular and systems-level experiments are among the chosen studies. When combined, they demonstrate that extinction and reconsolidation are two distinct yet related processes, each with specific treatment implications for trauma. With an emphasis on brain circuits, boundary conditions, and molecular signaling, the first section explores the fundamental neuroscience of reconsolidation and extinction. Beginning with reconsolidation-based interventions, such as propranolol reactivation and Reconsolidation of Traumatic Memories therapy (RTM), the following sections assess how these processes have been addressed in clinical settings. From there, they shift to extinction-based tactics like pharmacological facilitation, sleep modulation, and stimulus salience.

MECHANISMS OF RECONSOLIDATION AND FEAR EXTINCTION

Memory Reconsolidation Mechanisms

One of the main triggers of reconsolidation is a phenomenon known as prediction error, which occurs when the recalled memory does not align with what actually happened. Fukushima et al. (2021) tested this process in mice using an inhibitory avoidance task. After an initial round of inhibitory avoidance training, in which the mice learned to associate a specific chamber with a foot shock, they were briefly re-exposed to that shock-associated context. Under these conditions, molecular markers of reconsolidation appeared, such as phosphorylation of cAMP response element-binding protein (CREB). Longer re-exposures, however, led to increased extracellular signal-regulated kinase (ERK) activity, signaling the onset of extinction learning instead. Interestingly, a very short re-exposure of just one minute produced neither reconsolidation nor extinction. Yet, when ERK was inhibited in this condition, reconsolidation was restored. These findings suggest that ERK functions as a molecular switch, controlling whether a fear memory destabilizes or transitions towards extinction.

Protein synthesis has also been shown to play a critical role in reconsolidation. In a series of rodent experiments, MacCallum et al. (2024) administered rapamycin, a well-established inhibitor of the mechanistic target of rapamycin complex 1 (mTORC1), a pathway critical for initiating protein translation in neurons, immediately after memory reactivation. Because mTOR signaling is known to regulate the cellular processes underlying long-term memory storage, blocking it tests whether reconsolidation depends on new protein synthesis. They found that fear memory weakened even when rapamycin was given several hours later, indicating that reconsolidation requires multiple waves of protein translation across time. Repeated doses following reactivation made the memory especially fragile and resistant to reinstatement. Notably, rapamycin did not

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interfere with subsequent learning, highlighting that its effects were specific to the destabilized trace rather than the general cognitive process.

Pharmacological approaches further illustrate how reconsolidation can be manipulated step by step. In mice, Ahamed et al. (2025) first administered D-cycloserine, a partial N-methyl-D-aspartate (NMDA) receptor agonist, to increase the chance of destabilization. This was followed by MK801, an NMDA antagonist, to block restabilization. Administered separately, the drugs had limited effects, but in combination they sharply reduced both ERK activity in the amygdala and freezing behavior. This two-stage approach demonstrates how reconsolidation can be deliberately opened and then interrupted, leaving the memory trace in an unstable state that ultimately weakens it.

At a broader level, reconsolidation depends on coordinated activity across brain regions, not just molecular switches. Radiske et al. (2025) recorded local field potentials in rats and found that theta synchrony between the dorsal hippocampal CA1 region and the prelimbic cortex increased during reactivation, but only when reconsolidation actually occurred. Silencing hippocampal-prefrontal communication during this window blocked reconsolidation and caused amnesia. On the flip side, stronger theta coherence predicted stronger retention, suggesting that synchrony may not only be required for reconsolidation but could also serve as a marker of its occurrence.

Human evidence supports this view. Su et al. (2022) tested whether non-invasive stimulation could interfere with reconsolidation. Participants first learned a conditioned fear response and then, immediately after reactivation, received continuous theta-burst stimulation (cTBS) over the right dorsolateral prefrontal cortex (dlPFC). Only those stimulated within minutes of retrieval showed reduced differential skin conductance responses and failed to reinstate fear later. If stimulation was delayed by six hours, the effect disappeared. These results underline the time sensitivity of reconsolidation and highlight the essential role of prefrontal circuits in sustaining fear memories.

Across these studies, reconsolidation emerges as a protein-synthesis-dependent process, triggered by prediction error and dependent on ERK, mTOR, and NMDA signaling. It also relies on coordinated hippocampal-prefrontal activity. Interventions that interrupt any of these steps, whether through drugs or neuromodulation, can prevent the memory from stabilizing and lead to long-lasting reductions in fear.

