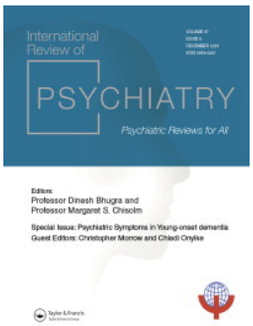

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REVIEW ARTICLE

Targeting active forgetting with non-invasive stimulation: toward novel treatments for intrusive memories in PTSD

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ABSTRACT

PTSD is characterized by persistent and intrusive recollections of traumatic events, often due to the failure of natural forgetting processes. Recent neuroscientific advances have identified active forgetting as a dynamic and regulated process involving specific brain circuits and molecular pathways, particularly within the hippocampus and prefrontal cortex. By leveraging targeted neurostimulation methods, such as transcranial magnetic stimulation (TMS), transcranial direct current stimulation (tDCS), transcranial ultrasound stimulation (TUS), or peripheral nerve stimulation, we explore how modulating these circuits may enhance adaptive forgetting and reduce pathological memory persistence. This review aims to: (1) map the neural correlates of active forgetting in individuals with PTSD; (2) explore the neurotransmitters implicated in active forgetting; (3) review the research on non-invasive neurostimulation aimed at enhancing forgetting of maladaptive memories; and (4) identify outstanding questions and future directions of such interventions. By bridging cognitive neuroscience, clinical psychology, and neurotechnology, this work seeks to establish a novel framework for PTSD treatment that moves beyond symptom management to address the core memory mechanisms underlying the disorder.

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PTSD; active forgetting; neurostimulation; prefrontal cortex; hippocampus

Introduction

Numerous studies over the years have investigated whether non-invasive neurostimulation techniques may have clinically meaningful benefit for patients with post-traumatic stress disorder (PTSD). These studies have most frequently employed transcranial direct current stimulation (tDCS) or transcranial magnetic stimulation (TMS) techniques (Gouveia et al., 2020; Harris & Reece, 2021; Kan et al., 2020). However, as highlighted in previous systematic reviews and meta-analyses, a small number of studies have also investigated non-invasive techniques such as vagus nerve stimulation or trigeminal nerve stimulation (Koek et al., 2019; Liu et al., 2024; Tseng et al., 2024). With regards to TMS and tDCS protocols, accumulating research suggests that although these do appear to improve PTSD symptomology, the evidence supporting these are low, and their sustained efficacy is limited (Belsher et al., 2021; Janssen-Aguilar et al., 2025; Liu et al., 2024). Furthermore, there is evidence to suggest that pairing non-invasive stimulation with memory reactivation protocols may be more effective for sustained improvement in PTSD symptomology (Brown et al., 2025; Saccenti et al., 2024).

Individuals can (but don't always develop) post-traumatic stress disorder (PTSD) after the experiencing or witnessing of a traumatic event (American Psychiatric Association, 2013). PTSD is characterised by persistent and intrusive recollections of these traumatic events, otherwise known as 'intrusive memories' (American Psychiatric Association, 2013; Iyadurai et al., 2019). Cognitive models of PTSD suggest that these intrusive memories may lie at the centre of PTSD symptomology and act as the driver of these other symptoms of avoidance, negative mood/cognition and hyperarousal (Iyadurai et al., 2019). Thus, modulating these intrusive memories via non-invasive stimulation techniques may have clinically meaningful benefit for PTSD patients, that is rooted in addressing the core memory mechanisms underlying the disorder.

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One argument that has been put forth is that intrusive memories stem, at least to some degree, from deficient active forgetting processes (Berry et al., 2024). Active forgetting refers to the intentional or unintentional suppression or weakening of unwanted memories (as opposed to their passive decay) (Fawcett et al., 2024). Recent accumulating research highlights the specific neural circuits and molecular pathways that regulate this process (Anderson & Hanslmayr, 2014; Anderson & Hulbert, 2021; Berry et al., 2024; Lu et al., 2023; Meyer & Benoit, 2022). In particular, top-down control of the prefrontal cortex on regions such as the hippocampus has been linked with inhibitory control mechanisms that support the suppression of these unwanted memories.

Moreover, individuals with PTSD demonstrate impaired active forgetting abilities in lab-based tasks, alongside altered activity in the neural circuits underlying active forgetting (Berry et al., 2024; Catarino et al., 2015; Costanzi et al., 2021; Hulbert & Anderson, 2018; Stramaccia et al., 2021; Sullivan et al., 2019; Waldhauser et al., 2018). For example, disrupted memory control abilities have been observed in those with PTSD and are associated with maladaptive intrusive memories or resilience after trauma (Mary et al., 2020). However, importantly, novel research is demonstrating the potential therapeutic value of targeting these maladaptive memory control processes. Specifically, training individuals to suppress unwanted thoughts has been shown to reduce persistence of future-based fears alongside sustained improvements in anxiety, negative affect and depression, especially in individuals with higher baseline levels of anxiety or post-traumatic stress (Mamat & Anderson, 2023). As such, there is increased support for the notion that strengthening active forgetting abilities in individuals with PTSD may have clinically-meaningful benefits for these patients' symptomology (Mamat et al., 2024; Mamat & Anderson, 2023; Varma et al., 2024). Alongside this, emerging research is investigating whether neurostimulation techniques can bolster this effect and support sustained improvement for individuals with PTSD (Khan et al., 2024; Penolazzi et al., 2014; Smits et al., 2021).

Nevertheless, this is still a very nascent research area with respect to investigating the potential clinically meaningful benefit of modulating active forgetting processes in PTSD. Thus, the aim of this integrative narrative review is to explore the therapeutic potential of active forgetting mechanisms in the treatment of PTSD, with a focus on the novel use of neurostimulation techniques to modulate memory networks. This review focuses on theoretical integration, drawing on representative and relevant studies, rather than attempting exhaustive coverage. Relevant literature was identified using searches of PsycINFO, PubMed and Google Scholar using keywords related to memory suppression, cognitive control, retrieval-induced or suppression-induced forgetting, PTSD, neurostimulation and peripheral nerve stimulation. Reference lists of reviews or meta-analyses were also examined.

Firstly, this review will summarise the types of active forgetting, and the lab-based tasks that are typically employed to study it. It will then outline the neural correlates of active forgetting both in healthy individuals, and specifically those with PTSD. Thirdly, it will briefly outline relevant neurostimulation approaches and their proposed mechanisms. Fourthly, it will assess the preliminary evidence on the efficacy of non-invasive neurostimulation and active forgetting protocols in enhancing forgetting of maladaptive memories. Finally, outstanding questions and future directions of such interventions will then be discussed.

Active forgetting

Active forgetting, also called motivated forgetting, can involve the suppression of an unwanted memory (at encoding or retrieval) leading to its forgetting (Anderson & Hanslmayr, 2014). It is now widely acknowledged that inhibitory control mechanisms can contribute to the suppression of unwanted memories, weakening the memory itself (Anderson & Hanslmayr, 2014; Meyer & Benoit, 2022). Active forgetting can involve either intentional or unintentional cognitive processes (Costanzi et al., 2021; Fawcett et al., 2024). In *unintentional forgetting*, forgetting is the inadvertent consequence of other memory processes - for example, when you're trying to remember the name of someone, but another person's name keeps coming to mind instead. In the lab, unintentional forgetting is typically measured through retrieval-induced or recognition-induced forgetting (RIF) paradigms (Costanzi et al., 2021; Fawcett et al., 2024).

On the other hand, *intentional forgetting* describes (as the name suggests) the ability to deliberately forget information (Costanzi et al., 2021; Fawcett et al., 2024). In the lab, intentional forgetting is typically assessed experimentally through (item-based or list-based) directed forgetting tasks, or the think/no-think task (TNT) (Costanzi et al., 2021; Fawcett et al., 2024). However, these intentional forgetting tasks differ in terms of the point at which the instruction to suppress information is given. In directed forgetting, the individual is instructed to suppress the information at the

time of encoding, whereas in the TNT task, it is at the point of retrieval (Anderson & Hanslmayr, 2014).

With respect to PTSD, it has been proposed that understanding inhibitory control processes at the time of retrieval is of more relevance, given that intrusive memories for these individuals have had time to consolidate (Marks et al., 2018; Stramaccia et al., 2021). Thus, this review will focus on studies that have investigated active forgetting in individuals with PTSD using RIF paradigms or TNT task variations.

