Active Forgetting: Adaptation of Memory by Prefrontal Control

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Abstract
Over the past century, psychologists have discussed whether forgetting might arise from active mechanisms that promote memory loss to achieve various functions, such as minimizing errors, facilitating learning, or regulating one’s emotional state. The past decade has witnessed a great expansion in knowledge about the brain mechanisms underlying active forgetting in its varying forms. A core discovery concerns the role of the prefrontal cortex in exerting top-down control over mnemonic activity in the hippocampus and other brain structures, often via inhibitory control. New findings reveal that such processes not only induce forgetting of specific memories but also can suppress the operation of mnemonic processes more broadly, triggering windows of anterograde and retrograde amnesia in healthy people. Recent work extends active forgetting to nonhuman animals, prefiguring the development of a multilevel mechanistic account that spans the cognitive, systems, network, and even cellular levels. This work reveals how organisms adapt their memories to their cognitive and emotional goals and has implications for understanding vulnerability to psychiatric disorders.
Active forgetting: an internal process that functions to foster retention loss by altering a memory’s state or that of pathways to it

Memory adaptation problem: a need to reduce access to memories when their ongoing accessibility disrupts cognitive or emotional goals, tuning memory to behavior

INTRODUCTION

Over the past century, psychology has taken two broad approaches to explaining how we forget. The first argues that forgetting occurs because of incidental changes that happen to organisms or their environments. Thus, we forget because new experiences lead to memories that interfere with remembering the past; because our contexts change, eliminating cues needed to revive older memories; or because the physical bases of memories decay. The second approach goes beyond this; it posits distinct mechanisms that promote forgetting. Although perhaps counterintuitive, many theories include active forgetting processes. In experimental psychology, the classical two-factor theory of interference proposed that intruding memories were unlearned to prevent future interference (Melton & Irwin 1940); later modifications of this theory proposed that whole sets of memories could be suppressed to limit their interference with new learning (Postman et al. 1968). Pioneering work using the directed forgetting procedure led Robert and Elizabeth Bjork to conclude that memories could be intentionally forgotten and that this retrieval inhibition was an adaptive feature of memory (Bjork 1989, Bjork et al. 1998). In clinical psychology, Freud argued that memories could be repressed to reduce psychological conflict [Breuer & Freud 1955 (1895)], highlighting emotional motives that people have to forget. These proposals underscore an unavoidable truth: Memories are, at times, too accessible for our own good. They highlight how the state of memory must often be actively tuned to suit our cognitive or emotional goals. We herein refer to this need as a memory adaptation problem. Given that memory adaptation problems arise, explaining all forgetting passively may not suffice because adapting memory could engage unique control processes that trigger forgetting. Active forgetting proposals traditionally have rested on behavioral findings, however, which necessarily provide indirect evidence for any novel processes proposed.

In this review, we argue that new methods in cognitive neuroscience provide an innovative window into active forgetting processes as they modulate brain structures supporting memory. By tracking the neural dynamics of control, they confirm long-hypothesized suppressive processes and allow a more specific account of memory adaptation. A key conceptual advance has been to view active forgetting not as a consequence of processes unique to memory but as a side
Inhibitory control: a mechanism that enables organisms to override reflexive actions, memories, or emotions by deactivating representations or processes underlying them.

Amnesic shadow: impaired recall or source recognition of events occurring before or after direct retrieval suppression, arising from disrupted hippocampal function.

Prefrontal control hypothesis: the proposal that some forms of active forgetting originate from top-down control signals mediated by the prefrontal cortex.

**INHIBITORY CONTROL OVER MEMORIES, PROCESSES, AND CONTEXTS**

Many modern accounts attribute active forgetting to mechanisms that allow people to control their behavior, such as inhibitory control. Inhibitory control enables humans and other organisms to override strong habitual (prepotent) responses when they become inappropriate (e.g., Aron et al. 2014, Boucher et al. 2007, Diamond 2013, Fuster 2015, Logan & Cowan 1984). The ability to override habitual responses enables flexible, goal-directed control over behavior without which we would be dominated by habits and reflexes. Inhibitory control supports at least two functions essential to directing behavior: selection and stopping (Figure 1a). During selection, we must isolate one response that is appropriate for current goals from competitors vying for control over behavior. During stopping, we must cancel a single dominant response that is inappropriate in a particular context or that, during execution, has become undesirable. We and other authors have proposed that these functions of inhibitory control extend to regulating internal cognitive actions such as memory retrieval (Anderson 2003, Anderson & Hanslmayr 2014, Anderson & Spellman 1995). Thus, just as stimuli elicit reflexive actions that may need stopping, reminders can evoke unwanted memories. A major theme of this review is that selection and stopping demands pervade the normal use of memory, setting occasions to inhibit memories that impede behavior and thought. These acts of control adapt patterns of accessibility to align memory with current behavioral and emotional goals.

When discussing brain mechanisms underlying inhibitory control, we refer to structures believed to initiate a top-down inhibitory control signal as a source of control; in contrast, we refer to regions supporting the to-be-controlled representations or processes as inhibition sites. Most work on active forgetting attributes the source of inhibitory control to the lateral prefrontal cortex, an idea we refer to as the prefrontal control hypothesis. This hypothesis is motivated by the established involvement of the prefrontal cortex in action stopping, an idea first proposed by Ferrier in the nineteenth century (for historical reviews, see Bari & Robbins 2013, Diamond et al. 1963) that has received voluminous support in research with humans and nonhuman animals (Aron et al. 2014, Diamond 2013, Fuster 2015). If active forgetting draws upon control mechanisms engaged during response inhibition, then shared anatomical localization and physiological characteristics...
Memory inhibition:
active forgetting arising from the impact of inhibitory control acting on representations of the forgotten memory

Figure 1
Schematic overview of the three-factor model of active forgetting. Active forgetting may be achieved through inhibition of the distracting memory representations (a, left). When memories share a common reminder, selectively retrieving one (weaker, especially) memory may inhibit competing memories (bottom, grayed-out circle), yielding retrieval-induced forgetting. Attempts to stop the automatic retrieval of a target memory in the face of a strong reminder (a, right) may also inhibit the unwelcome memory representation (bottom, grayed-out circle), causing suppression-induced forgetting. Suppressing retrieval can also inhibit mnemonic processes (b), e.g., encoding, stabilization, and retrieval, as has been observed around periods of direct memory suppression, impairing retention of memories that would otherwise rely on those processes—even unrelated bystander memories that are not the focus of suppression (bottom, grayed-out box and circles). The mental context associated with items on a list (small yellow circles in a context buffer of c) may be purged by inhibitory control (c, bottom), with new contextual elements (small green circles) replaced in the context buffer and associated with new experiences (teal circle). With less contextual cue support, associated memories (such as Memory 1) are harder to access, despite the underlying representation remaining unchanged.

should emerge, supporting a domain-general inhibitory control mechanism. We argue that evidence for domain generality exists.

Prefrontal control may contribute to active forgetting in several ways. Here, we introduce the three-factor theory of active forgetting, distinguishing three impacts of inhibitory modulation: memory, process, and context inhibition. Memory inhibition refers to the idea that inhibitory control affects a specific memory, reducing its accessibility (Figure 1a). Process inhibition, by contrast, would not solely affect a specific memory but rather would disrupt a memory process needed for retention (Figure 1b). If inhibitory control broadly suppressed episodic encoding, consolidation, or retrieval processes, for example, it would disrupt every memory reliant on the affected process. Although this suppression may be triggered to forget a particular unwanted trace, it has a global and systemic impact. Finally, with context inhibition, a memory becomes less accessible because the prefrontal cortex inhibits the mental context necessary to retrieve it (Figure 1c). Context inhibition predicts that affected memories remain intact in storage despite being inaccessible. The
Process inhibition: active forgetting arising from the impact of inhibitory control acting on processes necessary to form, stabilize, or retrieve a memory

Context inhibition: active forgetting arising from the impact of inhibitory control acting to purge context representations used to access a memory

Selective retrieval: the process of retrieving a particular target memory, given a cue that is associated with many competing memory traces

Retrieval-induced forgetting (RIF): the tendency to forget those memories that interfere with access to memories one needs to consistently retrieve

context inhibition hypothesis builds on influential work by Sahakyan and colleagues (Sahakyan & Kelley 2002, Sahakyan et al. 2013) showing that some forms of active forgetting arise from mental context shifts, although the emphasis on inhibition is our own. We argue that neural and behavioral phenomena exist for each of these targets of active forgetting.

ACTIVE FORGETTING BY MEMORY INHIBITION

To test whether memories can be actively forgotten, researchers have focused on long-term memory situations likely to engage inhibitory control. Typical approaches require people first to control competition from distracting memories and then to remember the distracting content. If controlling distraction leads people to forget the unwanted content and if forgetting exceeds what would be expected based on time alone, active processes may be at work. Behavioral research has identified such forgetting phenomena and has tested whether their functional profiles accord with an active process. Neuroscience research has provided a critical window into the brain mechanisms underlying this process. This work not only has revealed how active forgetting relies on the prefrontal cortex but also has (a) tracked competing memories as they are suppressed, using multivariate imaging methods; (b) linked active forgetting to inhibitory neurotransmitters that enable prefrontal control; and (c) tested the causal necessity of the prefrontal cortex in inducing forgetting. Two lines of work have examined active forgetting during selective retrieval and retrieval stopping.

Inhibition of Competing Memories During Selective Retrieval

Recalling a particular event or fact may place demands on inhibitory control. Inhibitory control may be needed if the cues guiding retrieval activate other memories in addition to the one we seek. When this happens, remembering poses problems similar to those arising during motor action when a single response must be selected from among competitors (Figure 1a). Indeed, most theorists assume that when cues activate multiple memories, the traces compete for access to awareness, a dynamic identified early in memory research (McGeoch 1942). Retrieval competition, if left unchecked, can cause retrieval to fail or even allow mistaken recall of competing memories. Thus, overcoming mnemonic distraction by inhibitory control is a valuable capacity but may also invite active forgetting. Indeed, this situation prompted early theorists to hypothesize active forgetting (Melton & Irwin 1940, Postman et al. 1968).