Fear Extinction Mechanisms

Extinction, by contrast, takes a different route than reconsolidation. Instead of reopening the old trace, extinction creates a new memory in which the conditioned stimulus is linked with safety. This new association competes with the old one, which is why relapse often happens through renewal or reinstatement (Burhans et

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al., 2018). Even so, extinction remains at the center of PTSD therapies because it can reduce symptoms even if the traumatic memory is still there.

Several things shape how effective extinction is. Burhans et al. (2018) tested propranolol in a rabbit eyeblink conditioning model. Rabbits that received the drug before extinction training showed faster decreases in conditioned responses during the session. That suggests blocking adrenergic arousal makes it easier to learn that the conditioned stimulus no longer predicts the aversive event. But the advantage faded quickly. On later tests, the propranolol group looked the same as the controls. The finding highlights extinction's main weakness: it works in the short term, but the original fear memory is still intact.

Physiology after training also matters a lot. Pace-Schott et al. (2023) reviewed evidence that rapid eye movement (REM) sleep helps consolidate extinction recall and supports the generalization of safety learning. In PTSD, REM is often fragmented, and elevated arousal during sleep undermines this process. Experimental findings suggest that napping after extinction strengthens learning, while poor sleep weakens it. This means extinction is not just about what happens in training but also about the state the brain is in afterwards.

The type of conditioned stimulus also makes a difference. Eales et al. (2025) compared the extinction of a standard conditioned stimulus (CS+) with a stimulus presented at the same time as the unconditioned stimulus, known as CSX. Extinguishing CSX, which was more directly tied to the aversive event, produced more durable effects. Participants who extinguished CSX did not show relapse, while those who extinguished CS+ did. This result matches with cognitive models of PTSD that argue treatment should focus on trauma-proximal cues rather than less central ones.

CLINICAL STUDIES TARGETING MEMORY RECONSOLIDATION IN PTSD

Findings from animal models and laboratory studies naturally raise the question of whether reconsolidation can be used in the clinic. The possibility is appealing. If a traumatic memory can be reactivated and then disrupted during its unstable period, the grip of the memory may be weakened or even lost. Clinical researchers have explored this idea using pharmacological interventions, behavioral methods, and non-invasive brain stimulation. Results are uneven, but they collectively show both the promise and the difficulties of applying reconsolidation in real patients.

Propranolol and Pharmacological Approaches

The beta-blocker propranolol has been tested more than any other drug for reconsolidation-based treatment. The logic is simple: noradrenaline strengthens emotional memories, so blocking adrenergic activity

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during reconsolidation might blunt their impact. Early reports looked encouraging, although subsequent trials have been mixed.

Taïb and colleagues (2025) conducted a six-week trial with forty-six PTSD patients. Participants were asked to write and then read aloud trauma narratives, which served as reactivation sessions. Ninety minutes before each session, they received either propranolol or a placebo. Both groups showed clear improvement, with PTSD and depression scores declining across the trial. Yet propranolol did not outperform placebo. What changed, in both groups, were qualities of the traumatic memories themselves. Emotional intensity and the sense of "nowness" decreased, and intrusive rehearsals became less frequent. These changes were strongly linked to reduced PTSD severity.

This trial fits with a wider pattern in the literature. Meta-analyses generally report modest and inconsistent benefits of propranolol. One interpretation is that the reactivation procedure itself carries much of the therapeutic value. Revisiting a trauma narrative repeatedly may destabilize and reshape the memory even in the absence of medication. Propranolol may still have an effect in certain contexts, such as when arousal is high or prediction error is strong, but its added value appears limited.

Behavioral Reconsolidation-Based Interventions

Because drugs have not produced consistent gains, researchers have turned to purely behavioral strategies. One approach is brief imaginal exposure. Patients recall their trauma and then undergo short sessions of re-experiencing timed to overlap with the reconsolidation window. Early trials suggest that just two sessions may reduce symptoms more effectively than exposure delivered outside the window. The efficiency of this approach is striking, since standard prolonged exposure requires many more sessions and is often associated with dropout.

A different example is Reconsolidation of Traumatic Memories (RTM) therapy. De Rijk and colleagues (2023) evaluated this method in Ukraine during the 2022 war. Twenty-six clinicians were trained online to deliver RTM, which uses guided visualization to destabilize and reframe traumatic memories. Fifty-three patients, many of them displaced or directly exposed to conflict, received an average of three sessions. Before treatment, nearly three-quarters met criteria for probable PTSD. Afterward, only two percent did. Depression and anxiety scores also fell significantly. The study lacked a control group, and therapists dropped out at a high rate, but the ability to train and deliver RTM under war conditions shows the method's feasibility.

