Factors Influencing Suppression-Induced Forgetting and Its Neural Mechanisms

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Abstract

When confronted with unpleasant reminders, people typically attempt to prevent unwanted memories from coming to mind. Prior research has demonstrated that suppressing retrieval of unwanted memories reduces their accessibility, resulting in forgetting—an effect termed suppression-induced forgetting (SIF). The neural mechanisms underlying SIF involve increased activation in the right dorsolateral prefrontal cortex and middle frontal gyrus, along with suppressed hippocampal activation. The magnitude of SIF is influenced by the emotional valence of memory materials, individuals' pathological emotional states, and training. Future research should, building upon a deeper understanding of the neural mechanisms of SIF, consider how to enhance SIF effects for clinical pathological memories to achieve therapeutic goals.

Full Text

Influencing Factors and Neural Mechanisms of Suppression-Induced Forgetting

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Abstract: When confronted with unpleasant reminders, people often attempt to prevent unwanted memories from entering awareness. Previous research has shown that suppressing the retrieval of unwanted memories reduces their accessibility, leading to forgetting—an effect known as suppression-induced forgetting (SIF). The neural mechanisms underlying SIF involve increased activation in the right dorsolateral prefrontal cortex and middle frontal gyrus, coupled with suppressed hippocampal activity. The magnitude of SIF is influenced by the

emotional valence of memory materials, an individual' s pathological emotional state, and training. Future research should investigate how to enhance the therapeutic efficacy of SIF for clinical pathological memories based on a deeper understanding of its neural mechanisms.

Keywords: suppression-induced forgetting, emotional valence, emotional state, training
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1 Introduction

Recalling the past is not always a pleasant experience. Many unpleasant memories in daily life can cause significant emotional distress, such as recalling traumatic experiences or embarrassing situations that damage self-image. Numerous emotional disorders are associated with intrusive negative thoughts and memories that impair individuals' ability to regulate negative emotions following exposure to stressors (LeMoult et al., 2010). For instance, both depression and posttraumatic stress disorder (PTSD) are linked to intrusive, uncontrollable negative memories (Nolen-Hoeksema et al., 2008). Therefore, forgetting negative memories is crucial for maintaining mental health. However, forgetting traumatic events is extremely difficult because many environmental cues similar to the traumatic scene can trigger intrusive recollections.

Accumulating evidence demonstrates that people can suppress the retrieval of unwanted memories to reduce recall of their content, a process termed retrieval suppression (Anderson & Huddleston, 2012; Gagnepain et al., 2014). Retrieval suppression decreases the accessibility of unwanted memories and induces forgetting, a phenomenon known as suppression-induced forgetting (SIF) (Benoit et al., 2015; Streb et al., 2016). Mediated by inhibitory control mechanisms, retrieval suppression provides a valuable cognitive model for understanding how people suppress unwanted memories and thoughts in daily life (Benoit et al., 2019; Haghighi et al., 2020). Additionally, research has found that suppressing episodic retrieval reduces the indirect influence of suppressed content on conceptual, emotional, and perceptual processing, which may benefit mental health (Wang et al., 2019).

Laboratory studies of retrieval suppression typically employ the think/no-think (TNT) paradigm developed by Anderson and Green (2001). Based on the go/no-go paradigm, the TNT task comprises three phases: a learning phase, a TNT phase, and a test phase, with learned pairs divided into think, no-think, and baseline conditions. During the learning phase, participants study word pairs (e.g., ordeal-cockroach) until they can recall the target word (cockroach) from the cue word (ordeal). In the subsequent TNT phase, previously learned cue words are repeatedly presented, and participants are asked to either recall the target word (think condition) or avoid thinking about it (no-think condition). Research confirms that participants can employ control mechanisms to suppress target recall in the no-think condition, making subsequent recall more difficult

compared to baseline (Anderson & Green, 2001; Liu et al., 2016; van Schie & Anderson, 2017; Taubenfeld et al., 2019), thereby demonstrating the SIF phenomenon (Anderson & Green, 2001; Anderson et al., 2004; Levy & Anderson, 2008; Hertel et al., 2012; Molet et al., 2016; Streb et al., 2016; van Schie & Anderson, 2017; Wang et al., 2019). SIF effects have been observed across different emotional valences and stimulus types, including words, objects, scenes, and autobiographical memories (Gagnepain et al., 2014; Küpper et al., 2014; Catarino et al., 2015; Wang et al., 2019). Recent TNT research has expanded in three main areas: using more specific stimulus materials to enhance task effectiveness (López-Caneda et al., 2019), investigating the neural mechanisms of retrieval suppression through behavioral studies (Depue et al., 2007; Dieler et al., 2010; Hulbert et al., 2016), and examining differences in SIF effects between patients with emotional disorders and healthy individuals (Sacchet et al., 2017; Sullivan et al., 2019).