A large body of work shows that selective retrieval triggers forgetting of competing memories, a phenomenon known as retrieval-induced forgetting (RIF) (Anderson et al. 1994). In a simple example, if participants retrieve several members of a studied category repeatedly (e.g., Fruit-Orange, Fruit-Lemon), later recall of nonpracticed exemplars (e.g., Fruit-Banana, Fruit-Cherry) often suffers. Repeated retrieval, known as retrieval practice, usually involves cued-recall trials providing the category (e.g., Fruit) and a distinctive stem to focus people on the retrieval target (e.g., Fruit-Or__). On retrieval practice trials, the category cue is thought to partially activate all studied exemplars, competition from which hinders recall of the target. Overcoming this distraction during retrieval practice trials reduces later memory for the competitors, compared to the retention of baseline categories (e.g., Sports-Soccer) that were also studied but none of whose members received retrieval practice, illustrating RIF. RIF reveals that competing memories suffer forgetting arising simply because they happen to be related to other memories that people retrieve consistently. These costs may arise because competitors interfered with retrieval practice, making them targets of inhibitory control (for alternative and complementary mechanisms, see Anderson & Bjork 1994, Norman et al. 2007).

RIF is highly general. It occurs whenever people must selectively retrieve a memory despite distracting retrieval competition. RIF occurs for visual objects (Ciranni & Shimamura 1999), facts
Functional properties of retrieval-induced forgetting that support active inhibition. Over the past three decades, the functional characteristics of RIF have been carefully studied. Considerable evidence shows that RIF exhibits properties consistent with an active inhibition process that is engaged to suppress competing memories, rendering them generally less accessible. Quantitative meta-analysis confirms many of these properties (Murayama et al. 2014), and many extend to other species. Because these functional properties are reviewed in multiple sources (Anderson 2003, Bäuml & Kliegl 2017, Marsh & Anderson 2020, Murayama et al. 2014, Storm & Levy 2012), we limit discussion to the essential features of each, with illustrative examples.

One important property of RIF is retrieval specificity, which refers to the importance of active retrieval in promoting forgetting. For example, whereas repeatedly studying to-be-practiced items (e.g., Fruit-Orange) tends not to induce forgetting of competitors, retrieval practice does (whether achieved by recall or by recognition practice) (e.g., Maxcey & Woodman 2014). Retrieval specificity aligns well with a special role for inhibitory control in resolving retrieval competition and arises with diverse materials. Intriguingly, whereas the retrieval attempt is critical to forgetting, retrieval practice success is not: Even when retrieval practice cues are impossible to complete, competitors are inhibited (Storm et al. 2006). RIF also is interference dependent because it is triggered by interference from competitors during retrieval practice. For example, competitors that are weakly associated with the retrieval practice cues suffer little RIF, suggesting that only distracting memories trigger inhibition. Interference dependence can be shown by varying the interference characteristics of the materials but also by manipulating how much interference competitors could cause during retrieval practice. For example, Chan and colleagues (2015) showed that, whereas retrieval practice impairs competitors learned before retrieval practice, competitors learned afterward escape unscathed. If competitors are not in memory during retrieval practice, they cannot interfere and trigger inhibition. Interference dependence also has been supported by neuroimaging measures and in an animal model of RIF, discussed shortly.

The contribution of noninhibitory forgetting factors to RIF has been considered carefully (see, e.g., Anderson & Bjork 1994). For example, retrieving a memory makes it more accessible, reflecting retrieval-based learning (Roediger & Butler 2011). Perhaps retrieval practice benefits come at a cost, with stronger practiced memories thwarting access to competitors on the final test, a possibility known as blocking. If so, RIF could arise from blocking, an exaggerated form of competition. Two properties speak against the sufficiency of this type of passive forgetting. First, strength independence shows that RIF is unrelated to retrieval practice benefits. Strength
independence is supported by (a) the lack of correlation between RIF and the facilitation of practiced memories, even with hundreds of participants (Aslan & Bäuml 2011, Hulbert et al. 2012); (b) retrieval specificity, which shows that strengthening memories via extra study leaves competitors unaffected; (c) interference dependence, which shows that retrieval-based strengthening exerts little effect when competitors are weak; and (d) the presence of RIF even when retrieval practice is impossible. Critically, however, blocking may contribute to RIF, especially when final tests do not control output interference (Anderson & Levy 2007, Marsh & Anderson 2020, Murayama et al. 2014, Schilling et al. 2014).

Selective retrieval often induces RIF that generalizes across cues, a property known as cue independence (Anderson 2003, Anderson & Spellman 1995). For example, retrieval practice on Fruit-Orange induces forgetting of competitors (e.g., Banana) when later tested not only with Fruit (e.g., Fruit-B___) but also with cues unrelated to the category (e.g., Monkey-B___). Occurring with many stimuli (for reviews, see Marsh & Anderson 2020, Murayama et al. 2014, Storm & Levy 2012), cue independence indicates that active forgetting affects the competing trace itself and does not act solely on the association linking the cue and the memory. Cue independence also argues against simple blocking accounts (for reviews of theoretical implications of cue independence, see Anderson 2003, Anderson & Bjork 1994, Anderson & Spellman 1995). This property is among the strongest indicators that RIF reflects an active process affecting the competing memory. Consistent with this interpretation, a large body of work shows that RIF occurs on item recognition (for a review, see Spitzer 2014) and also implicit memory tests (e.g., Gómez-Ariza et al. 2017). More broadly, many of the foregoing properties also can be triggered by semantic retrieval (Johnson & Anderson 2004) and in the retrieval of motor actions (Tempel & Frings 2015). Thus, RIF is not an episodic memory (EM) phenomenon but appears to be system general (Marsh & Anderson 2020).

Several findings link cognitive control to the foregoing evidence for inhibition in RIF, illustrating its attention dependence. First, dividing attention during selective retrieval with an updating task that requires executive control abolishes later RIF on competing memories (Ortega et al. 2012, Román et al. 2009). This finding suggests that selective retrieval and updating share demands on control processes that cause inhibition. Second, the amount of RIF found on cued-recall and item recognition tests increases with better working memory span (Aslan & Bäuml 2011) and better stop-signal reaction time (Schilling et al. 2014), a measure of inhibitory control over action. These findings suggest that a shared control process may inhibit actions and memories. Third, RIF can be abolished selectively without affecting retrieval practice performance or facilitation of practiced items by increasing stress prior to retrieval practice or depriving smokers of nicotine (Edginton & Rusted 2003, Kössler et al. 2009). Finally, participants with attention-deficit/hyperactivity disorder show little RIF, despite intact facilitation of practiced items (Storm & White 2010). RIF’s functional profile thus indicates an inhibition process mediated by cognitive control, perhaps supported by the prefrontal cortex.

**Brain imaging evidence for adaptive forgetting by prefrontal control.** Selectively retrieving a memory engages the lateral prefrontal cortex. For example, brain imaging and neuropsychological work tie the left ventrolateral prefrontal cortex (VLPFC), especially Brodmann area (BA) 45, to the need to isolate a single item from one or more competitors during semantic retrieval (Badre & Wagner 2007, Thompson-Schill et al. 1997). Similarly, resolving interference during episodic retrieval activates the lateral prefrontal cortex, although studies vary in whether the left VLPFC, right VLPFC, or both are engaged (e.g., Dulas & Duarte 2016; Kuhl et al. 2008, 2010). Damage to the lateral prefrontal cortex disrupts episodic retrieval, especially given interference
Conflict reduction benefit: a beneficial reduction in response conflict and cognitive control costs resulting from actively forgetting overly accessible and disruptive memories.

Shimamura et al. 1995, Simons & Spiers 2003. Thus, selective retrieval tasks that trigger RIF would be expected to engage the lateral prefrontal cortex.

Many studies have confirmed this expectation. Indeed, functional magnetic resonance imaging (fMRI) and electrophysiological studies link RIF to prefrontal regions that resolve retrieval competition and also highlight its adaptive benefits. Consider a landmark study by Kuhl and colleagues (Kuhl et al. 2007). Participants first studied cue-associate word pairs, encoding multiple associates for each cue. Next, they performed selective retrieval practice, retrieving some of the associates of some cues repeatedly as fMRI scanning occurred. As in the conventional RIF procedure, of the associates that were not practiced, some were competitors to items receiving retrieval practice (i.e., they shared a cue), whereas others were not. Finally, after a delay, memory for all of the cue-associate pairs was tested. As expected, retrieval practice induced forgetting of competitors on this test, confirming that RIF had occurred.

Kuhl and colleagues examined whether prefrontal regions engaged during selective retrieval predict active forgetting and whether such forgetting yields adaptive benefits visible in neural activity. They hypothesized that forgetting competitors should reduce the interference they cause during future retrievals of the same target memories. If so, then forgetting should reduce metabolic demands on prefrontal control mechanisms that overcome competition. In essence, forgetting competitors would tune memory to retrieve recurrently useful traces efficiently. Thus, as people retrieved the same memories repeatedly during retrieval practice, blood-oxygen level dependent activation in lateral prefrontal control regions should have declined, in relation to how much RIF people showed on the later test. Importantly, Kuhl and colleagues also focused on the anterior cingulate cortex (ACC), a brain region involved in detecting conflict, as a window into how interfering competitors are; thus, declining ACC activity over repeated retrievals may be a bellwether for the later forgetting of competitors, as competition is gradually suppressed.

These predictions were strongly confirmed. Activation in bilateral VLPFC, right DLPFC, and ACC declined from the first retrieval practice trial to the third, consistent with reduced demands on cognitive control and conflict, respectively. The more ACC declined, the larger the reduction in lateral prefrontal activity (DLPFC and, to a lesser degree, VLPFC). Of course, this decline could reflect practiced items growing highly accessible with practice, due to retrieval-based learning. However, declines in left ACC (BA32, BA24) and right VLPFC activity correlated selectively with RIF and not with the strengthening of practiced memories. Thus, successfully forgetting distracting memories reduced putative ACC conflict signals and also prefrontal control demands. Interestingly, the most successful forgetters exhibited higher ACC activity in the first retrieval practice trial than did those showing little RIF. If ACC activity indexes conflict, this suggests that high forgetters forgot in order to combat elevated competition from related memories. Supporting this interpretation, higher hippocampal activation predicted elevated ACC activity on the first retrieval practice, suggesting that conflict signals could have been triggered by hippocampal retrieval processes.

