7 The functional neuroimaging of forgetting

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Forgetting is a common, often troubling, experience. Failing to remember where we left our keys, the name of a colleague, the meaning of a word we once knew, or an errand that needed to be done on the way home, can be embarrassing and, at times, quite costly. Not all instances of forgetting are unpleasant, however. More often than we realize our goal is actually to forget, rather than remember. For example, forgetting is adaptive when we move and must unlearn information that is no longer relevant, such as our old phone number and address. Similarly, workers who must repeat similar activities throughout a workday, such as a waiter who takes many similar orders in a shift, would likely be better off if they could forget the orders from earlier in the day. Thus, while many of us desire to have a perfect memory, in many ways we would be disadvantaged if we were to remember every experience.

Why do we forget? This question was once one of the most prominent topics of research on memory, with much of the original work inspired by Ebbinghaus (1885/1913), who carefully documented the rate at which he forgot nonsense syllables. Early accounts pitted the idea that memories passively decay over time against the notion that subsequent learning interferes with our prior experiences, either by disrupting the consolidation of those traces into durable memories or by interfering with our ability to retrieve them. Over time, each of these theories has experienced difficulty explaining some aspects of forgetting and, thus, none has been able to provide a unified account of forgetting. Regrettably, this has meant that the field has never settled on a cohesive theory of forgetting, with modern overviews tending to focus on describing a set of experimental results without a clear theoretical account of why forgetting occurs. Given the ubiquity of forgetting in everyday life, however, a comprehensive understanding of its causes is of prime importance to theories of memory. Perhaps the primary failing of these earlier theories was the implicit assumption that forgetting is produced by a single mechanism. Instead, forgetting may arise from a disruption to any of the events that promote successful memory. Here we propose five distinct mechanisms that produce forgetting, none of which alone is sufficient to account for all types of forgetting. In the following sections, we describe the behavioral and neuroimaging evidence supporting the existence of each of

these mechanisms in order to better understand why we sometimes fail to remember past experiences.

Forgetting due to failed encoding

Perhaps the most obvious, though somewhat underappreciated, reason why we forget is because we often poorly encode events as they happen. This can be due to absent-mindedness, distraction, or any other factor that limits attention as we engage with the world. For example, forgetting where you left your keys may simply reflect a failure to pay attention to what you were doing when you set them down. Similarly, if you are distracted when introduced to a new co-worker you are unlikely to later remember that person's name. In these instances, forgetting does not arise because of the loss of information over time; rather, forgetting arises because the initial episode was never transformed into a durable memory representation. Many theories of forgetting have ignored this cause, since in these cases nothing is successfully stored in memory and, thus, nothing is ever truly lost from memory. It seems likely, though, that many of the memories that we describe as "forgotten" are attributable to failures to encode. Therefore, it is worth considering the factors that influence encoding lapses.

To understand why encoding sometimes fails, it is helpful to understand how successful encoding occurs. Functional neuroimaging studies have typically examined this issue by using the subsequent memory paradigm, where brain activity is monitored during an experience and then related to behavioral evidence about whether or not the experience is later remembered (e.g., Brewer, Zhao, Desmond, Glover, & Gabrieli, 1998; Paller, Kutas, & Mayes, 1987; Wagner et al., 1998). In this paradigm, activity during encoding trials that are subsequently remembered is compared to activity on trials that are subsequently forgotten, yielding a pattern of activity that is specifically associated with successful memory encoding.

There are now over 100 functional magnetic resonance imaging (fMRI) studies using this subsequent memory paradigm, and they have consistently revealed a network of regions that positively relate to subsequent remembering, including ventrolateral prefrontal cortex (PFC), medial temporal lobe (MTL), and dorsal parietal cortex (see Figure 7.1; for reviews see Blumenfeld & Ranganath, 2006; Davachi, 2006; Paller & Wagner, 2002; Uncapher & Wagner, 2009). One interpretation of these findings is that fronto-parietal control mechanisms are engaged during encoding to modulate processing in posterior cortical regions in a goal-directed fashion. This modulation is thought to regulate the inputs that are received by the MTL, which ultimately binds these distributed patterns of activity into durable episodic memory traces. Increased activity for subsequently remembered items presumably reflects the increased engagement of this network. According to this framework, memories may be doomed to forgetting when we fail to sufficiently engage these neural mechanisms during encoding.