Since successfully suppressing intrusive negative memories can modulate emotional responses to them—such as reduced fear reactions (Legrand et al., 2020), decreased emotional experience (Gagnepain et al., 2017), and diminished future fear (Benoit et al., 2016)—studying SIF holds significant importance for alleviating distress caused by intrusive memories. This paper reviews and analyzes relevant research on SIF, concluding that its neural mechanisms primarily involve increased activation in the right dorsolateral prefrontal cortex and middle frontal gyrus, which inhibit hippocampal activity. During suppression of negative materials, the prefrontal cortex also downregulates amygdala activity. The magnitude of SIF is influenced by the emotional valence of memory materials, individual pathological emotional states, and training. Based on this synthesis, we analyze and summarize reasons for inconsistent findings in current SIF research and propose future research directions.

2 Influencing Factors and Neural Mechanisms of SIF

Intrusive memories typically appear in consciousness as distressing images. Understanding the neural mechanisms through which people control these memories and reduce their negative emotional impact is of great significance. Comprehensive review of previous research reveals that current studies primarily focus on how emotional valence of memory materials, individual pathological emotional states, and training affect SIF and its neural mechanisms, which will be discussed in turn below.

2.1 Effects of Emotional Valence on SIF and Related Neural Mechanisms

Early SIF research predominantly used neutral materials, yet the thoughts people avoid in real-life situations are likely negative emotional memories rather than the neutral word pairs typically used in TNT studies. Emotional information automatically captures attention (Blaney, 1986) and facilitates encoding and retrieval (Canli et al., 2001). Consequently, emotional memories form

stronger representations than non-emotional ones. Recent applications of the TNT paradigm have expanded to examine potential differences across various stimulus materials, including negative emotional stimuli. While researchers have used emotional words and pictures to investigate SIF effects, results remain inconsistent. Some studies find stronger SIF effects for negative than neutral information (Depue et al., 2006; Wessel & Merckelbach, 2006; Lambert et al., 2010), possibly because cognitive control over emotional information is stronger than over neutral information (Storbeck, 2013). Other studies find negative information more difficult to suppress than neutral information (Chen et al., 2012), perhaps because negative information is more salient and intrusive (Ritchey et al., 2019), making it harder to inhibit. Still others find no difference in SIF effects between negative and neutral materials (Murray et al., 2015; Benoit et al., 2016; Gagnepain et al., 2017), possibly because neutral and negative materials were matched for associative learning during the encoding phase, eliminating encoding differences and thus obscuring differential suppression effects for emotional materials (Gagnepain et al., 2017). However, this cannot explain the inconsistent results for emotional material suppression, as nearly all studies categorized emotional valence during the learning phase. These inconsistent findings may arise because, although material emotionality and arousal were controlled, emotional experiences show substantial individual differences. Most experiments did not have participants rate the emotional valence and arousal of materials beforehand. Additionally, the emotional materials used varied across studies, with emotional words, faces, and scenes eliciting different emotional experiences and arousal levels and activating different brain regions.

2.1.1 Neural Mechanisms of Non-Emotional Memory SIF When studying SIF with non-emotional materials, researchers have focused on general memory control mechanisms. Two possible pathways for suppression-induced forgetting have been proposed: direct suppression following retrieval inhibition and forgetting caused by attentional diversion (e.g., thought substitution) (Benoit & Anderson, 2012; Küpper et al., 2014; van Schie & Anderson, 2017). However, post-TNT questionnaire surveys of participants' compliance with instructions reveal that nearly all participants reported frequently using suppression strategies consistent with the instructions, leading researchers to conclude that direct suppression strategies can induce forgetting effects (van Schie & Anderson, 2017). Moreover, neuroimaging studies of retrieval suppression provide evidence for forgetting following direct suppression. When participants used thought substitution strategies during the TNT task, activation increased in the caudal prefrontal cortex (cPFC) and left mid-ventrolateral prefrontal cortex (mid-vlPFC) (Benoit & Anderson, 2012), without decreased hippocampal activation (Anderson & Hanslmayr, 2014). In contrast, when using direct suppression strategies, activation increased in the right middle frontal gyrus (MFG) (Depue et al., 2007; Anderson & Hanslmayr, 2014; Depue et al., 2016) and right dorsolateral prefrontal cortex (dlPFC) (Anderson et al., 2004; Benoit & Anderson, 2012; Paz-Alonso et al., 2013; Benoit et al., 2015; Benoit et al., 2016; Liu et