The foregoing findings suggest that prefrontal regions involved in conflict detection and its resolution contribute to RIF. Importantly, they illustrate the adaptive effects of active forgetting, including reduced demands on metabolically costly control mechanisms and improved retrieval efficiency. We introduce the term conflict reduction benefit (see Table 1), to refer to reduced strain on control resources over repeated control attempts, arising from successful forgetting (see also Wimber et al. 2011, 2015). The size of the conflict reduction benefits in right VLPFC activity (and RIF) is linked to genetic variation in prefrontal dopamine (Wimber et al. 2011), a neurotransmitter important to behavioral flexibility (Robbins & Kehagia 2017). Both the lateral prefrontal and ACC regions that benefit from forgetting are tied to retrieval: They are activated more by...
| Benefit                                                                 | Measure                                      | Interpretation                                                                 | Paradigm | Example citation(s)               |
|------------------------------------------------------------------------|----------------------------------------------|--------------------------------------------------------------------------------|----------|-----------------------------------|
| Conflict reduction (comparing early retrieval practice or retrieval suppression trials with later ones) | ACC BOLD reduction                           | Conflict by competitors is reduced as forgetting occurs                        | RIF      | Kuhl et al. 2007; Wimber et al. 2011, 2015 |
|                                                                         | Lateral PFC BOLD reduction                   | Control grows less necessary as competitors are forgotten                      | RIF      | Kuhl et al. 2007; Wimber et al. 2011, 2015 |
|                                                                         | Mid-frontal theta power reduction            | EEG effect reflecting reduced ACC conflict signals with forgetting             | RIF      | Ferreira et al. 2014, Hanslmayr et al. 2010, Staudigl et al. 2010 |
|                                                                         | Reduced pupil diameter                       | Control grows less necessary as competitors are forgotten                      | RIF      | Johansson & Johansson 2020        |
|                                                                         | Reduced cFos expression in rodent mPFC       | Control grows less necessary as competitors are forgotten                      | RIF      | Bekinschtein et al. 2018          |
|                                                                         | Reduced fronto-hippocampal coupling          | As intrusive memories are suppressed, hippocampal retrieval no longer needs to be inhibited | SIF      | Benoit et al. 2015                |
|                                                                         | Reduced frontal negative slow wave           | As intrusive memories are suppressed, working memory is no longer consumed by intruding memories | SIF      | Hellerstedt et al. 2016           |
| Intrusion reduction effect                                              |                                              | As competitors are forgotten they no longer intrude as often, given reminders | SIF      | Benoit et al. 2015, Gagnepain et al. 2017, Harrington et al. 2020, Legrand et al. 2018, Levy & Anderson 2012, Mary et al. 2020, van Schie & Anderson 2017 |
| Affect reduction (comparing suppression versus baseline items)          | Reduced subjective valence                   | As intrusions are controlled, emotional content is disrupted                   | SIF      | Gagnepain et al. 2017, Harrington et al. 2020, Legrand et al. 2018 |
|                                                                         | Reduced skin conductance response            | As intrusions are controlled, emotional content is disrupted                   | SIF      | Harrington et al. 2020            |
|                                                                         | Reduced heart-rate deceleration              | As intrusions are controlled, emotional content is disrupted                   | SIF      | Legrand et al. 2018               |
| Reduced perceptual distraction (comparing suppression versus baseline items) | Reduced attentional capture                 | Cortical memory traces that ordinarily facilitate attention to recent items are suppressed, reducing capture | SIF      | Hertel et al. 2018                |
|                                                                         | Reduced perceptibility                       | Cortical memory traces that ordinarily facilitate attention to recent items are suppressed, reducing perceptibility | SIF      | Gagnepain et al. 2014, Kim & Yi 2013, Mary et al. 2020 |
| Learning (comparing learning after Forget versus Remember instruction)  | Reduced proactive interference               | A change in internal context limits interference from prior encoding and enables undistracted focus on new material | LM DF    | Pastötter et al. 2012             |
|                                                                         | Resetting of attention and encoding          | Inhibition of a context re-engages attention and encoding, improving learning  | LM DF    | Pastötter et al. 2008             |

Abbreviations: ACC, anterior cingulate cortex; BOLD, blood-oxygen level dependent; EEG, electroencephalography; LM DF, list-method directed forgetting; mPFC, medial prefrontal cortex; PFC, prefrontal cortex; RIF, retrieval-induced forgetting; SIF, suppression-induced forgetting.
selective retrieval than by repeated study of the same items (Wimber et al. 2008), consistent with the retrieval specificity of the inhibitory processes underlying RIF.

Conflict reduction benefits also arise in electrophysiological and psychophysiological studies of RIF. Oscillatory activity in the theta band (5–9 Hz) measured with scalp electroencephalography (EEG) increases in cognitive interference tasks, with theta power over midline prefrontal cortex often source-localized to the ACC. For example, interference in Stroop and Flanker designs increases theta power (e.g., Cavanagh et al. 2009, Hanslmayr et al. 2007). Similarly, in RIF studies, selective retrieval practice robustly increases midline-frontal theta power more than does repeated study of the same items (Staudigl et al. 2010) or retrieval practice without competitors (Ferreira et al. 2014, Hanslmayr et al. 2010). Critically, paralleling conflict reduction benefits in fMRI, Staudigl et al. (2010) found that midline-frontal theta power declined over retrieval practice blocks (see also Ferreira et al. 2019), with steeper declines predicting greater RIF. These effects were source-localized to the ACC. In contrast, repeated study elicited no conflict reduction benefits or forgetting. Research has isolated this theta effect to mnemonic conflict, rather than its control (Ferreira et al. 2014). Thus, by indexing competition, midline-frontal theta reveals the conflict reduction benefits of active forgetting. Analogous benefits are observed in psychophysiological indices of attentional control, such as pupil diameter, which increase during retrieval competition (Johansson et al. 2018) and decline over retrieval practice trials, with steeper declines predicting larger RIF (Johansson & Johansson 2020).

Conflict reductions support a role of inhibitory control in suppressing competing memories. More specific support for inhibition, however, requires measuring the competing memory representations themselves, rather than upstream influences on conflict detection. But where are competing memories stored? What brain activity do we examine? Innovative work has addressed this problem using multivariate imaging analysis (Wimber et al. 2015). This work starts with a simple observation: Memories usually contain remnants of what we saw during the event. Given this, measuring activity patterns in perceptual areas while a person views pictures may allow us to compute a canonical pattern template to serve as the marker for how a person’s brain responds to each picture. To build these templates, Wimber and colleagues (2015) repeatedly exposed people to famous faces (e.g., Marilyn Monroe, Albert Einstein), famous places (e.g., the Taj Mahal), and everyday objects (e.g., goggles, a hat) during fMRI. Wimber et al. averaged across repetitions to construct the canonical template for each picture over voxels in the ventral temporal cortex, a brain region representing the content of interest. This activation matrix constitutes a definition of what one should find given a perfectly remembered image of Marilyn Monroe or the Taj Mahal, for example. During retrieval, Wimber et al. measured how well the ventral temporal cortex pattern matched this template as an item-specific index of memory reactivation (Figure 2).

Wimber et al. used these pattern templates to track competition during selective retrieval and its resolution by control. Participants learned associations between cue words and pictures, with each cue paired with two competing pictures from distinct categories (e.g., a face and a scene). Next, participants performed selective retrieval practice on some cues: On each trial, the cue appeared, and participants retrieved the first picture they had associated with it. Participants performed four retrieval practice trials on each practiced item while undergoing fMRI scanning. Replicating prior work (for a review, see Spitzer 2014), Wimber et al. found RIF on a recognition test for pictures. Across the four retrieval practice trials, participants also showed robust conflict reduction benefits in bilateral VLPFC and ACC. Did these benefits arise from suppressing competitors? To test this, they examined how retrieval practice affected the competitor itself; during each retrieval practice trial, they computed how similar the activation pattern was to the competitor’s template, allowing an estimate of whether the cue reinstated the competitor. Strikingly, evidence for the competitor’s pattern increased on the first retrieval trial but then declined over
the remaining three selective retrievals. This decline was associated with both conflict reduction benefits in lateral prefrontal cortex and RIF on the later test. A control analysis demonstrated that reduced pattern reinstatement was driven by suppressed activity in those voxels most diagnostic of the particular picture (Figure 2e), suggesting that declines in pattern similarity originated from suppression.

The foregoing findings suggest that conflict reduction benefits in the prefrontal cortex reflect, in part, the benefits of competitor suppression, illustrating the adaptive nature of forgetting. The robust activation in right DLPFC and VLPFC has led to research confirming their causal role in inducing active forgetting. For example, electrical stimulation over right DLPFC or VLPFC via transcranial direct current stimulation prior to retrieval practice abolishes RIF, which is reduced relative to sham stimulation (Penolazzi et al. 2014, Stramaccia et al. 2017, Valle et al. 2020). RIF is abolished even though stimulation leaves retrieval practice success and retrieval-based learning of
Retrieval suppression: a controlled attempt to cancel an episodic retrieval when a reminder begins to elicit automatic retrieval of unwelcome content

Suppression-induced forgetting (SIF): the tendency to forget memories that one tries to exclude from awareness when confronted with unwelcome reminders

Inhibition of Intruding Memories During Retrieval Stopping

In addition to selective retrieval, inhibitory control may also be engaged when cues remind us of unwelcome memories or thoughts that are unpleasant or distracting. When such remindings occur, people often try to stop retrieval to limit awareness of the intruding memory, a behavior referred to as retrieval suppression. Stopping retrieval in this manner resembles the process of action stopping, except that people seek to control retrieval rather than behavior (Figure 1a). Given these parallels, stopping retrieval may evoke prefrontal inhibitory control mechanisms and trigger active forgetting of the inhibited content. If retrieval stopping induces active forgetting, this process may adapt memory to a person's emotional or task goals. Conversely, failing to forget may underlie difficulties with intrusive memories, worries, or ruminations, hallmark symptoms of many psychiatric conditions. These symptoms may be viewed as failures to solve memory adaptation problems. Understanding whether and how retrieval suppression induces active forgetting may thus have significant clinical implications.