Although no single RIF task is universally adopted across researchers, most paradigms share a common structure: consisting of a study phase, a retrieval practice phase and a test phase where participants learn cue-target pairs (often category-exemplar pairs) (Murayama et al., 2014). These tasks assess the phenomenon of RIF by demonstrating how retrieval practice of some cue-target pairs can cause forgetting of other related but unpractised items, despite no instructions to forget any items being given (Costanzi et al., 2021) (Figure 1). Thus, it reflects a form of unintentional forgetting.

TNT tasks on the other hand, assess a form of intentional forgetting, where instructions to think or not think of specific cue-target pairs are given at the time of retrieval (Scotti & Maxcey, 2021). The TNT task was first introduced by Anderson and Green (2001), with Nardo and Anderson (2024) later developing standard procedures for conducting the TNT task. Essentially, in the TNT paradigm, individuals learn cue-target pairs in a study phase. In the subsequent think/no-think phase of the task, some of the cues are presented in green, indicating that participants should actively think of the associated target, while some other cues are presented in red, indicating that individuals should avoid thinking of the associated target, and the remaining pairs are reserved as baseline items (Nardo & Anderson, 2024). Memory recall (for all the pairs) is later tested in the final phase. Studies demonstrate how the act of intentionally suppressing retrieval of these no-think items leads to greater rates of forgetting for these pairs compared to baseline or successfully retrieved (i.e. think) items (Nardo & Anderson, 2024) (Figure 1).

In one recent meta-analysis, suppression-induced forgetting rates were assessed across psychological disorders by analysing rates of forgetting (using the TNT task) for healthy controls, compared to clinical and subclinical samples (Stramaccia et al., 2021). Notably, impaired ability to intentionally forget information was observed across these studies for individuals with sub(clinical) PTSD, generalised anxiety disorder, or elevated anxiety (Stramaccia et al., 2021) in comparison to healthy controls. Although only a small number of the studies in this meta-analysis tested active forgetting (using the TNT task) in individuals with PTSD specifically, the findings are further supported by additional related research.

For example, in one study of healthy individuals, researchers demonstrated how active forgetting

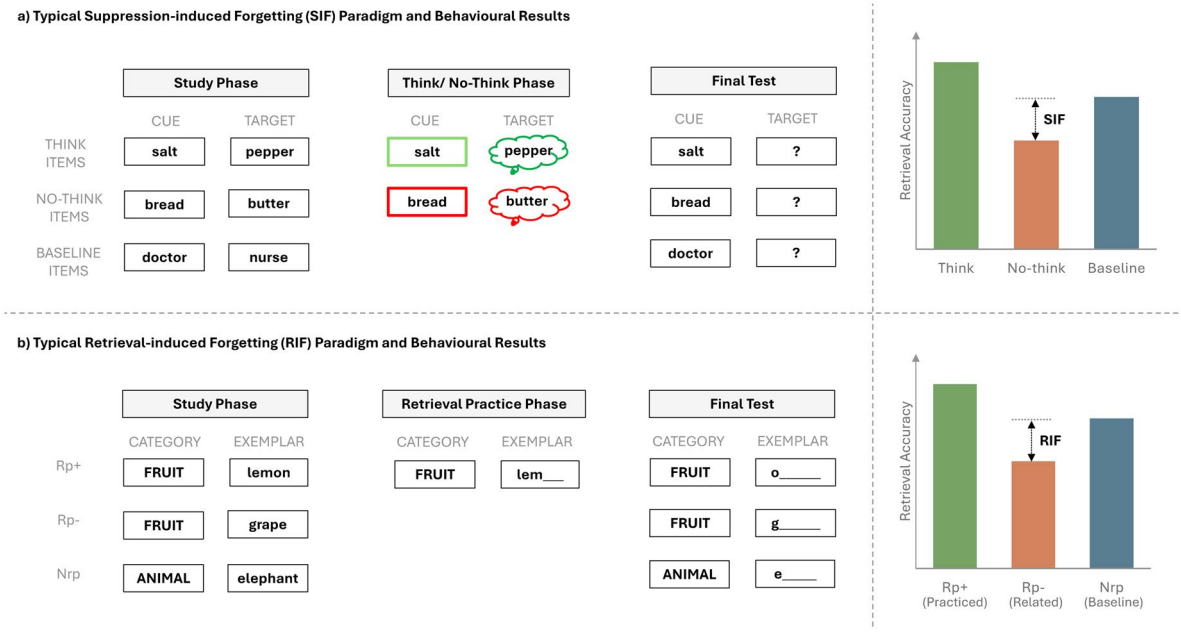

Figure 1. Typical suppression-induced forgetting (SIF) and retrieval-induced forgetting (RIF) paradigms and behavioural results. This figure outlines the typical structure of SIF and RIF tasks, and the expected behavioural results. *Abbreviations include*: Related practiced words (Rp+); Related unpracticed words (Rp-) and Non-related unpracticed words (Nrp).

capacity may be a risk factor for developing PTSD (Streb et al., 2016). To do this, the researchers investigated whether suppression ability (measured through the TNT task) and its neural correlates, were related to intrusive memories after viewing a traumatic video and PTSD symptoms (acc. to the Impact of Event Scale). Specifically, the authors highlight how suppression-induced forgetting (on the TNT task) and its event-related potential (ERP) correlates predicted the distress levels of intrusive memories after watching a traumatic video (Streb et al., 2016). In other words, greater memory suppression predicted less distress from intrusive memories. Moreover, the authors found that the neural correlates of this active forgetting also predicted PTSD symptoms 6 days after watching a traumatic video (Streb et al., 2016). In other words, a larger ERP response to the to-be-forgotten items on the TNT task predicted lower scores on the IES subscales of intrusion, avoidance and hyperarousal. This suggests that active forgetting mechanisms in the brain may not only be related to PTSD symptoms of intrusions, but also of general PTSD symptomology, and thus may be a risk factor for developing PTSD.

In another study, a classic aversive conditioning paradigm was adapted to include TNT-type instructions to assess whether active forgetting could be used to reduce conditioned threat (fear) memories in healthy individuals (Wang et al., 2021). Notably, the authors demonstrated that after establishing the acquisition of fear (using a standard conditioned stimulus (CS) – unconditioned stimulus design (UCS) paradigm), intentional memory suppression during the extinction training was able to successfully diminish the return of fear response (Wang et al., 2021). Essentially, when instructions to 'not think' about some of the CS are given during the extinction phase, physiological fear responses (measured via skin conductance responses) were significantly reduced when the UCS and then CS were later presented (Wang et al., 2021). This study critically demonstrates how active forgetting mechanisms can extend beyond pure cognitive domains, and can impact neurophysiological responses as well. In line with this, other studies have similarly demonstrated how active forgetting can not only suppress intrusive emotional or aversive memories, but also associated physiological indices such as cardiac activity and skin conductance responses (Harrington et al., 2021; Legrand et al., 2020). Although these studies were conducted in healthy individuals, they have potential relevance for PTSD given that individuals with PTSD exhibit impaired fear extinction across humans and animal models (Sep et al., 2023).

Another study examined RIF in individuals with PTSD compared to trauma-exposed individuals, or non-trauma exposed healthy controls (Amir et al., 2009). In this study, both the trauma-exposed individuals and those with PTSD exhibited impaired RIF abilities. As a result, the authors suggest that experiencing a traumatic event may contribute to active forgetting deficits, potentially leading to the development of PTSD (Amir et al., 2009). These studies demonstrate how both intentional and unintentional active forgetting mechanisms are impaired in individuals with PTSD, and may confer a risk factor for the development of PTSD (see Table 1 for a summary of these study results). However, what are the neural mechanisms underlying these processes? The next section will review the neuroimaging data on active forgetting in healthy individuals, and then specifically in those with PTSD as well.

Neural correlates of active forgetting

fMRI data on active forgetting

There is a large body of research, which implicates prefrontal cortical regions in active forgetting processes. Specifically, in healthy individuals prefrontal cortical regions (namely the dorsolateral prefrontal cortex (DLPFC) and ventrolateral prefrontal cortex (VLPFC), and anterior cingulate cortex (ACC)) have been shown to inhibit hippocampal activity via top-down control during memory suppression (Anderson & Hanslmayr, 2014; Anderson & Hulbert, 2021). Recent research and a meta-analysis suggest that this prefrontal activity is largely right-lateralized (Apšvalka et al., 2022; Guo et al., 2018). Moreover, activity in these regions during suppression can predict the extent of forgetting (Anderson et al., 2004).