al., 2016; Zhang et al., 2016; Sacchet et al., 2017), while hippocampal activity decreased (Benoit & Anderson, 2012; Anderson & Hanslmayr, 2014; Anderson et al., 2015; Hulbert et al., 2016; Liu et al., 2016; Gagnepain et al., 2017). The right MFG supports memory control processes by reducing momentary awareness of memories and impairing their subsequent conscious recall (Gagnepain et al., 2014). Furthermore, decreased hippocampal activity results from inhibitory modulation by the right MFG (Anderson & Hanslmayr, 2014) and dlPFC (Anderson et al., 2004; Benoit & Anderson, 2012), which suppresses activity in these regions and causes forgetting of suppressed memory traces while reducing their tendency for involuntary intrusion (Benoit et al., 2015). Additionally, Depue et al. (2016) found that during inhibitory regulation, individuals primarily activate the anterior portion of the right MFG, which participates in both emotional and cognitive inhibition, with the cingulate gyrus playing an important role in the right anterior MFG-hippocampus pathway (Depue et al., 2016). When suppressing visual memories, the right MFG also inhibits neural activation in the fusiform cortex (Depue et al., 2007; Gagnepain et al., 2014). Beyond these regions, some medial temporal lobe (MTL) areas are also modulated by retrieval suppression (Anderson et al., 2015; Benoit et al., 2015; Gagnepain et al., 2017), with the magnitude of modulation depending on the type of suppressed memory. For example, when memories are scenes rather than objects or words, the bilateral posterior piriform cortex and right parahippocampal gyrus are inhibited (Benoit et al., 2015). Event-related potential (ERP) studies have also identified two ERP components associated with memory suppression and facilitation processes. Increased N2 amplitude in the no-think condition is related to suppression processes (Bergström et al., 2009; Chen et al., 2012; Zhang et al., 2016), while a larger parietal late positive component (LPC) in the think condition is associated with memory facilitation processes (Bergström et al., 2009; Depue et al., 2013; Zhang et al., 2016). The LPC, an index of conscious recall, predicts successful retrieval when enhanced.

2.1.2 Neural Mechanisms of Negative Emotional Memory SIF Emotional memory suppression involves both memory control and emotion regulation systems. Despite their different goals, these systems engage similar brain regions, such as the right MFG (Gagnepain et al., 2017). Given the amygdala' s crucial role in emotion generation, recognition, and regulation, modulating emotional responses to negative stimuli requires the right MFG to inhibit amygdala activity (Pannu et al., 2010; Anderson et al., 2015). Depue et al. (2007) demonstrated that emotional memory suppression involves at least two pathways: the first includes the right inferior frontal gyrus (IFG) downregulating activation in the fusiform gyrus and thalamic nuclei to exert cognitive control over sensory components of memory representations; the second includes the right MFG downregulating activation in the hippocampus and amygdala to exert cognitive control over both memory processes and emotional components of memory representations (Depue et al., 2007). However, Butler and James (2010) found that suppressing negative stimuli elicited greater activation in the

hippocampus, amygdala, insula, anterior cingulate cortex (ACC), and fusiform gyrus compared to neutral stimuli (Butler & James, 2010). Since hippocampal activation during encoding and retrieval reflects successful conscious recall, hippocampal activation during suppression may indicate stronger memory for negative stimuli despite efforts to suppress them. The ACC participates in conflict and error detection, signaling the need for greater cognitive control (Weiss et al., 2018). Therefore, greater ACC activation during negative memory suppression may reflect increased inhibitory control demands due to conflict between automatic recall and suppression processes (Anderson et al., 2015), or it may indicate more frequent suppression failures leading to greater error detection (Butler & James, 2010). Notably, the regions showing greater activity during negative memory suppression in Butler and James (2010) overlap with those downregulated during successful emotional memory suppression in Depue et al. (2007), further highlighting the importance of these regions for emotional memory.

The discrepancy in hippocampal activation between these studies may relate to differences in suppression training repetition and trial duration. Given that negative memories may be better encoded than neutral memories during the learning phase, Butler and James (2010) used suppression training trials lasting only 2 seconds and repeated only six times—both less than in Depue et al. (2007)—which may have prevented effective suppression of negative memories during the suppression phase. Increased suppression repetitions are associated with greater forgetting (Anderson & Green, 2001) and lower hippocampal activity.