Retrieval suppression often induces forgetting of unwanted memories, known as suppression-induced forgetting (SIF) (for reviews, see Anderson & Hanslmayr 2014, Anderson & Huddleston 2011, Marsh & Anderson 2020). This form of active forgetting is studied through the Think/No-Think (TNT) paradigm (Anderson & Green 2001, Anderson et al. 2004). Participants are trained on cue-target pairs until they can recall the targets, given the cues. Pairs can be words (e.g., Ordeal-Roach), pictures, or even autobiographical materials. Participants then perform the TNT task, which requires retrieval stopping. On each trial, a reminder from a pair appears in green or red. For green reminders (Think trials), participants must remember the paired item and retain it in awareness; for red reminders (No-Think trials), participants are asked to prevent the paired item from entering awareness. Because the reminders and paired items were associated, seeing the cue will elicit automatic retrieval, which in the No-Think condition is unwelcome. This conflict between automatic retrieval and the goal to not retrieve is hypothesized to trigger inhibitory control. Participants suppress or retrieve a given item many times, allowing inhibition to build in the former case. A final test assesses memory for all pairs, with recall measured on Think (repeatedly retrieved), No-Think (repeatedly suppressed), and Baseline items that were studied but that did not appear in the TNT phase.
Retrieval suppression typically impairs recall of No-Think items, compared to Baseline items, yielding SIF. Thus, even though reminders for No-Think items appear frequently in the TNT task, suppressed items are recalled less than Baseline items, which never had reminders presented. This pattern suggests that No-Think reminders evoked processes that countermanded retrieval, disrupting suppressed items, consistent with inhibitory control. SIF has been reported for words, visual objects, scenes, and autobiographical memories, and for neutral and negatively valenced content (for a review, see Marsh & Anderson 2020). SIF occurs on cued-recall tests and indirect memory tests including perceptual identification, free association, and category verification, as well as on remote association tests that tap conceptual content.

Functional properties of suppression-induced forgetting that support active inhibition.

Several characteristics of SIF suggest inhibition as a cause. In fact, the properties of RIF that support inhibition also are true of SIF. For example, SIF is often cue independent, suggesting a generalized forgetting consistent with disruption to the memory. Anderson & Green (2001), for example, found that after learning pairs such as Ordeal-Roach, if participants later suppressed Roach when cued with Ordeal, Roach was forgotten regardless of whether it was tested with Ordeal or Insect-R___. Thus, suppression induces forgetting that generalizes across cues. Cue independence arises on cued-recall tasks with extralist or intralist associates of various kinds, and forgetting even generalizes to indirect tests (for reviews, see Marsh & Anderson 2020, Wang et al. 2019). However, cue independence is most consistent when participants receive direct suppression instructions (Benoit & Anderson 2012, Bergström et al. 2009). With these instructions, participants are asked, during No-Think trials, to refrain from generating distracting thoughts while avoiding retrieval and to instead suppress the intruding memory if it emerges into awareness.

A clear illustration of how suppression counters intrusive memories comes from studies that quantify intrusions as people suppress retrieval. To identify intrusions, the TNT task is modified to include intrusion reports after each trial, wherein participants classify whether the unwanted item entered awareness despite efforts at control. The tendency for memories to come to mind despite efforts at control vividly illustrates the automatic retrieval that inhibitory control is recruited to counter. Intrusions occur frequently during early suppressions (e.g., 60%) (Levy & Anderson 2012) but tend to be well controlled (although usually not eliminated) after repeated attempts (e.g., 30%). We herein refer to this reduction as an intrusion-control effect. Such effects partially reflect the aftereffects of inhibitory control on excluded memories, making them less likely to intrude. Consistent with this, intrusion-control effects often predict later forgetting (Hellerstedt et al. 2016, Levy & Anderson 2012), illustrating the interference dependence of SIF. However, this effect also partially reflects proactive control, which may not involve inhibition (for discussion, see Levy & Anderson 2012).

SIF derives substantially from inhibition and cannot be explained solely by passive interference. Interference could, however, play a role. For example, confronting reminders and avoiding retrieval can involve generating substitute thoughts. Those thoughts might grow associated to the reminder and block the avoided memory on the recall test (Anderson & Green 2001, Hertel & Calcaterra 2005), a form of passive interference. Although blocking contributes to SIF when people use thought substitution (e.g., Hertel & Calcaterra 2005, Wang et al. 2015), SIF does not require substitute thoughts. For example, direct suppression instructions discourage participants from generating substitutes yet generate significant SIF (for a meta-analysis, see Stramaccia et al. 2019). Moreover, direct suppression and thought substitution have been electrophysiologically (Bergström et al. 2009), hemodynamically (Benoit & Anderson 2012), and behaviorally dissociated (Hertel & Hayes 2015, Hulbert et al. 2016, Wang et al. 2015), indicating that distinct mechanisms induce these forgetting phenomena (Marsh & Anderson 2020) (for a summary of dissociations,
SIF appears to be attention dependent. For example, dividing attention during retrieval suppression abolishes cue-independent SIF (Noreen & de Fockert 2017). Individual differences in SIF also correlate with stop-signal reaction time on motor stopping tasks (Schmitz et al. 2017), indicating a relationship with inhibitory control ability. Finally, reduced SIF is observed in attention-deficit/hyperactivity disorder, suggesting that suppression depends on attentional control (Depue et al. 2010).

**Brain imaging evidence for adaptive forgetting by prefrontal control.** Brain imaging illustrates that not retrieving a memory when exposed to a reminder is effortful, demanding cognitive control resources. Retrieval suppression engages the prefrontal cortex, and detailed accounts of this process are emerging. Suppression engages largely right-lateralized regions, including the right anterior dorsolateral and mid-ventrolateral prefrontal cortices (aDLPFC and mVLPFC), posterior middle frontal gyrus, and bilateral insula (Anderson et al. 2004, 2016; Depue et al. 2007; Schmitz et al. 2017) (**Figure 3**). Among these, key regions related to SIF are found in the right aDLPFC (BA9/10/46) and right mVLPFC. Within-subject (**Figure 3a**) and meta-analytic conjunction analyses (**Figure 3b**) aggregating over one thousand subjects in motor and retrieval stopping studies reveal colocalized activations in right aDLPFC and VLPFC (Depue et al. 2015, Guo et al. 2018, Schmitz et al. 2017). These colocalizations suggest that inhibitory control contributes to retrieval stopping. Bolstering these parallels, retrieval and action stopping engage shared regions within the basal ganglia, including the caudate nucleus (head) and the putamen (Guo et al. 2018). Fronto-striatal interactions, regarded as critical to motor stopping (Wiecki & Frank 2013), are important to retrieval stopping, perhaps by engaging processes that gate content out of working memory (Chatham & Badre 2015).

Event-related potential (ERP) studies also suggest that retrieval suppression recruits inhibitory control. For example, stopping actions elicits a fronto-centrally distributed N2 component for Go/No-Go and Stop-Signal tasks. Suppressing retrieval also elicits a larger N2 component than does retrieval (Bergström et al. 2009; for a review, see Dutra et al. 2019). Individual differences in the N2 elicited by retrieval or action stopping are correlated, even when more than a year separates the measurements (Mecklinger et al. 2009). Larger N2 amplitude increases during retrieval suppression predict more SIF (Streb et al. 2016), suggesting that the N2 indexes processes that promote forgetting. Compelling parallels between retrieval and action stopping also occur in time-frequency analyses, building on a well-documented signature of action stopping (Castiglione et al. 2019). Comparing retrieval suppression and motor stopping, Castiglione et al. (2019) found right frontal beta power increases during suppression, an oscillatory signature shared with motor stopping in the same participants. Importantly, for motor stopping, the onset of this beta increase matched the estimated onset of action stopping derived from the stop-signal procedure and predicted stopping success; analogously, frontal beta during No-Think trials was greater when participants prevented an intrusion. Together, fMRI and EEG data suggest a shared source of top-down inhibitory control in the right prefrontal cortex that modulates processes involved in action or retrieval.

**Brain imaging evidence for hippocampal inhibition by prefrontal control.** To promote active forgetting, prefrontal sources of inhibitory control must modulate brain structures involved in representing the unwelcome memories. Moreover, modulation should be related to forgetting, tying active inhibition to a memory’s fate. Strong evidence for these features exists. Retrieval suppression consistently downregulates activity in brain areas supporting episodic recollection, including
Within-subject conjunction analysis
\((N = 24\) humans\)

A neural path linking lateral and medial PFC to MTL (rhesus monkey)

Meta-analytic conjunction analysis
\((N = 1,173\) humans across 53 studies\)

Task-dependent connectivity with hippocampus during retrieval suppression (psychophysiological interaction analysis)

Figure 3

The prefrontal cortex may serve as a supramodal control center. A comparison of motor (Stop-Signal) and memory (Think/No-Think) stopping tasks (center) allows for the possibility of a shared control resource operating across modalities. Evidence suggesting the prefrontal cortex, including the right anterior dorsolateral prefrontal cortex (aDLPFC), may be involved in such supramodal control stems, in part, from a within-subject functional magnetic resonance imaging conjunction analysis (a) of brain regions reliably engaged in both motor and memory stopping (red patches). Strikingly similar evidence was obtained in a meta-analytic conjunction analysis (b), which included 53 other studies involving these types of tasks. (c) A psychophysiological interaction (PPI) analysis using the same functional neuroimaging data collected by Schmitz et al.’s (2017) memory-control task and presented in panel a revealed regions, including within the prefrontal cortex, that exhibited memory-suppression-related coupling with a hippocampal seed. Brodmann areas (BAs) in the prefrontal cortex have been outlined in this panel, with the relevant BA numbers superimposed. Independent neuroanatomical evidence from rodents and nonhuman primates (d) has revealed pathways (indicated by directional arrows) linking the lateral (DLPFC) and medial (anterior cingulate cortex, ACC) prefrontal cortices (PFCs) to the medial temporal lobe (MTL), which could support prefrontal cortical control over memory observed elsewhere. BA numbers have been superimposed on the rhesus monkey brain for comparison. Anatomical directions (D, V, P, and A) refer to dorsal, ventral, posterior, and anterior, respectively. Panel b adapted from Guo et al. (2018). Panel d adapted from Anderson et al. (2016). Elements of this figure were adapted from material licensed to the public under the terms of the Creative Commons Attribution (CC BY) License, http://creativecommons.org/licenses/by/4.0.

the hippocampus. Specifically, retrieval suppression reduces hippocampal activity compared not only to Think trials but also to a passive baseline condition. Hippocampal downregulation has been linked to SIF (Depue et al. 2007, Levy & Anderson 2012) and correlates with right aDLPFC engagement (e.g., Depue et al. 2007). Importantly, reduced hippocampal activation is not merely
a by-product of people not retrieving anything during suppression. Indeed, hippocampal down-regulation is especially pronounced when the need for control is greatest, with intrusions showing larger reductions than nonintrusions (Gagnepain et al. 2017, Levy & Anderson 2012). This pattern is opposite to what should occur if downregulations merely reflected a lack of retrieval. This pronounced reduction may reflect the need for inhibition to purge intruding memories from awareness. Consistent with this interpretation, hippocampal downregulation during intrusions robustly predicts SIF, whereas that during nonintrusions does not (Levy & Anderson 2012). These findings link hippocampal modulation to the control of intrusive memories and to forgetting.