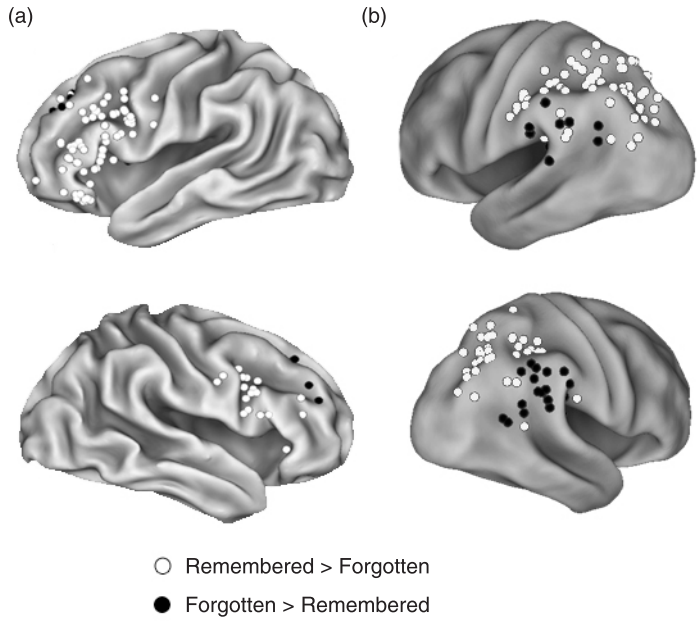

Figure 7.1 Meta-analyses of subsequent memory effects. (a) The local maxima within PFC from 33 fMRI studies of LTM formation (from Blumenfeld & Ranganath, 2006) reveal that positive subsequent memory effects (i.e., remembered > forgotten) tend to fall within VLPFC. (b) The local maxima within parietal cortex from 93 fMRI studies of LTM formation (from Uncapher & Wagner, 2009). Positive subsequent memory effects tended to fall within intraparietal sulcus and superior parietal cortex, while negative effects appeared exclusively in inferior parietal regions.

There is considerable behavioral evidence that directing attention to specific aspects of a stimulus has a profound impact on subsequent memory, both in the likelihood that it will be remembered (Craik & Lockhart, 1972; Craik & Tulving, 1975) and the type of representation that is stored (Mitchell, Macrae, & Banaji, 2004; Morris, Bransford, & Franks, 1977; Otten, Henson, & Rugg, 2002; Otten & Rugg, 2001a; Tulving & Thomson, 1973). These findings suggest that the allocation of attention during study ultimately influences what is stored in memory. More direct evidence about the importance of attention during encoding comes from studies where subjects are given an attentionally demanding secondary task to perform during encoding. The typical finding from these studies is that doing this severely impairs later memory for those items (e.g., Craik, Govoni, Naveh-Benjamin, & Anderson, 1996), and also leads to reduced activation in fronto-parietal regions (Fletcher, Frith, Grasby, Shallice, Frackowiak, & Dolan, 1995; Iidaka, Anderson, Kapur, Cabeza, & Craik, 2000; Kensinger, Clarke, & Corkin, 2003; Shallice, Fletcher, Frith, Grasby, Frackowiak, & Dolan, 1994;

Uncapher & Rugg, 2005, 2008). The idea that goal-directed attention plays a critical role during encoding has been further elaborated by Uncapher and Wagner (2009), who recently highlighted the contribution of dorsal parietal regions, in and around the intraparietal sulcus, to positive subsequent memory effects. This region is known to be involved generally when subjects must maintain attention in a goal-directed fashion (Corbetta, Patel & Shulman, 2008; Corbetta & Shulman, 2002), suggesting that the recruitment of dorsal parietal mechanisms during successful encoding reflects the allocation of top-down attentional control toward the inputs that are to be remembered. Thus, it seems plausible that fronto-parietal neural activity observed in subsequent memory analyses at least partially reflects the allocation of attention to perceptual and conceptual representations related to the studied item.