For instance, one study in healthy individuals reported successful suppression of autobiographical memories in a variation of the TNT task, which was associated with reduced hippocampal activity during suppression attempts (Noreen et al., 2016). However, interestingly they did not find evidence of increased DLPFC or VLPFC activity. Nevertheless, the lack of activity in top-down regions, might simply suggest that participants experienced minimal memory intrusions, and thus top-down control was not required. Indeed, support for this proposition can be seen from another study in healthy individuals using a TNT task which found that DLPFC activity was largest when unwanted memories intruded into awareness for participants, and needed to be suppressed - with this increased activity predicting

Table 1. Studies assessing active forgetting (using TNT or RIF Tasks) in PTSD or trauma-related studies.

Study	Sample	N	Paradigm	Neuroimaging/Measures recorded	Key findings
Amir et al. (2009)	PTSD patients, trauma-exposed without PTSD, and healthy controls	48 (17 PTSD, 15 trauma-exposed no PTSD, 16 controls)	RIF	Behavioural	• Both PTSD and trauma-exposed participants showed impaired RIF compared to healthy controls. • Suggests trauma exposure may contribute to active forgetting deficits.
Catarino et al. (2015)	PTSD patients, vs trauma-exposed without PTSD	36 (18 PTSD, 18 trauma-exposed controls)	TNT	Behavioural/Clinical	• PTSD patients showed impaired suppression of memories compared to controls, which correlated with PTSD symptom severity. • Suggests inhibitory-control deficits contribute to persistence of intrusive memories in PTSD.
Leone et al. (2025)	Trauma-exposed individuals with chronic or remitted PTSD, and healthy controls	172 (34 chronic PTSD, 19 remitted PTSD, 72 controls)	TNT	Behavioural/ Clinical + fMRI (dynamic causal modelling) + structural MRI	• Hippocampal neuroplasticity/ memory control improvements preceded intrusive memory improvements and predicted PTSD remission. Chronic PTSD was associated with progressive hippocampal atrophy. • Suggests PTSD recovery is linked with plasticity in memory control circuits and hippocampal subfields, and resilience depends on adaptive reorganisation of memory control systems.
Mary et al. (2020)	Trauma-exposed with vs without PTSD, and healthy controls	175 (55 trauma-exposed with PTSD, 47 trauma-exposed without PTSD and 73 controls)	TNT	Behavioural/ Clinical + fMRI (functional connectivity)	• PTSD group exhibited persistent intrusions and failed to show suppression-related reductions in connectivity between prefrontal and memory regions, unlike resilient trauma-exposed individuals or healthy controls. • Suggests disruption of memory control networks may underlie persistence of intrusive memories in PTSD.
Postel et al. (2021)	Trauma-exposed with vs without PTSD, and healthy controls	148 (53 trauma-exposed with PTSD, 39 trauma-exposed without PTSD and 56 controls)	TNT	Behavioural/ Clinical + High-resolution structural MRI	• PTSD individuals (but not trauma-exposed with no PTSD or healthy controls) had reduced hippocampal subfield volumes. Changes in CA1 integrity were associated with intrusive memories. Resilient individuals with intact CA1 volumes showed stronger memory control network functioning. • Suggests subfield-specific hippocampal changes may be a key factor in distinguishing vulnerability to/ resilience from PTSD after trauma.
Streb et al. (2016)	Healthy participants (analogue trauma paradigm)	24	TNT + traumatic video	Behavioural + EEG/ ERP (early N2) + PTSD symptoms	• Better suppression ability (and associated N2 ERP) predicted fewer intrusive memories and lower PTSD symptoms after analogue trauma. • Suggests active forgetting may be a risk factor for developing PTSD.
Sullivan et al. (2019)	PTSD patients, vs trauma-exposed without PTSD and healthy controls	48 (16 PTSD, 19 trauma-exposed without PTSD and 13 healthy controls)	TNT	Behavioural + fMRI (BOLD)	• Trauma-exposed (with or without PTSD) individuals exhibited impaired memory suppression and reduced prefrontal cortical activity during suppression compared to controls. • Suggests impaired prefrontal control mechanisms may contribute to persistence of intrusive memories in PTSD.

(*Continued*)

Table 1. Continued.

Study	Sample	N	Paradigm	Neuroimaging/Measures recorded	Key findings
Waldhauser et al. (2018)	Trauma-exposed with vs without PTSD	24 (11 PTSD, 13 trauma-exposed without PTSD)	TNT	Behavioural/Clinical + MEG (gamma oscillations)	• PTSD group showed impaired suppression and abnormal oscillatory dynamics (reduced gamma downregulation in hippocampus/visual cortex, reduced frontal gamma), which correlated with memory intrusions. • Suggests memory suppression deficits in PTSD stem from impaired top-down memory control, with gamma-band activity during suppression potentially acting as a biomarker of resilience after trauma exposure.
Wang et al. (2021)	Healthy participants (fear conditioning + TNT instructions)	55 (28 in Exp 1; 27 in Exp 2)	TNT + extinction	Behavioural + Psychophysiology (SCR)	• Intentional forgetting during extinction training reduced return of fear responses (SCR). • Extends active forgetting effects to fear/physiological domains.

Abbreviations include: Post-traumatic stress disorder (PTSD); retrieval-induced forgetting (RIF) paradigm; think/no-think (TNT) paradigm; functional magnetic resonance imaging (fMRI); electroencephalogram (EEG); event related potential (ERP); blood oxygenation level-dependent (BOLD); magnetoencephalography (MEG); and skin conductance response (SCR).

better 'control of intrusive memories over time' (Benoit et al., 2015).

Similar results have been reported when a RIF task was used. In this study, successful repeated retrieval of target memories initiated a dynamic forgetting cascade, whereby greater forgetting of competitor memories was associated with reduced activity in the ACC and right VLPFC (Kuhl et al., 2007). The authors interpret these findings as reflective of how successful memory suppression is associated with reduced demands on top-down brain regions such as PFC and ACC regions (Kuhl et al., 2007). Similarly, more recent evidence confirms how over repeated suppression attempts, activity in top-down regions (DLPFC/VLPFC) declines, with greater reductions reflective of greater forgetting rates (Apšvalka et al., 2022). In other words, successful forgetting results in decreased demands on these memory control regions, suggesting the dynamic and adaptive nature of forgetting.

Importantly, these inhibitory control processes also influence emotional processing and activity in related brain regions. For example, during the TNT task, suppression of emotional images involved dorsolateral prefrontal inhibitory control of the hippocampus and amygdala, leading to forgetting and downregulated emotional affect in healthy individuals (Gagnepain et al., 2017). Extending this, another study (again using the TNT task) found that emotional memories were suppressed via a two-phase downregulation process beginning with prefrontal activity (Depue et al., 2007). In this study, suppression of emotional memories in healthy individuals is associated with top-down prefrontal cortex (PFC) control from right inferior frontal gyrus to sensory-related brain regions incl. visual cortex and thalamus, and subsequently top-down control from right medial frontal gyrus to memory and emotion-related brain regions incl. the hippocampus and amygdala (Depue et al., 2007).

Interestingly, for patients with PTSD, there is research that implicates abnormal activity in these brain regions in response to emotional or trauma-related stimuli. Specifically, studies suggest increased activation of regions such as the amygdala, and decreased activation of prefrontal regions including the ACC and ventromedial PFC (Aupperle et al., 2012). Essentially, this is the inverse pattern associated with successful memory suppression in healthy individuals. These contrasting patterns are further supported by neuroimaging evidence derived from the Neurosynth database (Yarkoni et al., 2011), which was used to compare typical functional magnetic resonance imaging (fMRI) activity patterns associated with PTSD versus with memory suppression. As shown in Figure 2, the top row depicts an automated meta-analysis of 106 PTSD-related studies, whereas the bottom row depicts an automated meta-analysis of 314 suppression-related studies. Notably, suppression-related studies in the Neurosynth meta-analysis are domain-general, rather than memory-specific, encompassing tasks such as motor inhibition. However, converging evidence indicates that domain-general inhibitory control mechanisms can operate across both overt actions and internal memory processes (Anderson et al., 2025; Wessel & Anderson, 2023).