That reduced hippocampal activity during suppression accompanies prefrontal engagement suggests that aDLPFC actively inhibits mnemonic processing. Effective connectivity analyses reinforce this impression. For example, dynamic causal modeling analyses reveal a causal influence of right aDLPFC on hippocampal activity during suppression, consistent with top-down control (Benoit & Anderson 2012; Benoit et al. 2015; Gagnepain et al. 2014, 2017; Schmitz et al. 2017). Connectivity between the prefrontal cortex and the hippocampus is negative and often predicts forgetting (Benoit & Anderson 2012), and intrusion declines on later suppression trials (Benoit et al. 2015). Using psychophysiological interaction analysis, Schmitz et al. (2017) found that aDLPFC was one of the few regions showing differential connectivity with the hippocampus during Think and No-Think trials, exhibiting negative coupling during No-Think trials (Figure 3c). Importantly, on trials when participants reported intrusions, negative coupling between the right aDLPFC and the hippocampus is more pronounced, suggesting a purging of intruding content (Gagnepain et al. 2017, Mary et al. 2020). How right aDLPFC suppresses hippocampal activity remains unclear, given that direct projections from DLPFC to the hippocampus do not exist. Primate neuroanatomy suggests several pathways, including via the ACC (Figure 3d) and via the nucleus reuniens in the thalamus (Anderson et al. 2016).

Although the pathways that enable the prefrontal cortex to influence the hippocampus are unclear, recent research has elucidated the nature of hippocampal inhibition. Multimodal imaging has been used to relate hippocampal downregulation during retrieval suppression to inhibitory neurotransmitters. Theoretically, inhibitory neurons could suppress retrieval-related activity of principal cells within the hippocampus. If so, reduced hippocampal activity during suppression may be correlated with the neurotransmitter gamma aminobutyric acid (GABA) because hippocampal inhibitory neurons are GABAergic. Schmitz et al. (2017) quantified GABA in both the hippocampus and the prefrontal cortex with magnetic resonance spectroscopy and also conducted fMRI during suppression. Strikingly, during retrieval suppression, higher resting hippocampal GABA predicted greater downregulation and more successful forgetting. Effective connectivity analyses established that the right DLPFC modulated the hippocampus only for participants with higher hippocampal GABA. GABA in the prefrontal or visual cortex was unrelated to memory control. These findings suggest that hippocampal GABAergic interneurons enable control signals from the prefrontal cortex to suppress retrieval and disrupt memory. In contrast, no prefrontal-hippocampal coupling arose during stop-signal motor inhibition trials, and hippocampal activity was unrelated to GABA. Thus, only when participants aimed inhibitory control at memory did the prefrontal cortex drive GABAergic inhibition to actively forget. These data suggest that hippocampal inhibition achieves active forgetting, orchestrated by the right aDLPFC.

EEG studies provide a temporally precise window into the purging of intrusions from awareness during active forgetting. One approach assumes that intrusions reflect the entrance of retrieved content into working memory. If so, ERP indices of working memory storage may increase during suppression trials accompanied by intrusions, compared to those without intrusions. If so, such markers should be truncated rapidly by inhibitory control, unlike in the Think condition, in which retrieved memories are retained in awareness. Hellerstedt et al. (2016) studied
this possibility using the frontal negative slow wave (NSW) index of working memory storage (for a review, see Drew et al. 2006). They found that, whereas during Think trials the NSW emerged within 550 ms and lasted the whole trial, No-Think trials without an intrusion showed no NSW. Critically, intrusions triggered an NSW that persisted for only 1,500 ms but was then rapidly eliminated, with the duration of the intrusion-related NSW inversely related to SIF. These findings reveal the emergence and then the purging of intruding memories and illustrate how controlling intrusions rapidly is key to successful forgetting. Retrieval suppression also robustly modulates ERP markers of episodic recollection, such as the parietal EM effect: Whereas the EM effect shows marked increases during Think trials starting at 300 ms, suppression trials reveal no evidence of recollection (Bergström et al. 2009). Together, these findings indicate that suppression’s impact is visible quickly, consistent with the rapid deployment of inhibitory control (Castiglione et al. 2019).

That evidence for working memory storage and episodic recollection is abolished in EEG studies suggests that retrieving distracting thoughts is unnecessary for successful memory control. If participants needed to retrieve distracting thoughts to suppress retrieval, those thoughts should occupy working memory and generate recollective effects. Supporting this prediction, Bergström et al. (2009) found that generating substitute thoughts led to EM effects in both the Think and No-Think conditions, eliminating the modulation of this effect by suppression. Similarly, Benoit & Anderson (2012) found that, whereas asking participants to directly suppress retrieval downregulated hippocampal activity, asking them to use thought substitution did not. Thus, generating substitute thoughts drives hippocampal activity, rendering suppression and retrieval trials similar. Benoit & Anderson (2012) also dissociated the prefrontal control processes involved in direct suppression and thought substitution: Whereas direct suppression engaged right aDLPFC, thought substitution robustly engaged left VLPFC regions often found during selective retrieval. Importantly, both mechanisms induced forgetting but by engaging different neural processes, with opposite effects on hippocampal activity. These EEG and fMRI dissociations of direct suppression and thought substitution reinforce the claim that SIF does not arise from purely passive interference and is strength independent.

Brain imaging evidence for cortical and subcortical inhibition by prefrontal control. Suppressing intrusive memories also downregulates regions outside the hippocampus in a context-specific manner. The hippocampus is thought to support episodic retrieval by propagating signals outward to cortical and subcortical areas involved in processing the to-be-retrieved experience when it was first encoded; collectively, the reinstated patterns across cortical and subcortical regions represent the retrieved memory. When memories intrude during retrieval suppression, these reinstated cortical activations become targets of inhibitory control. Growing evidence supports a reinstatement principle, in which the cortical regions targeted by suppression are determined by the nature of the reinstated content. For example, when people suppress visual object memories, the prefrontal cortex downregulates the hippocampus but also fusiform cortex regions involved in object perception (Gagnepain et al. 2014, Mary et al. 2020). Strikingly, this fusiform downregulation during suppression impedes the later ability to see the suppressed objects (see Table 1). For example, suppressed objects are harder to perceive in visual noise than are baseline objects (Gagnepain et al. 2014, Kim & Yi 2013, Mary et al. 2020). This pattern suggests that inhibitory control disrupts perceptual representations, altering perception. Effective connectivity analyses confirm that the right aDLPFC modulates both the fusiform cortex and the hippocampus. Suppressing object memories thus purges the intruding visual object image but also makes suppressed content less perceptible. Converging with this view, suppressed content evokes less interference in flanker tasks (Hertel et al. 2018), suggesting it is less prone to capture visual attention (see Table 1).
Other content types produce distinct suppression patterns. For example, suppressing intrusions of upsetting scenes downregulates the hippocampus, parahippocampal place area, and amygdala in parallel (Depue et al. 2007, 2010; Gagnepain et al. 2017), reflecting inhibition of the scene’s spatial context and emotional aspects, respectively. Effective connectivity analyses confirm that the right dLPFC modulates these regions in parallel, mainly during intrusions (Gagnepain et al. 2017). This pattern suggests that purging intrusive content triggers inhibitory control to target reactivated cortical regions, consistent with the reinstatement principle. Cortical suppression also has been observed using magnetoencephalography. Waldhauser et al. (2018) found that suppressing visual objects reduced gamma power, with reductions in a region encompassing occipital, parietal, and middle temporal cortices, suggesting the suppression of perceptual content. Using EEG, Waldhauser et al. (2015) also found that successful retrieval suppression induced widespread decreases in theta power across sensory regions and the medial temporal lobes, taken to reflect suppression of a broad hippocampo-cortical memory pattern. Together, these findings suggest that suppression modulates hippocampal activity together with cortical regions.

Cortical modulation during retrieval suppression leaves aftereffects on neural indices of memory. One such index is repetition suppression (Grill-Spector et al. 2006). Repetition suppression refers to decreased activity in brain regions involved in processing a stimulus arising from having already encountered it. Reduced neural activity reflects the ease people have in reprocessing a familiar stimulus and is interpreted as a neural index of perceptual memory. Building on this work, Gagnepain et al. (2014) measured fMRI activity as people suppressed retrieval of visual objects. Later, participants performed a perceptual identification test for the objects while being scanned, allowing the researchers to measure repetition suppression in fusiform cortex. Critically, participants showed reduced repetition suppression for No-Think items compared to Baseline items, which exhibited robust repetition suppression. Negative coupling between the aDLPFC and the fusiform cortex during retrieval suppression predicted the later reversal of repetition suppression for No-Think items, with greater negative coupling predicting larger reversals. Retrieval suppression thus induces neural aftereffects on perceptual memory, consistent with active forgetting by inhibitory control.