While the evidence above suggests that unsuccessful encoding arises simply from a failure to engage top-down control, there is also evidence that subsequently forgotten trials can be associated with a distinct pattern of brain activity (e.g., Otten & Rugg, 2001b; Wagner & Davachi, 2001; for a review see Uncapher & Wagner, 2009). Specifically, increased activity in ventral lateral parietal, medial parietal, and posterior cingulate cortical areas has consistently been found to predict subsequent forgetting. This suggests that these regions play some role in producing forgetting, but the mechanism(s) through which they negatively influence learning remains unclear. One hypothesis, which focuses on activity in ventral parietal cortex, near the temporo-parietal junction (TPJ), is that this activity reflects reflexive orienting toward representations that are not related to the encoding task (Cabeza, 2008; Uncapher & Wagner, 2009). For example, if someone nearby says your name while you are being introduced to a co-worker you are likely to reflexively orient to this salient perceptual input and thus fail to attend to the name of your co-worker. This interpretation builds on a rich attention literature documenting that the ventral parietal cortex is involved in attentional capture by abrupt onsets or salient stimuli (Corbetta & Shulman, 2002; Corbetta et al., 2008). From this perspective, when subjects engage ventral parietal reflexive attention mechanisms to orient to information that is not relevant to the later memory test, they are prone to subsequently forget the to-be-encoded information. Thus, one potential mechanism by which failed encoding may arise is by distraction from task-irrelevant inputs that steal attention from the to-be-encoded items. Further work is needed to isolate this as the mechanism behind these negative subsequent memory effects.

In summary, many instances of forgetting can be explained by a disruption of event encoding. This can occur either because we fail to activate fronto-parietal control mechanisms that orient attention to the relevant dimensions of the event or because ventral parietal regions related to reflexive attentional capture are engaged by distracting, task-irrelevant information. These processes have been described as making separate contributions, but it is also possible that goal-directed control and reflexive capture interact in some competitive fashion. For example, a lapse in top-down control may set

the stage for attention to be captured by irrelevant representations (e.g., Weissman, Roberts, Visscher, & Woldorff, 2006), or reflexive shifts of attention to irrelevant representations may interrupt our top-down focus of attention. Of course, failures to encode cannot explain all instances of forgetting. It is clear that in many situations we form a memory of an event and are able to recall it for some time afterwards, only to later lose that ability and be left with the distinct feeling of having forgotten something we once knew. Thus, other mechanisms are necessary to explain why and how some memories transition from memorable to forgotten.

Forgetting due to disrupted consolidation

What could cause us to forget something that we once knew? One possibility is that a memory trace, once formed, may be subject to damage or disruption. The most prominent modern version of this account focuses on disruption that occurs during consolidation – the process by which memories that are initially stored in a temporary, fragile state in the MTL are slowly "consolidated" into more durable, long-term representations distributed throughout the cortex (McGaugh, 2000; Müller & Pilzecker, 1900; Squire & Alvarez, 1995). During this initial period of consolidation – which has been argued to last anywhere from hours to years – recent memories are thought to be vulnerable to disruption from new experiences. By this account, forgetting arises because we experience new events before we have a chance to fully develop lasting traces of earlier events (Wixted, 2004, 2005).

One of the strongest forms of evidence in favor of disrupted consolidation is that damage to the MTL causes a pattern of forgetting known as temporally graded retrograde amnesia. In addition to impairments in learning new information, amnesics show forgetting of memories that were acquired before the damage occurred, even extending years prior to the onset of amnesia. Importantly, such instances of retrograde amnesia display a temporal gradient, where the most recent memories are the ones most likely to be forgotten (Ribot, 1882; Squire, Slater, & Chace, 1975; Zola-Morgan & Squire, 1990). This empirical observation led to the suggestion that memories require some period of time to consolidate (Squire, 1992).