Imaginal exposure and RTM converge on a similar point: structured memory reactivation, even without drugs, can alter traumatic memory in ways that patients experience as clinically meaningful. What is less certain is how long these changes last and how they compare with well-established therapies.

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Neuromodulation and Reconsolidation

Another avenue is non-invasive brain stimulation. Su and colleagues (2022) demonstrated that continuous theta-burst stimulation (cTBS) applied to the right dorsolateral prefrontal cortex can disrupt reconsolidation in healthy volunteers. Participants underwent fear conditioning, followed by reactivation, and then received cTBS. Those who received stimulation immediately after reactivation displayed weaker physiological fear responses and did not show reinstatement. The effect disappeared if stimulation was given without reactivation or six hours later. This design confirms that reconsolidation is time-limited and reactivation-dependent.

Although this work did not involve patients with PTSD, it offers a promising direction. Neuromodulation avoids drug side effects, can be precisely timed, and targets a region known to regulate emotion and memory. Trials in clinical populations will be necessary to determine whether cTBS or related methods can safely weaken traumatic recall, but the early evidence suggests a potential new tool.

Memory Reconsolidation in Psychotherapy Theory

Reconsolidation has also reshaped how some theorists view psychotherapy more broadly. Ecker and Vaz (2022) argue that reconsolidation is the only known neural process that can truly erase maladaptive emotional memories. By contrast, extinction-based therapies suppress the original trace, which helps explain why relapse is so common. The reconsolidation framework has been used to interpret sudden, lasting symptom relief occasionally reported after single therapeutic sessions. These events may represent accidental triggering of reconsolidation under the right conditions. While the idea of permanent erasure remains debated, the framework underscores why reconsolidation is such an attractive target for trauma treatment.

Synthesis

Clinical studies targeting reconsolidation in PTSD highlight both opportunities and limits. Propranolol trials show that trauma reactivation reliably alters the character of memories, but the drug itself adds little benefit. Behavioral methods like imaginal exposure and RTM suggest that well-timed reactivation alone can reduce symptoms, sometimes dramatically. Neuromodulation has not yet been applied to PTSD patients, but it demonstrates in principle that reconsolidation can be disrupted with precision. Theoretical work frames reconsolidation as fundamentally different from extinction because it modifies the original trace rather than layering inhibition over it. Overall, reconsolidation-based approaches remain in development, yet they carry the potential to produce briefer, more durable, and perhaps more acceptable treatments for PTSD if their boundary conditions can be reliably managed.

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CLINICAL STUDIES TARGETING EXTINCTION IN PTSD

Extinction forms the backbone of many PTSD treatments, since it reduces fear responses when patients repeatedly encounter trauma-related cues in safe settings. Unlike reconsolidation, extinction does not dismantle the original fear memory. Instead, it builds a new association that competes with the old one. This distinction helps explain why exposure therapies can work well yet often leave patients vulnerable to relapse. Research has examined ways to make extinction more effective, focusing on pharmacological influences, physiological states, and the nature of the stimuli being extinguished.

Adrenergic Modulation of Fear Extinction

Burhans et al. (2018) used a rabbit eyeblink conditioning model to test how propranolol influences extinction. Animals received extinction training either with propranolol or without it. Those who received the drug extinguished their responses more quickly during the training sessions. This result suggested that lowering adrenergic arousal can make extinction easier to acquire. The effect, however, did not last. When tested later, the propranolol group no longer differed from controls. The finding is important because it captures extinction's central challenge. It can be sped up or strengthened in the moment, but the original memory remains and eventually reasserts itself.

Sleep as a Modulator of Fear Extinction

Pace-Schott et al. (2023) examined the role of sleep in extinction, with particular attention to trauma-related disorders. Their review pointed out that rapid eye movement (REM) sleep appears to help stabilize extinction recall and broaden safety learning. In experimental contexts, naps following extinction improved recall, while disrupted sleep had the opposite effect. Because sleep disturbances are so common in PTSD, it seems plausible that problems with extinction retention in patients may be linked to unstable or fragmented sleep, especially in REM stages. The broader implication is that extinction therapies may be more effective if patients are able to consolidate safety learning during healthy sleep cycles.

Stimulus Salience and Trauma-Proximal Cues

Eales et al. (2025) asked whether the type of conditioned stimulus being extinguished mattered. They compared the extinction of a standard CS+ with the extinction of a stimulus presented simultaneously with the unconditioned stimulus, labeled CSX. The CSX was more tightly linked to the aversive event. Extinguishing CSX produced stronger and more durable effects, preventing relapse, while extinguishing the CS+ did not. This result suggests that extinction outcomes are shaped not only by the process itself but also by which cues are

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chosen. In a clinical setting, it may be especially important to prioritize cues that patients themselves identify as closest to the trauma, rather than those that are more peripheral.