**Retrieval suppression yields conflict reduction benefits and affect suppression.** As with selective retrieval, stopping retrieval yields conflict reduction benefits that are linked to forgetting. Behaviorally, benefits are evident in steeply declining intrusions over repeated suppressions, which correlates with SIF (Hellerstedt et al. 2016, Levy & Anderson 2012). Thus, active forgetting reduces conflict triggered by involuntary retrieval. Using ERPs, Hellerstedt et al. (2016) found that declines in the frontal NSW over blocks were associated with intrusion reductions, suggesting that controlling conflict reduced control demands. Conflict reduction benefits also have been found with fMRI by Benoit and colleagues (2015). Replicating prior work, Benoit and colleagues found that suppressing scenes engaged the right aDLPFC to modulate hippocampal activity. Importantly, they found that participants exhibiting steeper declines in intrusions over blocks showed larger reductions in negative coupling between the aDLPFC and the hippocampus; thus, as intrusions were controlled, inhibitory control demands declined. Indeed, participants with strong negative coupling in the first half of the TNT task showed far fewer intrusions in the second half, compared to participants with less negative coupling. These findings link active forgetting of intrusive memories to reductions in control demands when confronted with the same reminders.

Active forgetting reduces access to unpleasant memories, yielding emotional benefits (Anderson & Hanslmayr 2014, Engen & Anderson 2018, Fawcett & Hubert 2020). Retrieval
Affect reduction benefit: A beneficial reduction in unwanted affective responses to a stimulus resulting from actively forgetting overly accessible and disruptive memories.

Active Forgetting by Process Inhibition

Research on memory inhibition suggests that prefrontal control causes forgetting by inhibiting representations underlying an experience. However, a major conceptual development is the discovery that active forgetting can be accomplished by suppressing memory processes more broadly. Process inhibition arises when inhibitory control suppresses episodic encoding, consolidation, or retrieval, affecting every memory relying on the process. Process inhibition happens for limited temporal windows, but its effects may be long-lasting; by suppressing encoding or consolidation, for example, new events within the suppression window will not be durably stored. Although process inhibition may be engaged to control a particular memory, it accomplishes forgetting systematically. This hypothesized process inhibition resembles global stopping of actions (e.g., Aron & Verbruggen 2008, De Jong et al. 1995). Global stopping suppresses the expression of all actions at a given time (even if the intention is to stop one action), in contrast to selective stopping, in which the targeted action is inhibited while other actions move forward. Process inhibition also resembles theories of forgetting that emphasize interrupted consolidation (e.g., Wixted 2004) but differs in that control mechanisms that disrupt mnemonic function drive interruptions rather than interference from new encoding.

Several findings indicate that the prefrontal cortex can globally suppress episodic encoding and stabilization. Process inhibition arises from systemic (untargeted) suppression of hippocampal activity, a mechanism with broad consequences (Hulbert et al. 2016, 2018). This work suggests more control over hippocampal processes than imagined by theories of hippocampal function, which often construe this structure as operating automatically (e.g., Moscovitch et al. 2016). Strikingly, process inhibition induces anterograde and retrograde amnesia in healthy individuals that mirrors organic amnesia for short periods. We discuss three examples. We then argue that this
A revealing discovery about retrieval suppression is that its effects extend beyond the memory people seek to inhibit. People who suppress retrieval of a memory also forget events occurring before or after the attempted suppression (Hulbert et al. 2016). Remarkably, these so-called innocent bystander memories need not be related to the suppressed content. For example, after suppressing a simple verbal pair, participants are more likely to forget a visual object presented before the suppression trial, revealing retrograde amnesia; objects appearing after the suppression trial suffer a similar fate, illustrating anterograde amnesia. These effects appear additive, with objects surrounded by suppression on both sides suffering larger deficits. Together, we refer to forgetting of adjacent events, surrounding retrieval suppression, as an amnesic shadow (Figure 4).

The amnesic shadow illustrates that inhibitory control may not target the to-be-suppressed trace selectively but rather affects mnemonic processing broadly.

The amnesic shadow is a unique cognitive prediction derived from knowing that retrieval suppression downregulates hippocampal processing. If suppression reduces hippocampal activity via GABAergic inhibition (Schmitz et al. 2017), it may create a virtual lesion, undermining hippocampal processes (Figure 4a,b). Strikingly, this model predicts that suppression should mimic organic amnesia, at least for brief windows. To test this prediction, Hulbert et al. (2016) inserted pictures between Think and No-Think trials and tested memory for the pictures after the TNT task ended (Figure 4d). In some experiments, these innocent bystander pictures included an object in a background, and participants simply imagined how the object got there. If thinking about the picture encodes a hippocampal trace, and retrieval suppression follows afterward, will later memory for the bystander be harmed even though the suppressed content is unrelated to it? If retrieval suppression instead happens before viewing the bystander, will the adverse hippocampal state created by downregulation disrupt its encoding? Rather than inserting bystanders directly between Think and No-Think trials, Hulbert et al. bounded the bystander pictures with 5–7 s of odd/even judgments on digit stimuli. Inserting this irrelevant task ensured that the same task occurred just before and after the pictures, holding task-set switching demands constant. The key question concerned whether either the prior or the following suppression trials would adversely affect bystander recall (Figure 4e).

The researchers found that pictures surrounded by No-Think trials suffered sizeable recall deficits compared to those surrounded by Think trials: Bystander pictures suffered as high as a 44% proportional retention loss (Figure 4f). Hulbert et al. showed that this amnesic shadow (a) arose specifically from retrieval suppression and not task difficulty, (b) reflected disruption from No-Think trials, rather than enhancement from Think trials, (c) was composed of both anterograde and retrograde effects, (d) lasted at least 24 hours, and (e) affected recognition memory but only on source-memory tests, consistent with hippocampal involvement. Amnesic shadow effects also arise for previously encoded memories when reminders to those memories appear as bystanders during retrieval suppression (Zhu & Wang 2020).

Two findings link the amnesic shadow to hippocampal modulation. First, instructing people to directly suppress retrieval induces the effect, whereas thought substitution does not, paralleling the differential suppression of hippocampal activity in these tasks (Benoit & Anderson 2012). Second, the size of the amnesic shadow correlates with hippocampal downregulation during
The hippocampal modulation (HM) paradigm, predictions, and results. Whereas organic hippocampal amnesia (e.g., Patient H.M., panel a, whose hippocampal lesions are presented at the top of panel b) is caused by physical damage, direct retrieval suppression induces functional deactivations (in blue), prominently within the hippocampus (b, bottom) that may induce functional amnesia. The HM hypothesis holds that HMs of this sort (c) reflect global disruptions to hippocampal memory processes, with consequences extending beyond the targeted unwanted memory (cued by a reminder in red during a No-Think trial, for instance). By presenting bystander images unrelated to the materials from the surrounding Think/No-Think tasks (e.g., a soccer ball on a coffee table or a peacock in a parking lot) and buffered by odd/even judgments to match the immediately surrounding context, the HM paradigm (d) assesses whether cued-recall memory for the central objects is impaired on a final test (e) as a function of the hippocampal state in which the bystander was exposed. As predicted, memory for the bystanders exposed to two suppression epochs (e.g., the peacock) exhibited both anterograde and retrograde amnesia relative to those bystanders originally presented between two retrieval-based Think trials (in green), consistent with a disruption to early stabilization processes. The magnitude of this amnesic shadow effect was predicted by modulations in the bilateral hippocampus (not shown) and the degree of frontal engagement, here presented in arbitrary units (a.u.) (f). Portrait of Henry Molaison in panel a taken by Suzanne Corkin. Copyright © Suzanne Corkin Estate, used by permission of The Wylie Agency LLC. Panel b, top, adapted from Corkin et al. (1997); copyright 1997 Society for Neuroscience. Panel b, bottom, adapted from Schmitz et al. (2017). Panels d–f adapted from Hulbert et al. (2016). Elements of this figure were adapted from material licensed to the public under the terms of the Creative Commons Attribution (CC BY) License, http://creativecommons.org/licenses/by/4.0.

retrieval suppression. The amnesic shadow thus confirms that retrieval suppression induces a reversible hippocampal lesion, disrupting encoding and consolidation processes, akin to organic amnesia. More broadly, this finding supports the possibility that inhibitory control can induce active forgetting by broadly suppressing mnemonic processing, in accordance with our cognitive goals. Hulbert and colleagues (2016, 2018) suggest that chronic recruitment of retrieval suppression after trauma may contribute to global memory deficits often found in trauma victims, even for events unrelated to the trauma (Brewin 2011).
Disrupting Episodic Encoding by Working Memory Updating

Retrieval suppression is not the only task that suppresses hippocampal activity. Complex working memory tasks involving updating, such as the N-back task, reduce hippocampal activity with high memory loads. In such tasks, participants view items, such as digits, and press a button indicating the number from N trials back. The easiest version is the 0-back condition, in which participants press the button for the current digit; in harder versions (e.g., the 2-back condition), participants must recall the digit from two trials ago, press its key, and then forget that digit, while revising working memory to include the current digit. Interestingly, the 2-back task engages DLPFC and reduces hippocampal activity, compared to the 0-back task (for a discussion, see Mullally & O’Mara 2013), as with suppression. These findings suggest that suppressing outdated digits may be achieved by inhibiting hippocampal encoding.

Difficult working memory updating tasks that suppress hippocampal activity may cause anterograde amnesia for subsequent episodic events. Mullally & O’Mara (2013) confirmed this prediction in two studies, each contrasting the impact of a 2-back and a 0-back task on later encoding and retrieval of episodic items. In Experiment 1, participants performed short working memory blocks (e.g., 1–2 min) before encoding and retrieving 15 unrelated words; in Experiment 2, participants encoded 8 face-name pairs instead of words. Interestingly, both experiments demonstrated robust memory deficits for items encoded after the 2-back, compared to 0-back, task. On a final test after the working memory/encoding blocks, Mullally & O’Mara (2013) found that the 2-back task disrupted recognition of the words but not priming, an implicit memory effect that does not depend on the hippocampus. The anterograde deficit was striking for the face-name pairs, consistently near or exceeding 30%. Although the authors did not test retrograde amnesia, their anterograde encoding deficits provide converging evidence for process-level inhibition, induced by systemic inhibition of hippocampal activity.

Disrupting Episodic Encoding by Directed Forgetting Instructions

Hippocampal downregulation during working memory updating suggests that hippocampal encoding can be suppressed whenever durably forgetting information in working memory is useful. Deficits on later memory tasks unrelated to the N-back confirm that memory processes were globally disrupted, as happens with retrieval suppression. If correct, this interpretation suggests that people can strategically inhibit hippocampal encoding to prevent experiences from being stored. A growing body of work confirms this prediction. For example, intentional encoding suppression may contribute to item-method directed forgetting. In this procedure, participants view items, such as pictures or words, and, following each one, are asked to remember the item or forget it. At the end, participants are tested on all items, typically by a recall or recognition test. People usually recall fewer Forget than Remember items. Thus, instructing people to forget leads to worse memory (Figure 5a).