Suppressing negative memories also involves increased dlPFC activation (Dieler et al., 2010; Benoit et al., 2016). Using neutral, positive, and negative word pairs, Dieler et al. (2010) found that suppression processes activated bilateral dlPFC and bilateral ventrolateral prefrontal cortex (vlPFC) regardless of emotional valence. However, when considering emotional valence, suppressing negative stimuli elicited greater activation only in the right dlPFC and right vlPFC compared to emotionally neutral and positive stimuli (Dieler et al., 2010). The right dlPFC and right vlPFC reflect functions of inhibitory control and emotion regulation (Aron et al., 2014; Depue et al., 2016). Therefore, greater activation during negative stimulus suppression may indicate the need for increased cortical control to intentionally inhibit materials with negative valence and high arousal, or it may reflect the need for greater activation to manage additional subcortical interference from the amygdala, which is extra-activated by negative stimuli. Additionally, ERP studies have found that in the no-think condition, negative materials elicit smaller late negativity (LN) and larger late parietal positivity (LPP) compared to neutral materials, with no such differences in the think condition (Chen et al., 2012). LN and LPP appear primarily in the right MFG and right superior frontal gyrus, with LPP reflecting emotional memory effects and relating to intentional recall.

In summary, although SIF effects have been demonstrated across different material types, behavioral and neuroimaging findings on emotional material SIF

remain inconsistent. Current research consistently shows that suppressing both neutral and negative stimuli involves increased activation in the right MFG (Gagnepain et al., 2017) and right dlPFC, with greater activation in the right dlPFC and vlPFC during negative stimulus suppression (Dieler et al., 2010). This suggests right-lateralized prefrontal activity during retrieval suppression, consistent with evidence that right frontal lobe damage impairs intentional forgetting (Jin & Maren, 2015). However, no consistent conclusions have been reached regarding activation patterns of the hippocampus and amygdala during negative stimulus suppression. These inconsistencies may arise because different types of emotional materials elicit varying activation levels, the non-monotonic plasticity hypothesis posits that moderately activated memories in the no-think condition are most likely to be forgotten (Detre et al., 2013), and different memory types may involve different prefrontally-regulated regions (Benoit et al., 2015).

2.2 Effects of Individual Pathological Emotional States on SIF and Related Neural Mechanisms

Many individuals in both clinical and real-world settings experience depression, anxiety, or persistent intrusive traumatic memories that cause continuous suffering. Pathological traumatic memories form the basis of PTSD, repeatedly intruding into patients' consciousness and causing re-experiencing symptoms (Sullivan et al., 2019; van Rooij & Jovanovic, 2019). A hallmark symptom of depression involves selective attention to emotionally negative materials and difficulty suppressing recall of negative events, leading to persistent rumination (Sacchet et al., 2017). Suppressing retrieval of traumatic memories to induce forgetting may have important therapeutic implications for PTSD and depression. Therefore, investigating the characteristics and neural mechanisms of negative memory SIF in patients with depression and PTSD is crucial for clinical treatment of these conditions.

Inhibitory control deficits have been observed in many PTSD patients across various paradigms, including motor response inhibition tasks (Falconer et al., 2013), directed forgetting tasks, and thought suppression paradigms (Hayes et al., 2012; Zwissler et al., 2012; Catarino et al., 2015; Fawcett et al., 2015). However, findings regarding SIF effects in PTSD patients remain inconsistent. Some studies have found that after a period of suppression training, patients recall more traumatic memories compared to baseline—a phenomenon termed the rebound effect (Shipherd & Beck, 2005; Catarino et al., 2015; Mary et al., 2020). For example, Catarino et al. (2015) used negative object-scene pairings and found that PTSD patients showed significantly impaired retrieval suppression ability compared to healthy controls with trauma histories, with SIF deficits positively correlated with PTSD symptom severity. This aligns with clinical observations that patients with more severe PTSD symptoms experience more frequent memory intrusions (Ehlers, 2010). Other studies have found no significant differences in SIF among PTSD groups, trauma-exposed non-PTSD

groups, and non-trauma-exposed controls, though individuals with trauma histories (with or without PTSD) were less likely to achieve successful suppression (Sullivan et al., 2019). These discrepant results may stem from methodological differences: Catarino et al. (2015) used context-related object-scene pairings that increased the likelihood of familiarity-based memory, whereas Sullivan et al. (2019) used unrelated face-scene pairings requiring participants to form new episodic associations, suggesting that the ability to suppress context-related, familiar items may be key to preventing traumatic memory intrusions (Sullivan et al., 2019). Overall, these findings indicate that retrieval suppression plays an important role in post-traumatic emotion regulation: the more effectively traumatic memories can be suppressed, the less frequently unpleasant emotions occur (Anderson et al., 2004; Anderson & Huddleston, 2012; Streb et al., 2016).