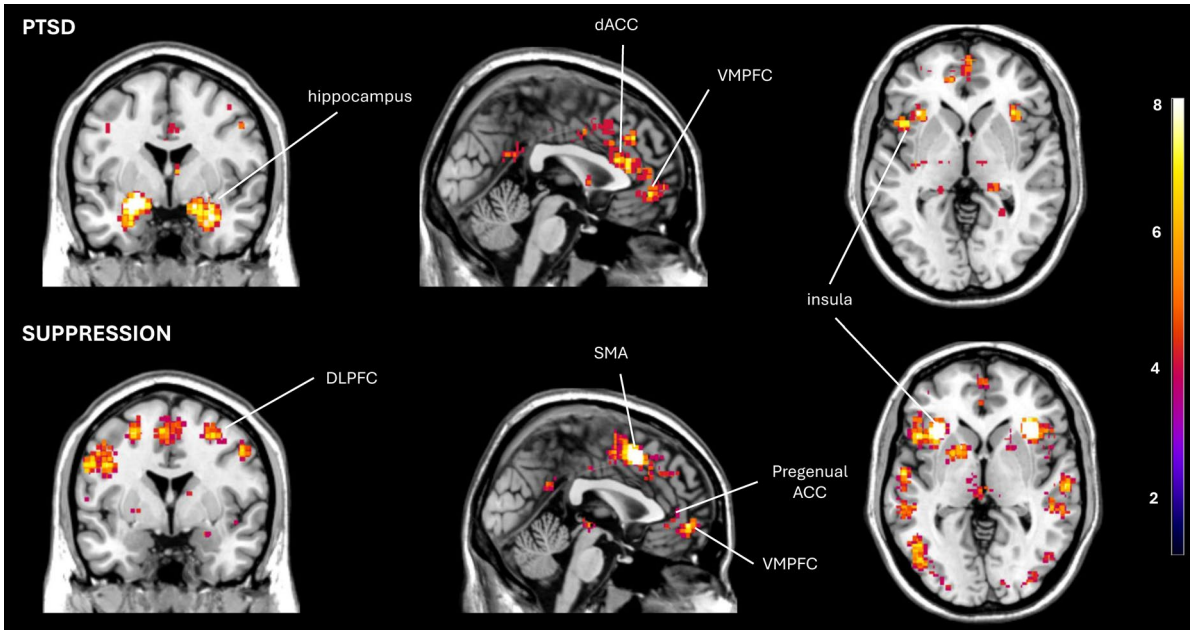

Figure 2. Exemplar brain activity patterns for PTSD versus memory suppression. These images are examples of typical functional magnetic resonance imaging (fMRI) activity patterns in PTSD versus memory suppression. These activity patterns were generated using the Neurosynth database and MRIcron for visualisation. The top row represents an automated meta-analysis of 106 PTSD-related studies, and the bottom row represents an automated meta-analysis of 314 suppression-related studies. Suppression-related studies in the Neurosynth meta-analysis are domain-general, meaning they are not memory-specific and include for example motor inhibition studies. However, research suggests domain-general inhibitory mechanisms can operate on both overt actions and internal memory processes. Lighter colours indicate higher z-score according to a uniformity test. *Abbreviations include*: post-traumatic stress disorder (PTSD); dorsal Anterior Cingulate Cortex (dACC); ventromedial prefrontal cortex (VMPFC); dorsolateral prefrontal cortex (DLPFC); supplementary motor area (SMA) and pregenual anterior cingulate cortex (pregenual ACC).

Thus, the inverse activity patterns observed (i.e. reduced engagement in control-related regions in PTSD relative to increased engagement during suppression in healthy individuals), support the view that deficient memory control processes may contribute to PTSD symptomology.

Indeed, these findings were further supported by a recent study which found that when memory intrusions were triggered, successful suppression attempts were associated with reductions in functional coupling between top-down regions and memory-related regions in healthy controls and trauma-exposed individuals without PTSD (Mary et al., 2020). However, in trauma-exposed individuals with PTSD, no such change in functional connectivity was observed (Mary et al., 2020). These contrasting fMRI findings between healthy individuals, trauma-exposed but resilient individuals and those with PTSD demonstrate how abnormal activity within and between top-down regions and memory-related regions may contribute to persistent PTSD symptomology such as intrusive memories. Although only a few fMRI studies have looked at individuals with PTSD, this viewpoint is further supported by EEG/MEG data on active forgetting.

EEG/MEG data on active forgetting

EEG and MEG studies corroborate the fMRI data, and converge on the idea that active forgetting is a top-down process that engages frontal control systems to suppress mnemonic retrieval signals. For example, in healthy individuals during the TNT task, suppression consistently elicits a fronto-central N2 ERP component (~350–450 ms) and enhanced theta/alpha oscillations in frontal networks, reflecting the detection of the need for control and the engagement of inhibitory mechanisms (Depue et al., 2013; Mecklinger et al., 2009; Streb et al., 2016; Waldhauser et al., 2015). Similarly, when investigating the neural correlates of RIF in healthy individuals, another study likewise reported greater early theta phase locking over parietal and frontocentral sites which predicted RIF (Hanslmayr et al., 2010).

In parallel, ERP signatures of recollection such as the late parietal positivity (400–800ms) are attenuated during suppression, suggesting the down-modulation of retrieval by prefrontal cortical regions (Bergström et al., 2009; Mecklinger et al., 2009). The late parietal positivity ERP is understood to reflect memory recall of an item, and is larger for older versus new words

(Anderson & Hanslmayr, 2014). Thus, suppressed late parietal positivity during no-think compared to think trials on the TNT task likely suggests successful memory suppression in healthy individuals (Anderson & Hanslmayr, 2014).

In line with this, one study assessed early and late ERP components in suppression of neutral vs emotional memories in healthy individuals (Chen et al., 2012). This study reported that less effective top-down control of emotional memories (which may be harder to suppress) was associated with smaller late negativity and larger late parietal positivity ERPs (Chen et al., 2012), suggesting the inverse pattern typically seen under successful suppression. Oscillatory findings further show that frontal synchrony (in theta and alpha frequency) increases while medial temporal and occipital theta power decreases, consistent with top-down inhibition of mnemonic reinstatement (Depue et al., 2013; Waldhauser et al., 2012; 2015).

A combined EEG-fMRI study further highlights the interactions between these brain regions, neural dynamics and memory suppression (Crespo-García et al., 2022). Specifically, in this study, a larger N2 ERP was associated with better suppression-induced forgetting in healthy individuals. Moreover, this N2 partially originated from the dorsal-ACC and was correlated with early frontal-midline theta power increases (Crespo-García et al., 2022). As the authors note, this activity has been acknowledged as a mechanism for cognitive control from the DLPFC (Cavanagh & Frank, 2014; Crespo-García et al., 2022). Expanding on this, one of the abovementioned studies demonstrates how the N2 ERP that's elicited during memory suppression overlaps in distribution and function with the N2 observed in motor stopping, pointing to a domain-general inhibitory mechanism that can operate on both overt actions, and internal memory processes (Streb et al., 2016).

Moreover, the potential clinical relevance of these neural findings for intrusive memories has also been highlighted. In one study, the N2 ERP component and early negativities during memory suppression not only predicted suppression-induced forgetting in the lab, but also predicted reduced distress from intrusive memories after exposure to a trauma film (Streb et al., 2016). Conversely, another study did report increased early right frontal beta power on no-think trials in the TNT task (Castiglione et al., 2019). However, this pattern of activity tends to be more reflective of general inhibitory mechanisms including motor-stopping tasks or alternative forms of active forgetting (such as in directed forgetting paradigms) as opposed to a specific intentional forgetting neural correlate (Berry et al., 2024).

Notably, only one study to-date (to the best of our knowledge) has specifically investigated the oscillatory correlates of active forgetting in individuals with PTSD, and the results of this study vary slightly compared to the abovementioned research in healthy individuals. In this study, neural correlates of suppression during the TNT task were assessed (using MEG) in trauma-exposed individuals with or without PTSD (Waldhauser et al., 2018). As expected, trauma-exposed individuals with PTSD exhibited impaired memory suppression abilities compared to control trauma-exposed individuals without PTSD. However, in this study, there were no differences between trauma-exposed controls and individuals with PTSD in oscillatory activity in top-down brain regions when looking at no-think versus think items (Waldhauser et al., 2018). Specifically, there were no changes in theta or alpha power for no-think compared to think trials, in the controls versus those with PTSD (Waldhauser et al., 2018). However, MEG data during suppression attempts demonstrated that those with PTSD exhibited impaired downregulation of gamma power and source activity in downstream brain regions such as the hippocampus and visual cortices. In addition, PTSD patients compared to controls exhibited reduced gamma activity in frontal regions (inferior frontal gyrus and ACC) that have previously been associated with the inhibitory control of unwanted memories (Waldhauser et al., 2018).

Together, these findings show that in healthy individuals, neural correlates of active forgetting involve an interplay between early control signals (N2, frontal theta/alpha) and subsequent dampening of retrieval-related parietal/temporal activity, and that individual variability in these neural markers predict susceptibility to intrusive, unwanted memories. However, critically, there is tentative evidence that in those with PTSD, the neural markers of deficient active forgetting abilities may vary slightly. Although there is overlap with the brain regions involved, this initial evidence suggests that the inability to suppress unwanted memories in those with PTSD may be related to modulation of gamma activity in these regions (Waldhauser et al., 2018). This point requires further investigation due to the small sample size of that study. Table 1 summarises the studies assessing active forgetting (using TNT or RIF tasks) in PTSD or trauma-related studies including the behavioural and/or neurophysiological results.