Cognitive psychologists have debated the cause of this phenomenon. On the one hand, people may recall more Remember items because they encode them more elaborately, known as selective rehearsal (Basden et al. 1993, Bjork 1989). By this view, an item is retained in working memory until the instruction specifies what to do; a Remember instruction would lead participants to encode the item elaborately; a Forget instruction would allow them to stop rehearsing it. On the other hand, people may recall more Remember items because they inhibit Forget items (Zacks et al. 1996), a process we refer to as encoding suppression (Anderson & Hanslmayr 2014). Distinguishing these accounts is difficult, and some researchers prefer the simplicity of selective rehearsal. Increasing evidence indicates, however, that active forgetting contributes. For example, contrary to selective rehearsal, the Forget condition requires more effort than the Remember condition, as reflected...
Item-method directed forgetting paradigm and results. A typical item-method directed forgetting paradigm (a) instructs participants to either remember (R, in green) or forget (F, in red) the preceding stimulus. Final memory tests generally reveal an impairment for the to-be-forgotten (TBF) compared to to-be-remembered (TBR) material, as depicted by representative recognition memory results to the right. Neuroimaging results (b) reveal that attempts to forget (relative to remember) recently presented material reflect an effortful process that is associated with increased prefrontal cortex activity (hot colors) and reduced activity in the medial temporal lobe (cool colors); further analyses by Rizio & Dennis (2013) revealed that the right superior frontal cortex and the left medial temporal lobe exhibited negative connectivity during successful forgetting. Intracranial recordings (lower portion of panel c) from electrodes embedded in the right dorsolateral prefrontal cortex (DLPFC) (green dots), left DLPFC (not shown), and hippocampus (red dots) of epileptic patients yielded time-frequency plots (top portion of panel c) revealing that voluntary forgetting enhances oscillatory power within the outlined low-theta (3–5 Hz) and broader 6–18 Hz ranges for the DLPFC (left) and anterior hippocampus (right), respectively. Power at 8 Hz within the hippocampus (indicated by the white dagger) was significantly greater for successful forgetting. Granger causality analyses (d) highlighted an asymmetric directional transfer of information from the DLPFC to the hippocampus (red ribbon) unique to TBF trials (top portion of panel d) within the beta frequency band (15–18 Hz; gray shaded), consistent with the involvement of beta-frequency oscillations in both action stopping and retrieval suppression (Castiglione et al. 2019). Panel b adapted with permission from Rizio & Dennis (2013); copyright 2013 MIT Press. Lower portion of panel c adapted with permission from Oehrn et al. (2018); copyright 2018 Elsevier.

by slower reaction times on a secondary task while executing the Forget instruction (Fawcett & Taylor 2008, Fawcett et al. 2016). Moreover, stopping a motor response after the task cue is more successful in the Forget condition (Fawcett & Taylor 2010), suggesting that Forget cues engage inhibitory mechanisms involved in stopping motor actions.
Multiple neuroscience methods confirm these behavioral findings, indicating that item-method directed forgetting recruits a prefrontal-hippocampal inhibitory mechanism similar to that engaged by retrieval suppression. fMRI studies find greater right prefrontal activation during Forget than Remember trials (e.g., Nowicka et al. 2011, Rizio & Dennis 2013, Wierzba et al. 2018, Wylie et al. 2008, Yang et al. 2016). Forget instructions also often reduce hippocampal/parahippocampal activity, relative to Remember instructions, especially when forgetting succeeds (for intracranial evidence supporting this, see Ludowig et al. 2010; Rauchs et al. 2011; Rizio & Dennis 2013). Connectivity analyses suggest that reduced hippocampal activity during Forget trials arises from interactions with the right prefrontal cortex (Rizio & Dennis 2013, Wierzba et al. 2018) (Figure 5b). Thus, a prefrontal mechanism that inhibits hippocampal encoding likely causes forgetting. These findings echo hippocampal downregulation during retrieval suppression and working memory updating tasks.

Recent innovative work has recorded brain electrical activity from the DLPFC and the hippocampus during item-method directed forgetting, providing unprecedented spatial and temporal resolution in tracking encoding suppression. Oehrn et al. (2018) studied 25 patients with intracranial electrodes in the DLPFC, the hippocampus, or both (n = 6 in the last case). Recording neural activity caused by the Remember and Forget instructions revealed distinct processes engaged during forgetting that altered information flow from the DLPFC to the hippocampus. Attempting to forget triggered greater EEG activity in the low-theta range (3–5 Hz) in the DLPFC, emerging 568–1,058 ms after the Forget cue, together with oscillatory power increases in a broader theta/alpha/beta range (6–18 Hz) in the anterior hippocampus (Figure 5c). In the hippocampus, the peak frequency more associated with the Forget condition was 8 Hz, and power in this frequency was greater for successfully forgotten items. Critically, effective connectivity analyses revealed a robust interaction between the DLPFC and the hippocampus in the beta range (15–18 Hz) (Figure 5d), revealingly echoing prefrontal beta oscillations in retrieval suppression and action stopping (Castiglione et al. 2019). Only during the Forget condition were beta interactions dominant in the top-down direction. The control signal in the DLPFC began 100–130 ms prior to affecting the hippocampus. These data provide compelling spatially and temporally specific support for a top-down signal of encoding suppression.

The Scope of Mnemonic Process Inhibition as a Mechanism of Forgetting

The preceding examples illustrate how active forgetting processes can induce untargeted, global hippocampal suppression. Hippocampal suppression disrupts encoding and stabilization processes, yielding generalized forgetting in a broader window. Process inhibition has, to our knowledge, never been proposed as a mechanism of forgetting, prior to the aforementioned work. Two points make this observation striking. First, the forgetting effect is big; both Hulbert et al. (2016) and Mullally & O’Mara (2013) found that retention of adjacent events can be cut nearly in half (proportional reductions between 40% and 50%) with hippocampal suppression. Thus, hippocampal suppression acts like a reversible lesion mimicking organic amnesia. Second, process inhibition may be pervasive (discussed next). Given these characteristics, process inhibition may be a fundamental and unrecognized mechanism of episodic forgetting.

Memory process inhibition may be pervasive because many goals could benefit from temporarily dampening mnemonic function. If so, hippocampal suppression may implement that orientation away from memory. The Mullally & O’Mara (2013) findings illustrate how readily such a task set is adopted; after 1–2 min of the N-back task, substantial anterograde memory deficits emerged. In general, tasks that sustain attention on perception, particularly those with demands to forget interfering content, may behave like intentional suppression. Consider visual search for a target object amid distractors; stored memories for the same items and their locations on previous
trials may interfere with visual search on the current trial, much like gazing toward yesterday’s parking spot when searching for today’s. Evidence for potential mnemonic suppression in such cases exists. For example, tasks requiring perceptually focused attention suppress medial temporal lobe activity as part of suppressing the default-mode network (Buckner & DiNicola 2019, Raichle et al. 2001, Shulman et al. 1997). Intracranial recordings in humans confirm default-mode network suppression and reveal its basis in reduced high-frequency gamma oscillations (Fox et al. 2018). Suppressed gamma power in default-mode areas such as the posterior-medial cortex increases with task difficulty and predicts how well people perform the externally focused tasks (Foster et al. 2012, Ossandón et al. 2011). Posterior-medial areas maximally involved in autobiographical memory suffered the greatest suppression (Foster et al. 2012). Mnemonic process inhibition could thus minimize distraction from memory during externally focused tasks. Might a consistent need for intense focus on our perceptual world trigger greater forgetting due to chronic default-mode suppression? Might people who wish to forget attempt to “avoid memory” by adopting a powerful outward bias of attention? Opportunities for mnemonic process inhibition may be plentiful.

The relationship between process inhibition and default-mode suppression remains speculative. It is unclear, for example, whether default-mode suppression regularly induces an amnesic shadow. Hippocampal suppression during memory control also may not be equivalent to default-mode suppression. For example, whereas both retrieval suppression and motor stopping are difficult tasks that reduce hippocampal activity, only retrieval suppression induces hippocampal down-regulation that (a) is driven by the DLPFC and (b) depends on hippocampal GABAergic inhibition (Schmitz et al. 2017). Thus, not all down-regulations are equivalent, and suppressing memory intentionally may be distinct. Nevertheless, as Mullally & O’Mara (2013) illustrate, difficult tasks that do not emphasize forgetting may benefit by being unshackled from memory’s distractions. Given these observations, future work should distinguish two hypotheses. The default-mode suppression hypothesis of mnemonic process inhibition attributes the suppression’s behavioral and neural effects to obligatory competition between the default network and networks supporting externally focused attention; the supramodal inhibitory control hypothesis, however, attributes such effects to prefrontal control processes targeted in a flexible, goal-directed fashion. Also, default-mode suppression itself, rather than explaining process inhibition effects, may be explained by them: Default-mode suppression may not reflect automatic competition between networks but the consistent desirability of strategically recruiting inhibitory control to suppress memory when attention is outwardly directed. Thus, memory control may explain default-network suppression.

ACTIVE FORGETTING BY CONTEXT SUPPRESSION

To actively forget a memory, one need not disrupt it. Another approach involves removing reminder cues, rendering retrieval ineffective. Because episodic retrieval relies on context cues, changing context ought to cause forgetting. Contextual cues are environmental features, such as one’s location, time of day, sensory environment (e.g., noise, odors), and general background activity, some of which become incidentally associated with events during encoding, making them potential cues. One’s mental context might also become associated with a memory; mental context denotes one’s frame of mind, including concepts and topics one has recently thought about, and one’s mood, filters through which we interpret events (see, e.g., Manning et al. 2014). Perhaps mental context might be controlled via mechanisms that purge its contents (Figure 1c). If so, then after mental context inhibition, recalling otherwise intact memories should fail due to a mismatch between one’s current and former context. Evidence supports this context change process and suggests that the DLPFC implements this capacity, possibly via inhibitory control.