PTSD patients exhibit structural and functional damage to the prefrontal cortex (Fani et al., 2012; Terpou et al., 2019). Compared to healthy individuals, PTSD patients show reduced volume in the prefrontal cortex, hippocampus, and amygdala (Pitman et al., 2012). Neurocircuitry models of PTSD suggest that prefrontal regions involved in cognitive control, including the dlPFC, ventromedial prefrontal cortex (vmPFC), and ACC, show reduced activity, leading to inability to regulate emotional regions (amygdala) and resulting in excessive fear responses (Hayes et al., 2012) and impaired adaptive extinction of conditioned emotional responses when cues no longer predict trauma (Pitman et al., 2012). Given the critical role of the prefrontal cortex in SIF, these findings suggest that PTSD patients may have difficulty with dlPFC-mediated inhibitory regulation of the hippocampus, leading to persistent re-experiencing symptoms (Catarino et al., 2015), a view supported by Mary et al. (2020). Additionally, Sullivan et al. (2019) found that impaired suppression ability in PTSD patients may relate to difficulty activating the right MFG, with PTSD patients showing significantly lower right MFG activation than healthy controls during the no-think condition (Sullivan et al., 2019). These impaired neural mechanisms in PTSD patients not only render suppression ineffective but may also increase symptom severity and persistence if intrusive memories are strengthened (Brewin, 2011). Numerous evidence indicates that PTSD is a disorder of neural circuitry rather than single-region dysfunction (Terpou et al., 2019), with different symptom clusters potentially involving distinct neural mechanisms. PTSD re-experiencing symptoms are associated with reduced ACC and IFG activity and enhanced insular function (Lanius et al., 2010), avoidance symptoms related to traumatic cues are linked to reduced cingulate and IFG function plus superior temporal cortex activation (Boeke et al., 2017), and dissociative amnesia symptoms are associated with greater activation in the mPFC, ACC, inferior frontal gyrus, superior and middle temporal gyri, and occipital and parietal lobes (van Huijstee & Vermetten, 2018). Thus, research on cognitive control and retrieval suppression in PTSD must consider specific symptoms. Furthermore, fear conditioning studies show that PTSD patients have impaired extinction abilities, that trauma memory reactivation triggers reconsolidation and thus hinders extinction, and that altered vmPFC volume, function, and left vmPFC-amygdala functional connec-

tivity relate to persistent fear extinction deficits in PTSD (Kaczkurkin et al., 2017). Accordingly, impaired SIF ability in PTSD patients may also result from cue presentation triggering memory reconsolidation during the TNT phase.

To date, findings on SIF effects for negative memories in depressed individuals compared to healthy controls also remain inconclusive. Some studies find that depressed individuals show greater SIF (Joormann et al., 2005), while others find reduced or equivalent SIF (Hertel & Mahan, 2008; Joormann et al., 2009; Zhang et al., 2016; Sacchet et al., 2017). In Sacchet and Levy' s (2017) study, TNT behavioral data showed no differences between depressed and healthy individuals or between neutral and negative material forgetting effects, but fMRI results revealed group differences. Both groups showed greater right MFG activation during suppression compared to healthy controls, regardless of material valence. However, when considering the valence of suppressed materials, the two groups differed in amygdala and hippocampus activation, specifically in clusters spanning the amygdala and anterior hippocampus across both hemispheres. Depressed individuals showed reduced activity in this region when suppressing neutral items relative to negative items, whereas healthy controls showed significantly reduced activity when suppressing negative items (Sacchet et al., 2017). The researchers therefore propose that differential SIF effects in depressed individuals may stem from the need for greater activation within the same neural network to maintain performance levels similar to healthy controls in specific tasks (e.g., working memory tasks) (Harvey et al., 2005). Zhang et al. (2016) found that in the no-think condition, depressed individuals showed smaller N2 amplitudes when suppressing negative pictures compared to controls, indicating failed suppression of negative memory retrieval.