Neurotransmitters implicated in active forgetting

Interestingly, one question that has been raised is: given that prefrontal cortical projections to subcortical regions are largely excitatory, what is the mechanism through which the PFC can inhibit hippocampal or amygdala activity (Anderson et al., 2025)?

At the cellular level, GABAergic interneurons and pyramidal glutamatergic neurons have been implicated in these inhibitory control pathways from the prefrontal cortex to downstream hippocampal and amygdala regions (Anderson et al., 2025; Costanzi et al., 2021). Both indirect and direct pathways from top-down regions to subcortical regions have been specified. In the *direct* route, prefrontal cortical projections to regions such as the hippocampus are predominantly glutamatergic. Accordingly, firing of excitatory glutamatergic neurons and their excitation of downstream inhibitory GABAergic neurons form the direct pathway of this memory control circuit (Costanzi et al., 2021). In the *indirect* pathway, prefrontal glutamatergic projections to subcortical regions (such as the ventral tegmental area, locus coeruleus and basal forebrain) indirectly modulate hippocampal or amygdala activity. This stems from neurotransmitter (inc. dopaminergic, noradrenergic, cortisol and acetylcholine) axons which project to the memory-related brain regions in this indirect inhibitory memory control pathway (Costanzi et al., 2021). Indeed, there is initial evidence for how these neurotransmitters are associated with active forgetting processes. For example, one study tested whether prefrontal dopamine availability modulates memory suppression abilities. In this study, higher levels of RIF were observed in individuals with higher levels of prefrontal cortical dopamine (due to a loss-of-function allele for catechol-O-methyltransferase (COMT))(Wimber et al., 2011).

However, dopamine neurons in mammals can in fact co-release neurotransmitters such as glutamate and GABA (Berry et al., 2024), and more research has implicated the roles of abnormal glutamatergic and GABAergic neurotransmission not only in active forgetting processes, but also in trauma-related disorders (Iqbal et al., 2023). In one study, GABA levels in the hippocampus, prefrontal cortex and visual cortices were quantified using multimodal imaging, while fMRI was conducted during the TNT task (Schmitz et al., 2017). In this study, elevated resting-state hippocampal GABA levels (but not prefrontal or visual cortices' GABA) were correlated with increased negative coupling between prefrontal cortical and hippocampal regions, as well as with greater hippocampal downregulation during successful memory suppression (Anderson et al., 2025; Schmitz et al., 2017). This emphasises the central role of local hippocampal GABAergic interneurons in active forgetting processes in healthy individuals, and is consistent with a direct fronto-temporal inhibitory pathway.

Furthermore, in individuals with PTSD, a review of preclinical studies, clinical studies neuroimaging studies and pharmacological research, highlights how reduced functioning of the GABAergic system was associated with the inhibitory PFC-hippocampal network PTSD (Huang et al., 2023). Not only that, but reduced GABA levels post-trauma was shown to predict subsequent PTSD development (Huang et al., 2023). In parallel, stress and the role of glucocorticoids may likewise regulate these pathways and influence active forgetting processes. For example, in rodent models, chronic stress remodels amygdala projection neurons increasing their size generally, and specifically increasing excitatory glutamatergic transmission (Zhang et al., 2019), subsequently reducing the number of GABAergic interneurons in the hippocampus (Czéh et al., 2015). In fact, increasing research suggests that chronic stress through glucocorticoid mechanisms precipitates an excitation/inhibition imbalance (McKlveen et al., 2019), with stress leading to neuronal architecture remodelling in the prefrontal cortex, hippocampus and amygdala (McEwen et al., 2016).

Notably stress was shown to modulate active forgetting processes in healthy individuals. For example in one study, acute stress not only increased cortisol, but negatively impacted individuals' ability to actively suppress memories in the TNT task, with this impaired forgetting effect associated with altered theta activity in the lateral parietal cortex during suppression, and this altered activity likewise associated with altered levels of cortisol (Quaedflieg et al., 2020). Similarly, in RIF tasks, stress abolishes forgetting and is associated with cortisol release (although cortisol alone is not sufficient to block forgetting) (Koessler et al., 2009; 2013).

With respect to PTSD, these individuals exhibit altered GABA receptor functioning and glutamate receptor density (Holmes et al., 2017; Reuveni et al., 2018). Not only that, but the neurobiology of PTSD involves aberrant glucocorticoid functioning, and the dysregulation of stress responses particularly in regions such as the prefrontal cortex, hippocampus and amygdala (Holmes et al., 2017; Sherin & Nemeroff, 2011). Thus, these pathways may likewise impact active forgetting processes in those with PTSD.

Indeed, early evidence supporting this comes from two recent studies. Both studies were conducted in the aftermath of the 2015 Paris terrorist attacks, to investigate differences between those who experience traumatic stress and do or don't develop PTSD, in comparison to healthy controls (Leone et al., 2025; Postel et al., 2021). The first study specifically looked at trauma-exposed individuals (with or without PTSD) and healthy controls. In this study, hippocampal subfield volumes were estimated using high-resolution MRI. The authors observed altered volumes in hippocampal subregions in those with PTSD but not resilient (i.e. trauma-exposed with no PTSD) individuals (Postel et al., 2021). Moreover, changes in CA1 integrity were associated with intrusive memories, and also in resilient individuals who exhibited optimal functioning of memory control networks (Postel et al., 2021). These findings propose a mechanism whereby trauma-induced stress may negatively impact hippocampal volume in some individuals, which could contribute to active forgetting deficits and the downstream development of PTSD.

In the second similar study, individuals were followed up with longitudinally over 5 years after the 2015 Paris terrorist attacks. In this study, trauma-exposed individuals with chronic or remitted PTSD, as well as non-exposed healthy controls were assessed. Building on prior research, this study aimed to investigate how hippocampal morphology and memory control mechanisms contributed to trauma resilience and PTSD symptoms. Critically, the role of hippocampal neuroplasticity and improvements in memory control processes were found to predict PTSD remission over time and preceded improvements in intrusive memory symptoms (Leone et al., 2025). Together, these findings further underscore the importance of active forgetting processes in reducing intrusive memories and shaping hippocampal plasticity, thereby contributing to resilience against, or vulnerability to, the development of PTSD (Table 1). In light of these neurotransmitters, brain regions and key mechanisms of interest implicated in active forgetting processes: one promising avenue for targeting these mechanisms to mitigate intrusive memories in PTSD, is the use of non-invasive neurostimulation techniques.

Non-invasive neurostimulation techniques

Research into transcranial electrical stimulation (tES) techniques has expanded substantially over the last three decades, alongside significant advancements in understanding their potential clinical applications (Davidson et al., 2024; T. Zhang et al., 2025). Among these approaches, transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS), are the most extensively studied (Davidson et al., 2024; T. Zhang et al., 2025). Since its initial conception, TMS has received FDA approval for the treatment of a several neuropsychiatric conditions including major depressive disorder, migraines, obsessive compulsive disorder, and addiction among others (Cohen et al., 2022). Similarly, an at-home tDCS device has recently been approved by the FDA for the treatment of major depressive disorder (Bikson et al., 2026). Growing bodies of research are also examining transcranial alternating current stimulation (tACS) and more recently, transcranial ultrasound stimulation (TUS)(Davidson et al., 2024), although these are yet to receive FDA approval for the treatment of neuropsychiatric conditions.

The above approaches were originally studied as a means of modulating the central nervous system. However, evidence has emerged for a peripheral pathway contributing to the effects observed in response to some tES approaches approaches (Asamoah et al., 2019; Majdi et al., 2024; van Boekholdt et al., 2021; Vöröslakos et al., 2018). Consequently, peripheral nerve stimulation techniques (such as those targeting the vagus nerve, trigeminal nerve or greater occipital nerve) are increasingly being investigated and validated for their effects on cognition, mood and motor function in healthy and clinical groups (Arulchelvan & Vanneste, 2025; Chen et al., 2025; Liu et al., 2025; Szeska et al., 2025; X. Zhang et al., 2025). Accordingly, the following section provides an overview of the most widely studied non-invasive neurostimulation approaches, as well as emerging techniques that are gaining empirical support, each of which have relevance to the study of PTSD in light of their mechanisms of action.

TMS

TMS involves applying electromagnetic pulses to the scalp, with the aim of modulating neuronal activity in the brain. This is achieved by placing a magnetic coil over the individual's head which generates a high-intensity magnetic field. This field induces an electrical current in the underlying cortical tissue, leading to either depolarisation or hyperpolarisation of neuronal populations, and consequently, modulation of oscillatory activity within targeted brain regions (Kricheldorff et al., 2022; T. Zhang et al., 2025).