A similar temporal gradient is observed in standard forgetting curves, motivating Wixted (2004) to propose that disrupted consolidation may account for forgetting in the healthy brain. The forgetting curve, first detailed by Ebbinghaus (1885/1913), shows that most forgetting occurs in the initial hours and days after a study episode, with more remote memories in the tail end of the curve often showing very little evidence of forgetting with the passage of additional time. Thus, in both the normal forgetting curve and in instances of MTL damage, the addition of time seems to render older memories more resistant to damage. One problem with evaluating this claim, and indeed the reason that this view might not be more widespread, is that most studies of human memory have tended to focus on fixed retention intervals

that are typically well within the consolidation period. Thus, many studies of memory may simply be poorly designed to detect forgetting due to disrupted consolidation.

While the disrupted consolidation account holds promise for explaining forgetting, it has not yet translated into functional neuroimaging research. To date, fMRI studies of consolidation have focused on demonstrating that consolidation involves the transfer of memories from the MTL to cortical regions. These studies have sought to show that retrieving older memories results in less hippocampal activity, suggesting that after time these memories have been transferred to cortical sites and no longer require the hippocampus to be retrieved (e.g., Haist, Gore, & Mao, 2001; Niki & Luo, 2002; but see Addis, Moscovitch, Crawley, & McAndrews, 2004; Gilboa, Winocur, Grady, Hevenor, & Moscovitch, 2004). However, even if these studies were able to conclusively provide evidence in favor of consolidation, none of the extant studies provides any insight into whether encoding new experiences can disrupt consolidation of earlier memories.

The disrupted consolidation theory described here is only one specific instantiation of a general class of theories that posit that stored memories are vulnerable and can be damaged by new experiences. As a general account of forgetting, these disrupted storage theories share a key limitation with the failure-to-encode account described earlier: They seem to predict that forgetting should be a permanent phenomenon. If the trace was not formed or has been disrupted in some way, then it is unclear why an experience that is forgotten at one point in time should ever be remembered later. In contrast to that view, however, we often experience momentary forgetting of some fact or event, only to later have this memory come back to mind. This common experience highlights the point that transient instances of forgetting can occur even when the underlying memory trace exists. To account for such findings, we need a mechanism that can explain why forgetting can occur in one retrieval situation and not another. In the following sections we detail several factors that promote forgetting, even when a memory trace still exists.

Forgetting due to retrieval competition

One situation that is known to induce forgetting is when a retrieval cue is related to multiple associated memories, especially when alternative memories are more strongly activated than the desired memory. In these situations, the alternative traces compete for access and interfere with the ability to retrieve the desired information. Consider, for example, trying to remember the name of a particular elementary school teacher. In some situations the retrieval cues lead directly to the desired memory (e.g., the name of your second-grade teacher) and the information is retrieved almost effortlessly. At times, though, we fail to remember the name because other memories that are strongly linked to the cues (e.g., the name of your third-grade teacher) spring to mind more readily. Once we have retrieved an alternate memory it can

often be difficult to move on to the desired target: The incorrect representation interferes with or blocks the ability to retrieve the desired memory. The proposal that memories compete for access and can block subsequent recall attempts has been long advanced as a primary cause of forgetting (e.g., McGeoch, 1942) and is instantiated in many modern computational models of memory as the primary mechanism by which forgetting occurs (e.g., Anderson, 1983; Mensink & Raaijmakers, 1988).

Retrieval competition is often investigated in fan effect studies (Anderson, 1974), where subjects are taught a set of propositions (e.g., "The *farmer* is in the park" and "The *doctor* is in the school"), with some items appearing in multiple propositions (e.g., "The *farmer* is in the bank"). The standard finding is that subjects are slower and less accurate at recognizing propositions when they contain items that are associated with multiple propositions. The interpretation of this effect has focused primarily on the idea that a finite amount of activation is shared between all the possible representations within a fan. Thus, when there are many possible responses it becomes more difficult to retrieve any one of them. Similarly, if one representation is strengthened, then the other representations are necessarily weakened.