In summary, behavioral and neural mechanisms of retrieval suppression in PTSD and depression patients remain inconsistent. A major reason for these inconsistent conclusions may be the heterogeneity of psychiatric disorders. Individuals diagnosed with PTSD and depression exhibit different symptoms with distinct underlying neural mechanisms (Drysdale et al., 2017; Zandvakili et al., 2020). Therefore, the divergent findings regarding SIF ability in PTSD patients between Catarino et al. (2015) and Sullivan et al. (2019) may also result from differences in symptom severity, symptom types, and trauma event types between the two PTSD samples. In studies of SIF in depressed individuals, Sacchet and Levy (2017) selected patients diagnosed with major depressive disorder according to DSM-IV criteria, whereas Zhang et al. (2016) selected individuals with high anxiety and depressive tendencies using the Beck Depression Inventory and State-Trait Anxiety Inventory. Although current research on retrieval suppression in PTSD and depression patients has attracted scholarly attention, behavioral findings for these populations are inconsistent, and their SIF neural mechanisms require further investigation. Future research should differentiate PTSD and depression patients into subtypes and examine homogeneous groups with identical symptoms, developing more rigorous experimental protocols that address previous limitations to deeply investigate the neural mechanisms of retrieval suppression in these clinical populations,

thereby improving suppression effectiveness in practical applications.

2.3 Effects of Training on SIF and Related Neural Mechanisms

Deficits in retrieval suppression contribute to the development of depression and PTSD (Wong & Moulds, 2011; Marzi et al., 2014; Catarino et al., 2015; Streb et al., 2016; Sacchet et al., 2017). Patients with psychiatric disorders such as depression and PTSD may be unable to effectively use SIF to alleviate symptoms, suggesting limitations in SIF applications. However, relevant research indicates that inhibitory control training can normalize inhibitory control abilities and shape their underlying neural networks (Draganski et al., 2014; Lawrence et al., 2015). Enhanced inhibitory control may result from both automatically formed inhibition and top-down inhibitory control (Lawrence et al., 2015). Like other cognitive skills that benefit from practice, memory control is also subject to training effects (Hulbert & Anderson, 2018). Researchers propose that inhibitory ability relates to trauma experience, with individuals having more trauma experience showing greater inhibitory capacity (Levy & Anderson, 2008; Hulbert & Anderson, 2018). Several lines of evidence support this view. First, longitudinal studies support the notion that moderate trauma may be beneficial: individuals who experience moderate adversity show better mental health and resilience later in life compared to those who experience either excessive adversity or none at all (Seery et al., 2010). People with more real-life experience suppressing memories show better suppression of unwanted memories (Hulbert & Anderson, 2018). Second, evidence for experience-dependent cortical plasticity indicates that cognitive training can produce lasting structural changes (May, 2011; Draganski et al., 2014). Lyoo et al. (2011) found that fire survivors showed selective increases in lateral prefrontal cortex thickness one year later (Lyoo et al., 2011), with cortical thickening regions largely overlapping those involved in retrieval suppression, which predicted PTSD symptom reduction (Anderson & Hanslmayr, 2014). Evidence suggests that hippocampal downregulation increases with retrieval suppression practice, reflecting inhibitory adjustments to intrusive memories (Depue et al., 2006; Depue et al., 2007; Butler & James, 2010). Depue et al. (2007) found that hippocampal memory activity was gradually modulated during the TNT phase: although initial suppression trials showed hippocampal activation above baseline, activity gradually decreased below baseline with increased practice. They proposed that practice may induce qualitative changes in the retrieval suppression network (Depue et al., 2007). Additionally, Benoit et al. (2015) found that effective connectivity between retrieval suppression-related regions changed as participants gained better control over intrusive memories. Individuals who showed negative dlPFC-hippocampus coupling during initial suppression attempts subsequently exhibited fewer intrusions, with this effect diminishing over time as the need to purge involuntary intrusions decreased (Benoit et al., 2015). This suggests that practice may enable more efficient engagement of retrieval suppression neural systems, implying the feasibility of training interventions for memory control (Anderson & Huddleston, 2012).