The effects of a single pulse of TMS are transient. Thus, repetitive TMS (rTMS) was developed to induce

longer-lasting neuromodulatory effects (Davidson et al., 2024). rTMS has become the most widely used form of TMS in clinical research and practice (Davidson et al., 2024).

As the name suggests, rTMS typically involves the delivery of thousands of magnetic pulses in rapid succession within a single session. The frequency of the stimulation, either high frequency (≥ 5 Hz) or low frequency (≤ 1 Hz), determines its effects on cortical excitability, with high-frequency stimulation generally increasing excitability and low-frequency decreasing excitability (i.e. increasing inhibition) (Davidson et al., 2024; T. Zhang et al., 2025). These excitatory or inhibitory effects of rTMS may stem from synaptic plasticity mechanisms such as long-term potentiation (LTP) or long-term depression (LTD) respectively (Fitzsimmons et al., 2024).

At the molecular and neurotransmitter level, rTMS appears to engage multiple systems that support synaptic plasticity (Fitzsimmons et al., 2024). In human studies, prefrontal high-frequency rTMS has been linked with increased dopamine release in connected frontal cortical regions and downstream striatal and extrastriatal regions in both healthy and neuropsychiatric individuals (Cho & Strafella, 2009; Pogarell et al., 2006; Strafella et al., 2001; 2003). Additionally, animal and human studies report rTMS-associated increases in brain-derived neurotrophic factor (BDNF) expression and TrkB signalling (Wang et al., 2011), which can facilitate NMDA receptor-dependent LTP and structural dendritic remodelling, mechanisms that are plausibly required for enduring changes after stimulation. Importantly, these synaptic plasticity mechanisms can alter brain connectivity, which have been argued to involve neurotransmitters, in particular the excitatory/inhibitory balance. For example, rTMS can modulate cortical glutamate and GABA concentrations (although rodent research suggests this effect is region-dependent (Yue et al., 2009)), which may shift circuit gain in a way that down-regulates hyper-reactive fear circuitry in PTSD (Claverie et al., 2024; Edinoff et al., 2022).

Notably, early evidence in a rodent model of PTSD has linked rTMS' effects on fear memories with anti-inflammatory pathways in the cortex and reduced glutamate and increased GABA neurotransmission in downstream basolateral amygdala region (Claverie et al., 2024). This may have distinct relevance for individuals with PTSD, because, as mentioned in previous sections of this review, PTSD is associated with reduced functioning of the GABAergic system: with reduced GABA levels associated with the inhibitory PFC-hippocampal network and capable of predicting PTSD development post-trauma (Huang et al., 2023). Further translational research is needed to validate if the same pathways are involved when rTMS is applied in humans with PTSD.

Clinically in PTSD, the DLPFC is a common rTMS target with the therapeutic rationale that modulating DLPFC activity restores top-down regulation of hyperactive limbic nodes, reconfiguring large-scale networks, thereby reducing hyperarousal and intrusive memory expression (Edinoff et al., 2022). As a standalone treatment however, there is conflicting evidence as to whether rTMS (either high or low frequency) improves PTSD symptoms (Brown et al., 2024; Kan et al., 2020). Nevertheless, positive outcomes are enhanced when rTMS is paired with relevant tasks (Brown et al., 2025). Thus, in light of its mechanisms, pairing rTMS with active forgetting paradigms may be a promising route for future research to investigate.

TUS

Transcranial focused ultrasound stimulation (tFUS or TUS) is a non-invasive neuromodulation technique that uses low intensity ultrasound pulses to perturb neuronal membranes with high spatial precision and the capacity to reach deep subcortical targets with millimetre-scale focal zones (Chou et al., 2024; Darmani et al., 2022; Keihani et al., 2024). Both preclinical and clinical research demonstrate how TUS is a viable neuromodulation tool for inhibiting or enhancing neural activity in cortical and subcortical regions (Pellow et al., 2024). Mechanistically, TUS differs from electromagnetic approaches in that its proposed direct effects include gating of mechanosensitive ion channels, transient modulation of membrane capacitance and perturbation of synaptic vesicle dynamics, while indirect pathways include glial and neurovascular responses that alter local excitability (Chou et al., 2024; Keihani et al., 2024).

Neurotransmitter effects of TUS are an active research frontier. Preclinical microdialysis and immediate early gene studies in rodents indicate that low-intensity TUS can change local extracellular levels of glutamate and GABA, and drive c-Fos/GAD expression consistent with altered excitatory/inhibitory signalling (Fomenko et al., 2020; Hsieh et al., 2024; Pellow et al., 2024; Yang et al., 2012). Given that TUS can access deep limbic structures implicated in fear encoding and PTSD symptomology, it is an attractive candidate for circuit-level interventions in PTSD. Indeed, parallel rodent research and early human studies show that sonication of amygdala or connected prefrontal cortical sites modulates fear

conditioning and extinction or reduces anxiety, which is mechanistically consistent with engagement of glutamate-dependent synaptic plasticity (Barksdale et al., 2025; Chou et al., 2024; Lee et al., 2024; Mahdavi et al., 2023). Although preliminary, these findings are encouraging in demonstrating that TUS can modulate neuronal firing and functional connectivity of targeted subcortical nodes such as the amygdala, resulting in reduced fear and anxiety in rodents, as well as healthy and clinical groups. Additionally, given that TUS can target subcortical nuclei, it may exert both local synaptic effects (through shifting the excitation/inhibition balance for example) or network-level changes through neurotransmitter release. Given that dopamine neurons can co-release GABA and glutamate, and dopamine is acknowledged for its role in regulating fear memory processing (Berry et al., 2024; Salinas-Hernández & Duvarci, 2021; Wimber et al., 2011), it is plausible that TUS' effects stem from co-modulation of neurotransmitters, although this remains to be directly tested. Verification of these mechanisms would position TUS as a potentially promising tool for modulating active forgetting in PTSD.

tDCS and tACS

tDCS and tACS are two of the most common forms of tES. In both forms of stimulation, low intensity current is applied through two electrodes (enclosed in saline-soaked sponges) placed on the scalp, with the aim of modulating underlying brain activity (Woods et al., 2016). However, they differ in terms of the modality of their stimulation, in essence whether direct or alternating current is applied. Given the differing current waveforms in tACS and tDCS, their mechanisms of action are proposed to differ (Woods et al., 2016).

tDCS mechanism

One important consideration in tDCS is the orientation of the electrical field (Nitsche et al., 2008). This orientation is primarily determined by the placement and polarity of the electrodes. The anode refers to the positively charged electrode, while the cathode refers to the negatively charged electrode, with electrical current flowing form the cathode towards the anode (Nitsche et al., 2008). tDCS results in sub-threshold shifts in resting membrane potentials. In particular, anodal tDCS biases neurons towards depolarisation (increasing cortical excitability) while cathodal tDCS biases towards hyperpolarisation (reducing excitability) (Nitsche et al., 2008; Nitsche & Paulus, 2000). These tonic shifts rarely induce immediate action-potential trains but change the probability of firing and the synaptic conditions for Hebbian plasticity. When paired with behavioural training or psychotherapy, tDCS can thereby facilitate learning and consolidation (Nitsche & Paulus, 2000; Stagg & Nitsche, 2011).

Pharmacologic and multimodal imaging studies suggest tDCS' effects arise from modulation of several neurotransmitter systems. Magnetic resonance spectroscopy studies show polarity-sensitive changes in GABA (with anodal tDCS often reducing local GABA concentration) and sometimes in glutamate, consistent with an increase in cortical excitability and plasticity potential (Medeiros et al., 2012; Stagg et al., 2009). Pharmacological studies further implicate dopamine and serotonin in tDCS' enduring effects, indicating these monoamines gates the consolidation of tDCS-induced plasticity (Nitsche et al., 2006; Yamada & Sumiyoshi, 2021). Additionally, the role of acetylcholine in modulating attention and plasticity during stimulation has likewise been identified (Kuo et al., 2007). For PTSD, these neurotransmitter interactions are important because monoaminergic and GABAergic dysfunctions are implicated in the pathophysiology of PTSD. Thus, tDCS may therefore act by both restoring prefrontal excitability and by biasing neuromodulatory systems that shape learning during cognitive interventions.

tACS mechanism

In comparison to tDCS where a constant current is applied, in tACS, a weak sinusoidal current at a chose frequency is applied with the intention of modulating endogenous brain rhythms. The primary mechanistic hypothesis is entrainment. Accordingly, tACS aims to bias the phase and amplitude of oscillatory frequencies (i.e. alpha, theta, gamma or beta bands), increasing their rhythmic synchrony within or between regions, and thereby changing effective neuronal communication and timing-dependent plasticity (Agboada et al., 2025; Liu et al., 2023).