Context control can explain an effect known as list-method directed forgetting. In this procedure people study an initial list (e.g., 10–20 pictures or words) and then are asked to remember or
to forget it (Bäuml et al. 2010, Bjork 1989). In the Forget condition, participants are led to believe the first list was for practice or was mistakenly presented and should be forgotten. Both groups then study a second list, after which both lists are tested. Interestingly, first-list recall performance suffers after a Forget instruction compared to a Remember instruction, illustrating the directed forgetting effect (for reviews, see Anderson & Hanslmayr 2014, Bäuml et al. 2010, Sahakyan et al. 2013). In addition to the aforementioned cost of directed forgetting, oftentimes second-list recall benefits from instructions to forget the first list (see Table 1). Although this effect occurs on free- and cued-recall tests, people have little trouble recognizing first-list items (on old/new tests) and show normal implicit memory effects. This pattern suggests that forgetting mechanisms do not affect the items but rather the mental context needed to retrieve them (Basden et al. 1993). Essentially, people seem to “change the channel” to focus on new activities (the second list), inducing forgetting. Supporting this interpretation, on the final test, getting people to restore the original mental context, either by reimagining their circumstances before learning the first list (Sahakyan & Kelley 2002) or by supplying first-list items as hints (Bäuml & Samenieh 2012), reduces forgetting. The capacity to undo forgetting suggests that the Forget instruction made mental context temporarily inaccessible. This outcome may be achieved either by inhibiting mental context (e.g., Bäuml et al. 2008) or by replacing it with a new context. We refer to these possibilities as context suppression and context substitution, mirroring the distinction between direct suppression and thought substitution in SIF.

fMRI studies reveal the contribution of DLPFC to list-method directed forgetting and vividly illustrate context control. For example, Hanslmayr et al. (2012) scanned people with fMRI during list-method directed forgetting. Compared to the Remember condition, instructing people to forget both engaged the left DLPFC (BA9) and reduced DLPFC-hippocampal coupling during List 2 encoding, consistent with an inhibitory influence (negative coupling) on memory. DLPFC activation also predicted reduced phase synchrony in the alpha/beta frequency range (11–18 Hz) during List 2 encoding after a Forget, relative to a Remember, instruction, as measured by simultaneous EEG (see also Bäuml et al. 2008). Reduced phase synchrony may reflect disrupted access to List 1, based on work showing that neural synchrony between remote cell assemblies is important to forming memories (Fell & Axmacher 2011). In a second experiment, Hanslmayr et al. (2012) stimulated the left DLPFC region from Experiment 1 (BA9) with transcranial magnetic stimulation during List 2 encoding. Strikingly, stimulation greatly increased directed forgetting and triggered large alpha-beta phase synchrony reductions during the Forget condition. These findings suggest the DLPFC as a source of a forgetting signal (possibly via interactions with the hippocampus) and point to alpha-beta desynchronization as a marker of its effects.

Imaging also illustrates how Forget instructions inspire a contextual purge to induce forgetting. To create a distinct mental context, Manning et al. (2016) inserted three outdoor scenes for brief viewing between each studied word on the first list. Manning and colleagues added these scenes to inject identifiable content into participants’ mental context by encouraging them to ponder nature while studying the words. After a standard Remember or Forget instruction, participants studied a second list (without scene buffers). If Forget instructions truly inspire a purge of the first-list mental context, neural activity related to nature scenes (a key element of that list’s context) should decline after the instruction, and this decline should exceed that in the Remember condition. To test this, Manning et al. trained a pattern classifier to distinguish brain activations elicited by nature scenes, phase-scrambled scenes of everyday objects, and rest that were obtained during an independent localizer scan. Applying this classifier, they found that Forget instructions rapidly reduced scene-related patterns of activity more than did Remember instructions. Importantly, the scene activity drop predicted forgetting of Forget items, suggesting that loss of mental context drove directed forgetting. Thus, forgetting may arise in part by altered mental context, perhaps induced by top-down signals from the prefrontal cortex (Hanslmayr et al. 2012).

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**Directed forgetting effect:** impaired memory for events that one intends to forget, often measured with the item and list-method directed forgetting procedures

**Context substitution:** forgetting caused by facilitating an alternative context by retrieval, generation, or perception, displacing context representations associated with a memory

**Imaging also illustrates how Forget instructions inspire a contextual purge to induce forgetting.**
More work should address the contributions of context suppression and context substitution to directed forgetting. Asking people to imagine a novel context right after studying a list (e.g., imagining being invisible) induces forgetting resembling directed forgetting (for a review, see Sahakyan et al. 2013), suggesting that context substitution may suffice to explain directed forgetting effects. However, directed forgetting and context substitution effects are not identical, exhibiting both parallels and dissociations (Abel & Bäuml 2017, Pastötter et al. 2008, Sahakyan & Kelley 2002, Sahakyan et al. 2013). Both context substitution and suppression may contribute to directed forgetting, just as both thought substitution and inhibition contribute to SIF. Regardless of the mechanism, the striking feature of this phenomenon is that forgetting is reversible, with forgotten material returning when context is restored. This context forgetting may help organisms to shift between activities efficiently. For example, whole collections of thoughts and facts relevant to prior goals and schemas, but not to the current one, may be rendered noninterfering by context inhibition, facilitating new activities. If the older task becomes current, restoring the context may reactivate relevant thoughts and information. Context inhibition may thus rapidly tune our mnemonic state to changing goals.

**CONCLUDING REMARKS**

A major goal of memory research is to understand why we forget. Here, we reviewed evidence that forgetting can arise actively from control mechanisms mediated by the prefrontal cortex. We argued that active forgetting adapts memory to be aligned with our cognitive and emotional goals. The prefrontal cortex solves memory adaptation problems by promoting the forgetting of memories whose persisting accessibility is disruptive, a process that happens in several ways. Prefrontal control processes act, in part, by inhibiting specific memories, reducing future demands on control. A major driver is the need to control retrieval, during either selective retrieval or retrieval stopping. Control mechanisms also disrupt retention by suppressing memory processes more broadly, attenuating encoding, stabilization, and retrieval by systemically suppressing hippocampal function. Finally, prefrontal control mechanisms can induce temporary forgetting by forcing changes in mental context and removing cues needed to access memories. The causal role of prefrontal inhibitory control in some forms of active forgetting has now been shown in humans and nonhuman animals. The generality of these processes over species suggests that prefrontal control is an evolutionarily old solution to memory adaptation, reflecting the fundamental nature of the memory control problems.

Active forgetting processes discussed in this article may be the tip of the iceberg when it comes to understanding how organisms promote memory loss. Over the past decade, innovations in modern neuroscience have led many neurobiologists to embrace an active view of forgetting (Davis & Zhong 2017, Richards & Frankland 2017), with processes previously thought of as passive, such as memory decay, revealed to be biologically regulated and active (Hardt et al. 2013, Migues et al. 2016). The work reviewed here provides a critical bridge between the high-level cognitive and behavioral forces that promote remembering and forgetting, and basic mechanisms of memory loss operating at a cellular level. The coming decade may witness the emergence of forgetting as a field in its own right and an integrated multilevel understanding of how organisms shape the fate of experiences in memory.

**SUMMARY POINTS**

1. Advanced neuroscience methods are revealing the operation of mechanisms that foster the forgetting of memories whose ongoing accessibility disrupts our cognitive or emotional goals, adapting the state of memory to better align it with behavior.
2. The lateral prefrontal cortex (especially on the right) plays an instrumental role in actively reducing accessibility of disruptive memories via inhibitory control mechanisms shared with the control of action but targeted at memory.

3. Inhibitory control can induce active forgetting by acting on memories, during either selective retrieval or retrieval stopping, yielding measurable benefits including reduced conflict, reduced expenditure of control resources, and reduced affective responses to suppressed stimuli.

4. Multivariate functional magnetic resonance imaging methods have been used to dynamically track neural indices sensitive to the activation of individual memories as they are being suppressed by inhibitory control, revealing the operation of active forgetting processes and linking changes in cortical patterns to later forgetting.

5. An animal model of active forgetting via selective retrieval has been developed and used to establish the causal necessity of the prefrontal cortex, opening the door to a multilevel model of active forgetting spanning cognition to synapses.

6. Suppressing the retrieval of an intrusive memory is achieved by parallel inhibition of both the hippocampus and content-specific cortical and subcortical regions, with the efficacy of hippocampal downregulation by the prefrontal cortex dictated in part by inhibitory gamma aminobutyric acid (GABA) neurotransmitters local to the hippocampus.

7. Inhibitory control acting on the hippocampus not only can impair particular memories but also can temporarily interrupt the functioning of fundamental memory processes more generally, including encoding, stabilization, and retrieval of memories, inducing virtual hippocampal lesions that mimic organic amnesia in healthy individuals; such global disruption may represent a pervasive cause of episodic forgetting that previously has gone unrecognized.

8. Control processes can also act upon representations of mental context rather than memories themselves and, in so doing, create a reversible change in accessibility that is useful for rapidly tuning the state of memory to task demands.

**FUTURE ISSUES**

1. The pathways through which the right lateral prefrontal cortex modulates the hippocampus and cortex remain to be understood and are critical to a comprehensive understanding of active forgetting.

2. Deficits in the ability to solve memory adaptation problems by inhibitory control may contribute to a range of psychiatric disorders characterized by persisting intrusive memories and thoughts. The brain mechanisms of active forgetting provide a powerful framework through which to understand intrusive symptomatology and may help to identify origins of key psychiatric symptoms as well as targets for remediation.

3. How active forgetting processes contribute to altered affective responses to suppressed stimuli and emotion regulation processes more broadly needs to be more thoroughly investigated to understand the role of memory adaptation in mental health.
4. The mechanisms by which GABAergic inhibition in the hippocampus disrupts individual memories and mnemonic processes need to be more fully understood, as this GABAergic mechanism may be the final step in the path of active forgetting in the brain.

5. Studying the relationship between the hippocampal downregulation arising during retrieval suppression and default-mode network suppression may yield important insights about the nature of both phenomena; it is possible that default-mode network suppression may not be the result of automatic network competition but rather strategic application of inhibitory control to manage unwanted influences of memory during task performance.

6. The newly developed rodent model of active forgetting provides an exciting opportunity to develop a highly detailed circuit- and molecular-level understanding of how high-level goals ultimately shape memory to align it with the needs of behavior.

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