3 Summary and Outlook

Since Anderson and Green (2001) developed the TNT paradigm, researchers have applied it to investigate SIF from perspectives of emotion, pathology, and training, yielding rich findings. Nevertheless, research on SIF remains a focus of attention because deficits in SIF ability may contribute to PTSD and depression. In recent years, with the development and maturation of cognitive neuroscience techniques, SIF research has made significant breakthroughs and progress, offering insights for treating PTSD, depression, and other disorders. To address current research limitations, future studies should pursue several directions:

3.1 Improving Ecological Validity

Regarding research materials, laboratory studies use weakly associated word, object, and scene pairings to control learning levels, whereas real-life cues that trigger unpleasant memories are typically contextually related to the remembered events. Moreover, due to strong associations formed during trauma memory encoding, trauma-related cues acquire emotional properties and are no longer completely neutral. Laboratory studies of emotional memory SIF typically use neutral cues paired with emotional materials, rarely assessing the emotional valence of cues during the learning phase or even after retrieval suppression. Current TNT research thus appears to focus more on memory connections between cue-target pairs while neglecting emotional connections. Molet et al. (2016) demonstrated affective learning between cues and targets: when faces (cues) were paired with prosocial or antisocial sentences (targets), the faces acquired corresponding positive or negative valence, and individuals not only forgot target sentences in the no-think condition but also reduced the emotional valence of the faces (Molet et al., 2016), providing evidence that retrieval suppression can reduce affective connections between cues and targets. Additionally, shared context is important for memory retention (Preusser et al., 2017). Future research should investigate how context-related materials or emotional cues affect SIF, examining how retrieval suppression influences cue valence and target memory recall when affective learning exists between cues and targets, as well as the underlying mechanisms. It should also consider whether emotional information related to unwanted memories is preserved when episodic memory is impaired and whether this further influences behavior.

Regarding individual states, current research has begun to examine SIF effects in individuals with pathological emotional states such as depression and PTSD, but limitations remain. In reality, people experience various stressful events, such as bereavement, career setbacks, bankruptcy, and exam failure. Due to stress hormones, unpleasant memories are often strongly encoded, and stress hormones can impair the function of brain regions involved in retrieval suppression. High stress levels during trauma event encoding are a major factor in those memory processes. Stress-related hormones can modulate memory for emotional materials, making them more memorable than neutral materials. Both endogenous and exogenous cortisol and norepinephrine enhance memory for emotional

stimuli during encoding and consolidation (Cheung et al., 2015; Wimber et al., 2015). Conversely, high circulating stress hormone levels impair memory retrieval performance (Wimber et al., 2015). Research examining stress effects on intrusive memories during trauma film memory reactivation found that stress and memory activation interact to enhance intrusive memories (Cheung et al., 2015). It remains unclear whether stress promotes or impairs retrieval suppression. Future research should investigate how stress affects SIF for emotional memories and the underlying neural mechanisms.

3.2 Improving Suppression Effects by Integrating Neural Mechanisms and Individual Differences

Given that patients with depression and PTSD suffer from distressing memories, future research should demonstrate the applied value of SIF for these populations. Poor retrieval suppression in depression and PTSD patients is often associated with difficulty activating the right MFG and dlPFC. Research confirms that repetitive transcranial magnetic stimulation (rTMS) can improve cognitive function in depression and PTSD patients (Namgung et al., 2019; Tateishi et al., 2019). Right dlPFC stimulation can effectively improve core PTSD symptoms, possibly by upregulating excitability in prefrontal and hippocampal regions and improving their function (Namgung et al., 2019). Future studies could use rTMS to enhance dlPFC and MFG activity during retrieval suppression to promote SIF. Whether this approach can improve SIF effects in depression and PTSD patients requires investigation. On one hand, the degree of brain region damage differs between depression and PTSD patients, with PTSD patients showing more severe structural and functional damage in regions involved in retrieval suppression. On the other hand, the sources and specific symptom manifestations of the two disorders differ considerably, with PTSD symptoms being more intense, explosive, and impulsive. These factors may lead to differences in SIF ability between the two disorders. Therefore, using rTMS to enhance dlPFC and MFG activity may be more effective for improving suppression ability in depressed individuals. Additionally, differences in SIF ability among depressed patients may be influenced by genetic and schema factors, which future research should examine to deepen understanding of SIF in depression and optimize clinical treatment.