In PTSD, aberrant oscillatory dynamics (such as altered theta synchrony during fear memory processing) provide conceptual targets for tACS. Arguably, by restoring more adaptive oscillatory activity, tACS could promote better fear memory processing, reduce intrusive memories or improve cognitive control over traumatic memories. Indeed, one ongoing randomised controlled trial is investigating whether theta-tACS can have a beneficial effect on memory consolidation in extinction learning after exposure-based

psychotherapy for post-active military veterans with PTSD or anxiety symptoms (Hendriks et al., 2025).

From a neurotransmitter perspective, tACS effects are less well characterised than for rTMS or tDCS. However, oscillatory state and neuromodulatory tone are tightly coupled. For example, cholinergic and noradrenergic systems strongly influence theta and gamma generation, while GABAergic interneurons are central to gamma and high-frequency rhythmogenesis (Agboada et al., 2025). Thus, tACS may act indirectly by biasing interneuron timing and thereby altering GABA-dependent rhythmic inhibition, or by changing oscillatory state which alters the timing of neuromodulator release and plasticity-related transmitter signalling (Nowak et al., 2017). In line with this, rodent and human research indicates that dopamine or acetylcholine bursts modulate the temporal dynamics of oscillatory activity (Andino-Pavlovsky et al., 2017; Gedankien et al., 2023; Zhang et al., 2010), which could influence memory consolidation and extinction relevant to PTSD.

Empirical human data linking tACS with specific neurotransmitter changes are still relatively sparse, so much of this account remains inferential. Nevertheless, the peripheral nerve mechanism behind traditional tACS or tDCS montages has been garnering increasing support across rodent and human studies, and provides a further intuitive account for how both tACS and tDCS may modulate neurotransmitter activity (Asamoah et al., 2019; van Boekholdt et al., 2021; Vöröslakos et al., 2018).

Peripheral nerve stimulation

Placement of traditional electrode montages often overlaps with peripheral nerves located just under the scalp, including the trigeminal and greater occipital nerves (Asamoah et al., 2019; Luckey et al., 2023; van Boekholdt et al., 2021; Vöröslakos et al., 2018). These peripheral nerves, together with the vagus nerve, synapse onto the nucleus tractus solitarius and project to multiple brainstem nuclei (Luckey et al., 2023). Through synapses on the nucleus tractus solitarius, these afferent pathways are proposed to influence key neuromodulatory hubs including the locus coeruleus, substantia nigra, ventral tegmental area, raphe nucleus and nucleus basalis. In turn, these nucelu regulate the release of noradrenaline, dopamine, serotonin and acetylcholine respectively (Luckey et al., 2023). Consequently, one proposed mechanism by which tDCS or tACS modulate neurotransmission is via the co-stimulation of peripheral nerves and their associated neuromodulatory systems.

Consistent with this account, noradrenaline, acetylcholine and serotonin have been shown to be necessary for the cortical plasticity effects of vagus nerve stimulation (Hulsey et al., 2016; 2019). Furthermore, converging evidence from rodent and human studies demonstrates that peripheral nerve stimulation engages these neuromodulatory pathways. In particular, stimulation of each of these nerves modulates noradrenaline activity originating from the locus coeruleus, with downstream effects on learning, memory and motor function (Arulchelvan & Vanneste, 2025; Asamoah et al., 2019; Chen et al., 2024; 2025; Hulsey et al., 2017; Luckey et al., 2023; Majdi et al., 2024; 2025; Seminck et al., 2024; Vanneste et al., 2020). Taken together, these findings position peripheral nerve stimulation as a promising neurostimulation approach that, when combined with active forgetting tasks, may help mitigate intrusive memories in in PTSD.

Modulating active forgetting in PTSD

Current treatments for PTSD revolve largely around extinction or exposure procedures, which show limited effectiveness and inconsistent clinically-meaningful results (Nader et al., 2013). However, modulating intrusive memories is increasingly being acknowledged as a potential viable route for ameliorating PTSD symptoms (Varma et al., 2024). In a systematic review and meta-analysis, behavioural, pharmacological and neuromodulating approaches that targeted intrusive memories in lab-based models of trauma in non-clinical populations, were found to be effective in reducing intrusive-memory related outcomes (Varma et al., 2024). Within this analysis however, only two studies assessed neurostimulation protocols (one tDCS and one rTMS) with conflicting results. The rTMS study reported that 1 Hz rTMS (aimed at inhibiting cortical excitability) applied to the visual cortex resulted in downregulated emotional intensity of intrusive memories (although it did not impact their frequency or explicit visual memory). The tDCS study applied stimulation to the left DLPFC whilst participants completed a cognitive control task prior to analogue trauma exposure (Voss et al., 2019). Contrary to the authors' hypothesis, tDCS had no effect on intrusive memories following trauma exposure, leading them to question the causal role of the DLPFC in cognitive control and intrusive memories (Voss et al., 2019).

However, given the well-established evidence that the effects of stimulation are task-dependent (Jun et al., 2020; Rizvi et al., 2023; Saucedo Marquez et al., 2013), these null results may instead reflect task

design artifacts, rather than the absence of a true effect. In other words, because stimulation was paired with a cognitive control task, one would expect any modulation to primarily influence task-related outcomes, rather than intrusive memories after trauma exposure. This led the authors to conclude that alternative methods of modulating intrusive memories (i.e. different active forgetting tasks) in combination with neuromodulation may offer better opportunities for modulating intrusive memories (Varma et al., 2024).

This sentiment was likewise noted in a randomized controlled trial which aimed to modulate inhibitory control training using tDCS and assess PTSD, aggression and anxiety symptoms in a military sample (Smits et al., 2021). In this study, tDCS over the right inferior frontal gyrus was applied during a stop-signal task, and inhibitory control was assessed using an emotional go/no-go task. However, stimulation had no impact on inhibitory control or any of the symptoms. As a result, the authors concluded that alternative cognitive tasks and/or stimulation parameters may be more effective (Smits et al., 2021). Thus, active forgetting paradigms such as the TNT or RIF tasks may be intuitive targets in conjunction with neurostimulation approaches.

Interestingly, only two studies to-date have applied neurostimulation alongside RIF tasks. In one recent study, high definition (active or sham) tDCS was applied in between the study and retrieval practice phase of a RIF task (Khan et al., 2024). In this study, tDCS increased memory for competing items, but not target items, resulting in reduced RIF which was associated with increased beta desynchronisation in fronto-parietal regions. Specifically, the change in RIF was correlated with greater early beta desynchronisation (<500ms) in the left DLPFC during retrieval practice, subsequently leading to stronger late beta desynchronisation in the parietal cortex (Khan et al., 2024). Beta desynchronisation in the parietal cortex has been associated with successful memory retrieval (Hanslmayr et al., 2012).

Thus, the authors suggest that tDCS to the prefrontal cortex may block inhibitory control, leading to increased retrieval of competing items which is reflected in increased beta desynchronisation in the parietal cortex. Notably, although theta activity in frontocentral and parietal sites is frequently implicated in RIF and TNT studies (Depue et al., 2013; Hanslmayr et al., 2010; Mecklinger et al., 2009; Streb et al., 2016; Waldhauser et al., 2015), neuromodulation of RIF in this study was not associated with any changes in theta activity. Specifically, although both sham and active groups exhibited decreased theta power in prefrontal regions across retrieval practice blocks, active tDCS had no impact on theta power or mid frontal theta activity (markers of competitive interference and cognitive control (Cavanagh & Frank, 2014; Hanslmayr et al., 2010)). One potential limitation of this study is that blinding conditions were not reported, and thus participants' expectations may have biased results.

Adding to this, somewhat conflicting findings were reported in an earlier single-blind sham-controlled study, where anodal, cathodal or sham tDCS were applied to the right DLPFC during the retrieval practice phase of a RIF task (Penolazzi et al., 2014). In this study, only cathodal tDCS over the right DLPFC blocked RIF in this study (Penolazzi et al., 2014). One possible reason for these opposing results could be how stimulation was paired with the retrieval practice phase in this study, but it was applied just prior to this phase in the previous study (Khan et al., 2024).