Interference has also been extensively explored in the classic A–B, A–C learning paradigm (for reviews, see Anderson & Neely, 1996; Wixted, 2004). In these experiments, subjects first learn a list of A–B cue-associate word pairs (e.g., Shoe–House) and then later study a second list of word pairs. Critically, some of the pairs in this second list share a cue word with a pair from the earlier list (e.g., Shoe–Rope; A–C pairs). Thus, competition arises between the B and C terms due to their shared retrieval cue (A), thereby increasing the likelihood of forgetting. Indeed, increased retrieval failures are observed when subjects are later tested on the B or C terms. For example, forgetting of B items is much greater if it is followed by a new list of A–C pairs than a condition where entirely unrelated C–D pairs are learned (Müller & Pilzecker, 1900). Historically, the distinction between the temporal order of these interference effects has been quite influential, with the impairment of originally studied A–B pairs referred to as retroactive interference and the negative influence of past learning on acquisition of the new A–C pairs referred to as proactive interference. Many modern theories of forgetting (e.g., Mensink & Raaijmakers, 1988), however, attribute both types of interference effects to a common competition mechanism.

The ability to overcome competition is clearly important for many acts of remembering, as available retrieval cues often remind us of many things beyond the memory we wish to retrieve. A large body of neuropsychological and neuroimaging evidence indicates that overcoming retrieval competition is heavily dependent on lateral PFC. Lesion evidence has shown that PFC damage causes increased distractibility and a tendency to persevere on incorrect responses. For example, frontal lobe patients often perform as well as controls in the initial acquisition of A–B word pairs, but suffer considerable difficulty recalling the subsequently learned A–C pairs (e.g., Shimamura,

Jurica, Mangels, Gershberg, & Knight, 1995). Indeed, when presented with the A retrieval cue on the final test and asked to recall the C items, frontal lobe patients often make competition-driven errors by recalling the B items.

While lesions to lateral PFC generally result in increased susceptibility to proactive interference, it is less clear from lesion studies which specific regions within lateral PFC are critical for resolving mnemonic competition. Increased proactive interference effects have been associated with damage to both left (Moscovitch, 1982; Smith, Leonard, Crane, & Milner, 1995) and right PFC (Smith et al., 1995; Turner, Cipolotti, Yousry, & Shallice, 2007), while other studies have found relatively normal proactive interference in patients with frontal lobe damage despite impairments on other tests designed to measure frontally mediated control processes (Janowsky, Shimamura, Kritchevsky, & Squire, 1989). This variability in outcomes is perhaps not surprising, though, given the variability in the extent and location of naturally occurring lesions. Therefore, it is often difficult to draw conclusions from the lesion data other than the general implication that lateral PFC is important for resolving competition in memory.

Greater specificity regarding the role of distinct PFC subregions in resolving competition has been obtained through the higher spatial resolution afforded by positon emission topography (PET) and fMRI. The consensus from extant neuroimaging studies is that resolving interference in memory is most commonly associated with left ventrolateral PFC (VLPFC). One of the earliest neuroimaging studies of retrieval competition, where subjects underwent PET while performing a standard A–B/A–C learning paradigm (Dolan & Fletcher, 1997; for a similar fMRI result see Henson, Shallice, Josephs, & Dolan, 2002), revealed increased activity in left lateral PFC (including both VLPFC and dorsolateral prefrontal cortex, DLPFC) when subjects studied A–C items compared to when they studied entirely new word pairs (D–E pairs; see Figure 7.2). Subsequent work revealed that rearranging previously studied word pairs also leads to increased left VLPFC activity (Fletcher, Shallice, & Dolan, 2000), suggesting that lateral PFC is engaged whenever irrelevant associations have been previously learned and are no longer relevant to the current encoding task. Henson et al. (2002) elaborated on this general pattern by showing that activation in left VLPFC decreases with subsequent presentations of a word pair, suggesting that activity declines as an association is strengthened and less interference is experienced.