Furthermore, PTSD is a delayed, persistent psychosomatic disorder; individuals do not develop PTSD immediately after trauma. If individuals learn active suppression early after trauma to induce forgetting, could this prevent PTSD development? Future research should investigate this question. Notably, retrieval suppression effects are time-dependent. Some studies show that SIF effects decrease when no-think trial durations are long (van Schie & Anderson, 2017), and that although retrieval suppression can effectively reduce negative thoughts in the short term, its long-term effects are limited (Geraerts et al., 2006; Davidson et al., 2019). Additionally, Freud' s view holds that repression prevents mental content from entering associative areas of consciousness, blocking its

extinction and modification processes, so repressed content remains influential; repression also prevents appropriate emotional release, with unreleased emotions manifesting in various pathological ways. In some cases, thought suppression can exacerbate intrusive memory development. Compared to healthy individuals, psychiatric patients are more likely to show rebound effects when attempting suppression (Magee et al., 2012). The white bear suppression hypothesis also suggests that suppressing a memory makes it more memorable (Wang et al., 2019). However, thought suppression differs from the retrieval suppression strategy used in SIF. A key distinction is that the former explicitly mentions a specific forbidden thought (e.g., white bear) that becomes the task target ("don' t think of a white bear"), so simply remembering the task instructions inevitably violates the task goal, making thought suppression unsuccessful. In contrast, SIF only requires participants to prevent cue-associated memories from entering awareness without mentioning what those memories are, making successful suppression possible (Anderson & Huddleston, 2012; Wang et al., 2019). Nevertheless, given deficits in inhibitory control in PTSD patients and some limitations of SIF effects, clinical application of this retrieval suppression method for PTSD treatment requires caution. Current clinical practice more commonly uses extinction-based cognitive behavioral therapy, which encourages patients to confront trauma-related cues to learn that these cues no longer predict traumatic events, thereby reducing strong emotional responses to them (Kar, 2011). An interesting hypothesis suggests that cognitive behavioral therapy may be effective partly because patients' confrontation with cues and modification of existing thoughts depends on suppression processes (Catarino et al., 2015). If so, training PTSD patients to improve memory control might enhance behavioral therapy effects, a possibility worth exploring in future research.

As reviewed above, appropriate training can improve individuals' ability to manage intrusive memories, and moderate adversity can foster later resilience, suggesting the feasibility of training interventions for inhibitory control. Future research should consider more effective training methods for improving SIF ability. Studies show that mindfulness training is associated not only with improved working memory, executive function, sustained attention, and selective attention, but also with positive effects on emotional processes, emotion regulation, and reduction of negative biases in thought and memory (Brisbon & Lachman, 2017). Mindfulness techniques are thought to modulate cognitive resources in operating and monitoring systems, promoting forgetting by reducing tension from resisting forgetting and helping attentional resources disengage from unwanted items and reallocate elsewhere (Gamboa et al., 2019). Gamboa et al. (2019) conducted mindfulness training before a directed forgetting paradigm and asked participants to use mindfulness strategies to remember or forget target items, ultimately finding no facilitative effect of mindfulness strategies on forgetting. The directed forgetting paradigm differs from the TNT paradigm in that forgetting occurs during encoding rather than retrieval. Some researchers believe mindfulness training only affects memory retrieval processes (Crawley, 2015). Therefore, future research should examine whether mindfulness-based forget-

ting strategies can improve SIF effects and whether individuals with long-term mindfulness practice (e.g., meditation) show greater retrieval suppression ability than untrained individuals.

In conclusion, current research on improving SIF ability is limited. Future studies should further address these issues to enhance SIF applications. Additionally, in terms of neuroimaging techniques, future research could combine fMRI, ERP, and other methods to explore connections among brain networks involved in SIF, investigate differences in brain region activation when suppressing different memory types, and examine activation patterns in relevant brain regions throughout suppression training. Furthermore, studies could examine whether using rTMS to improve function in relevant brain regions can enhance SIF effects, thereby better addressing current research limitations.

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WANG Hongbo (Institute of Cognition, Brain and Health, Henan University, Kaifeng 475004, China) (Institute of Psychology and Behavior, Henan University, Kaifeng 475004, China) (School of Educational Science, Henan University, Kaifeng 475004, China) Abstract: When confronted with reminders of an unpleasant memory, people often try to prevent the unwanted memory from coming to mind. Suppression-induced forgetting (SIF) means that the attempt to prevent unwanted memories from entering awareness results in a decrease in the long-term accessibility of these memories. Previous studies indicated that the suppression of retrieval is accomplished by control mechanisms that inhibit unwanted memories. Suppressing retrieval increased engagement of the right dorsolateral prefrontal cortex and middle frontal gyrus and concomitantly decreased engagement of the hippocampus. The degree of SIF is affected by the emotionality of information and an individual' s emotional state and training. Future studies should investigate ways to improve the therapeutic effects of SIF on clinical pathological memory based on an in-depth understanding of the neural mechanisms of SIF. Key words: suppression-induced forgetting, emotional valence, emotional state, training

Note: Figure translations are in progress. See original paper for figures.