Another interesting factor to consider in the study by Penolazzi et al. is the placement of the electrodes (Penolazzi et al., 2014). In this study, cathodal stimulation over the right DLPFC (site F4 on the 10–20 EEG system) with the anode placed over the left supraorbital region, was interpreted as reducing excitability in the right DLPFC, thereby abolishing RIF. This aligns with traditional models of tDCS polarity, where cathodal stimulation decreases cortical excitability while anodal stimulation increases it (Nitsche & Paulus, 2000). However, an alternative interpretation merits consideration: the supraorbital region may not be a neutral reference site in this cathodal stimulation montage, as it overlies branches of the trigeminal nerve, with trigeminal nerve stimulation known to modulate cognition and memory processes (Adair et al., 2020; Chen et al., 2025). As discussed earlier in this review, in recent years, accumulating research is validating this indirect transcutaneous route behind neurostimulation approaches (Asamoah et al., 2019; van Boekholdt et al., 2021; Vöröslakos et al., 2018). As such, anodal excitation in this area may therefore modulate arousal systems indirectly, contributed to the observed behavioural effects. Thus, while suppression of right DLPFC activity remains the most parsimonious explanation, the possibility that excitatory input at the left supraorbital site influenced inhibitory control processes cannot be excluded.

One way to disentangle these effects, would be for future research to use extracephalic reference electrodes when attempting to modulate these active forgetting processes. However, a potential important

direction for future research would be to further explore whether peripheral nerves offer a viable means of modulating active forgetting processes. In rodents, peripheral nerve stimulation can activate deeper brain regions involved in memory processes and neurotransmission (e.g. hippocampus and brainstem nuclei)(Chen et al., 2025; Hulsey et al., 2017). Moreover, peripheral nerve pathways are essential in reaching deeper brain regions, as blocking these nerves abolishes any activity in these regions in response to peripheral nerve stimulation (Luckey et al., 2023; Majdi et al., 2024; 2025). Thus, this is a nascent research area that warrants investigation.

Outstanding questions and directions for future neuromodulation research on active forgetting processes in PTSD

Despite growing evidence for the role of active forgetting in mitigating intrusive memories, research on how non-invasive neuromodulation can enhance these processes in PTSD remains in its infancy. Building on the current synthesis of cognitive, neuroimaging and neuromodulation findings, several outstanding questions and key directions for future research can be identified. These are outlined below and summarised in Table 2.

Table 2. Outstanding questions and directions for neuromodulation of active forgetting.

Domain	Outstanding question	Example experimental approach
Stimulation + Paradigm	Which stage is stimulation most effective at (encoding/ retrieval/ suppression)?	Compare tDCS/TMS applied before vs during TNT/RIF phases.
Stimulation parameters	Which montages/ frequencies/polarities best enhance forgetting?	Systematic variation with extracephalic reference electrodes.
Clinical translation	Do improvements in active forgetting translate to lasting PTSD symptom reduction?	Longitudinal randomized controlled trials with booster stimulation sessions.
Individual differences	Which neurophysiological biomarkers predict who benefits?	Stratify by ERP (N2), hippocampal subfield integrity, frontal gamma.
Neurochemical mechanisms	How do stimulation effects interact with GABA, glutamate, dopamine?	Combine tDCS/TMS with MRS imaging or pharmacological probes.

Abbreviations: Transcranial direct durrent stimulation (tDCS); transcranial magnetic stimulation (TMS); think/no-think (TNT); retrieval-induced forgetting (RIF); post-traumatic stress disorder (PTSD); event-related potential (ERP); gamma-aminobutyric acid (GABA); magnetic resonance spectroscopic (MRS) imaging.

Pairing non-invasive stimulation with active forgetting tasks

With research highlighting how pairing stimulation with memory reactivation protocols may be effective means of sustained symptom improvement in PTSD (Brown et al., 2025; Saccenti et al., 2024), active forgetting paradigms are one means through which this might be achieved (Mamat et al., 2024; Mamat & Anderson, 2023; Varma et al., 2024). Specifically, given the nascency of this research area with respect to PTSD, future studies should continue investigating pairing neurostimulation techniques with intentional and unintentional active forgetting paradigms (specifically, SIF or RIF tasks respectively).

Although one might assume that intentional forgetting tasks have more potential therapeutic value than unintentional forgetting paradigms, performance across these tasks is correlated, suggesting overlapping mechanisms (Fawcett et al., 2024; Scotti & Maxcey, 2021). Furthermore, unintentional forgetting tasks tend to elicit greater effects (Fawcett et al., 2024; Scotti & Maxcey, 2021). For this reason, given that individuals who struggle to forget unwanted thoughts/ memories often exhibit deficits in cognitive control (as evidenced by impaired performance on intentional forgetting tasks), unintentional forgetting paradigms may consequently offer greater clinical applications (Fawcett et al., 2024).

Additionally, open questions remain about at what stage in the active forgetting paradigms will the application of neuromodulation be most effective. Experimental protocols should systematically compare the impact of stimulation timing on SIF or RIF, and specifically test for example whether neurostimulation at memory retrieval/suppression or just prior to this has different effects.

Mechanistic specificity of non-invasive neuromodulation approaches

While preliminary studies demonstrate that non-invasive stimulation to the DLPFC can influence memory control processes, it remains unclear which stimulation approaches (rTMS, tES, TUS or peripheral nerve stimulation) and stimulation parameters (frequency, intensity, polarity, montage) most reliably enhance active forgetting. Specifically, future research should pay attention to the placement of reference electrodes, and consider extracephalic reference electrode sites where possible due, if trying to ensure that the effects of non-invasive stimulation are not due to the co-stimulation of peripheral nerves.

Future work must also identify whether distinct non-invasive neurostimulation approaches preferentially modulate intentional vs unintentional forgetting in those with PTSD, and whether their effects map onto specific neural oscillatory markers (e.g. theta vs gamma dynamics). For example, although there is overlap in the brain regions involved in active forgetting in healthy individual's vs those with PTSD, there appears to be initial evidence to suggest that the neural dynamics differs slightly between clinical and non-clinical samples. Specifically, in healthy individuals, neural correlates of active forgetting typically involve an interplay between early control signals (N2, frontal theta/alpha) and subsequent dampening of retrieval-related parietal/temporal activity, and that individual variability in these neural markers predict susceptibility to intrusive, unwanted memories. However, initial evidence suggests that the inability to suppress unwanted memories in those with PTSD may be related to modulation of gamma activity in these regions (Waldhauser et al., 2018). However, only one (small sample size) study has previously investigated this, thus this remains to be further validated in future work.

Additionally, evidence implicates neurotransmitter and neurochemical mechanisms (e.g. GABA, glutamate, dopamine and stress hormones) in active forgetting processes and trauma-related disorders. However, little is known about how non-invasive stimulation protocols interact with these neuromodulatory systems in PTSD populations. Combining neurostimulation with pharmacological probes (e.g. GABA agonists, dopaminergic modulators) or spectroscopic imaging could clarify how stimulation drives neurochemical changes that facilitate active forgetting.

Individual differences, biomarker development and longitudinal efficacy

Research has highlighted how traditional neurostimulation approaches appear to have limited sustained efficacy in improving PTSD symptomology (Belsher et al., 2021; Janssen-Aguilar et al., 2025; Liu et al., 2024). However, to date, no studies have tested the long-term clinical effects of enhancing active forgetting processes via non-invasive stimulation in those with PTSD. Thus, future trials should assess whether these approaches produce enduring reductions in intrusive memories, and whether booster sessions are needed.

In parallel, not all individuals with PTSD exhibit the same degree of memory control impairment, and resilience appears linked to variability in active forgetting-related neural signatures. Identifying biomarkers (e.g. N2 ERP amplitude, hippocampal subfield integrity, prefrontal gamma modulation) could guide personalised interventions and predict which patients are most likely to benefit from active forgetting-based non-invasive neuromodulation.

Conclusion

This review highlights the emerging role of active forgetting as a crucial mechanism in understanding and treating PTSD. Evidence from cognitive paradigms, neuroimaging and neurochemical studies converges on the notion that deficits in active forgetting contribute to the persistence of intrusive memories and related symptoms. Importantly, neuromodulation techniques (though still in their infancy in this domain) offer promising avenues for enhancing the neural and cognitive processes underlying memory suppression. Early findings suggest that pairing stimulation with active forgetting paradigms may hold therapeutic value, yet many mechanistic, methodological and translational questions remain unresolved. Future work will need to establish optimal stimulation parameters, identify precise neural and neurochemical targets, and clarify how such interventions can be personalised and sustained over time. In this sense, active forgetting-focused non-invasive neuromodulation represents not only a novel research frontier but also a potential paradigm shift in PTSD treatment: moving from symptom management to directly targeting the memory processes at the heart of the disorder.

Disclosure statement

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