Clinical Parallels

Exposure-based therapies such as Prolonged Exposure follow the logic of extinction by having patients repeatedly revisit trauma reminders in controlled contexts. The studies by Burhans, Pace-Schott, and Eales help explain why such therapies can succeed yet also why they sometimes fall short. Extinction is sensitive to an adrenergic state, depends on sleep for consolidation, and is influenced by stimulus salience. These features make extinction-based treatments both powerful and fragile at the same time.

Synthesis

Together, these studies reveal the complex nature of extinction as it applies to PTSD. Burhans et al. (2018) showed that adrenergic blockade can facilitate extinction in the short term but does not protect against relapse. Pace-Schott et al. (2023) demonstrated that sleep, particularly REM stages, supports extinction recall and may play a critical role in patient outcomes. Eales et al. (2025) found that extinction is most durable when directed toward trauma-proximal cues. These findings highlight extinction's strengths as the basis for existing therapies while also clarifying why relapse remains a challenge. They also explain why researchers are looking to reconsolidation as a complementary process that may overcome extinction's limits.

DISCUSSION

The evidence in this review shows that reconsolidation and extinction are not two versions of the same thing but different directions a memory can take once it is brought back up. Reconsolidation opens the original trace, which then becomes unstable and can be changed. Extinction, on the other hand, builds a new safety memory that competes with the old one. Reconsolidation looks like it can be more permanent, but it only works when certain conditions are in place, like the right timing or the presence of prediction error. Extinction is easier to bring about, and it underlies the exposure therapies that are commonly used, but its benefits do not always last. The truth is that these two processes are not neatly separate. How long a memory is reactivated, whether a drug is present, or even how well a person sleeps, can shift the outcome. Thinking of reconsolidation and extinction as two points on a continuum makes more sense than treating them as opposites, and this helps explain why they are both so difficult and so promising in PTSD treatment.

There are, however, clear limitations. Propranolol, the drug most often tested, has shown inconsistent effects, sometimes no better than a placebo. Behavioral methods like imaginal exposure or Reconsolidation of Traumatic Memories therapy (RTM) look more encouraging, but the question of how long the benefits last is

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still open. Extinction has its own problems. It is sensitive to adrenergic state, sleep, and the cues that are chosen for training. More importantly, exposure therapies that rely on extinction force patients to repeatedly face their traumas. This can be effective, but it also leads to high dropout and sometimes even makes distress worse in the short term. These difficulties may partly reflect the underlying brain alterations described by Sherin and Nemeroff (2011). Hyperactive amygdala responses, reduced prefrontal regulation, and hippocampal dysfunction all make extinction harder to consolidate and fear more likely to return. In this sense, the relapse seen in extinction-based treatments is not just a limitation of the therapy design but also a reflection of the neurobiology of PTSD itself. Most studies are also small, rely heavily on indirect measures like skin conductance, and often lack strong follow-up. We also do not yet have good head-to-head comparisons of reconsolidation-based and extinction-based treatments in clinical groups, which leaves a major gap.

Future work has to make these boundaries clearer. We need to know when reconsolidation can reliably be triggered in patients and whether combining it with extinction can create longer-lasting results. Reconsolidation is already being studied in other fields, such as addiction, where the aim is to weaken drug-cue memories. Those results matter here, too, because they show the process is not unique to PTSD. What is missing now are larger trials, clearer identification of which patients benefit most, and more attention to things like sleep and arousal that shape outcomes. In the end, reconsolidation and extinction both give us ways to target traumatic memories directly, but their clinical usefulness will depend on refining methods that work reliably and that patients are willing to go through.

CONCLUSION

This review aimed to compare reconsolidation and extinction as two pathways through which traumatic memories can be altered once recalled. Reconsolidation reopens the original memory trace, turning it temporarily unstable, while extinction involves the formation of a new safety memory that competes with the fear response. Both processes matter for PTSD. Reconsolidation appears capable of producing more enduring change, but only under specific conditions. Extinction, by contrast, is easier to elicit and forms the basis of therapies already in clinical use, though its effects often diminish over time. Overall, the evidence suggests that memory updating is not a single mechanism but a spectrum, with reconsolidation and extinction representing different outcomes shaped by timing, context, and physiology.

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