While the above studies revealed engagement of left VLPFC during encoding in the face of interference, similar activity is observed during interference-laden retrieval. Specifically, left VLPFC, along with anterior cingulate cortex (ACC), is engaged when subjects must retrieve A–C pairs after prior A–B learning (Henson et al., 2002). Similarly, left VLPFC engagement has been observed in studies of the fan effect, with increased VLPFC activity during high- compared to low-fan situations (Sohn, Goode, Stenger, Carter, & Anderson, 2003; Sohn, Goode, Stenger, Jung, Carter, & Anderson, 2005). More recently, Danker, Gunn, and Anderson (2008) showed that two distinct

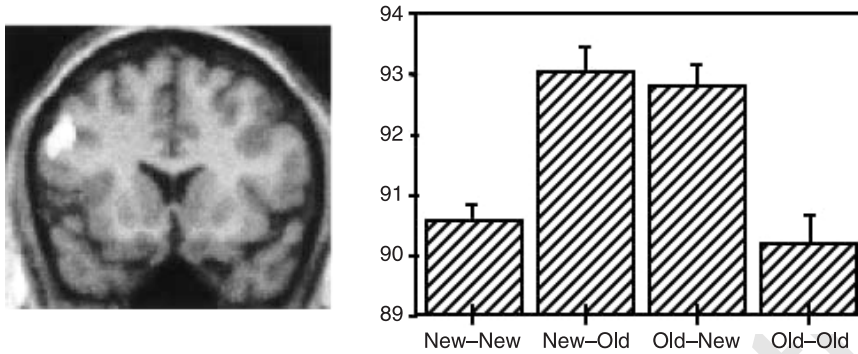

Figure 7.2 Activation in the left lateral PFC as a function of encoding condition (Dolan & Fletcher, 1997). "New–New" corresponds to encoding of a novel word pair; "New–Old" and "Old–New" correspond to a word pair in which one member of the pair is novel and the other was previously studied with a different word; "Old–Old" corresponds to a word pair that is repeated, intact. The left lateral PFC is maximally engaged (in the left panel, see the white activation overlaid on a structural image) when the word pair being encoded partially overlaps with a previous pair (i.e., when interference is present).

regions within left VLPFC respond differentially to two different aspects of controlled retrieval – manipulation of fan interference was associated with activation in left mid-VLPFC, whereas left anterior-VLPFC did not respond to the fan but was sensitive to the amount of training that was performed on the target association (see Figure 7.3 for more detail on PFC anatomy). This dissociation is consistent with the proposal that two distinct subregions in left VLPFC subserve separable processes during retrieval (Badre & Wagner, 2007). According to this model, anterior VLPFC mediates controlled retrieval of representations whenever retrieval cannot be done relatively automatically, whereas mid-VLPFC is engaged post-retrieval to resolve competition amongst active representations.

Beyond episodic memory, left mid-VLPFC is engaged in other situations that involve selection in the face of mnemonic interference. For example, activity in this region is consistently observed during semantic retrieval when one must select between multiple competing responses (e.g., Badre, Poldrack, Pare-Blagoev, Insler, & Wagner, 2005; Thompson-Schill, D'Esposito, Aguirre, & Farah, 1997; for a review, see Badre & Wagner, 2007). Critically, lesions studies have shown that damage to this region, in particular, is associated with difficulty retrieving relevant semantic representations from amongst competitors (Martin & Cheng, 2006; Metzler, 2001; Thompson-Schill, Swick, Farah, D'Esposito, Kan, & Knight, 1998). Left mid-VLPFC also plays a critical role in resolving proactive interference that accumulates over trials in working memory tasks, as revealed by functional neuroimaging (for a review, see Jonides & Nee, 2006), lesion (Thompson-Schill et al., 2002)

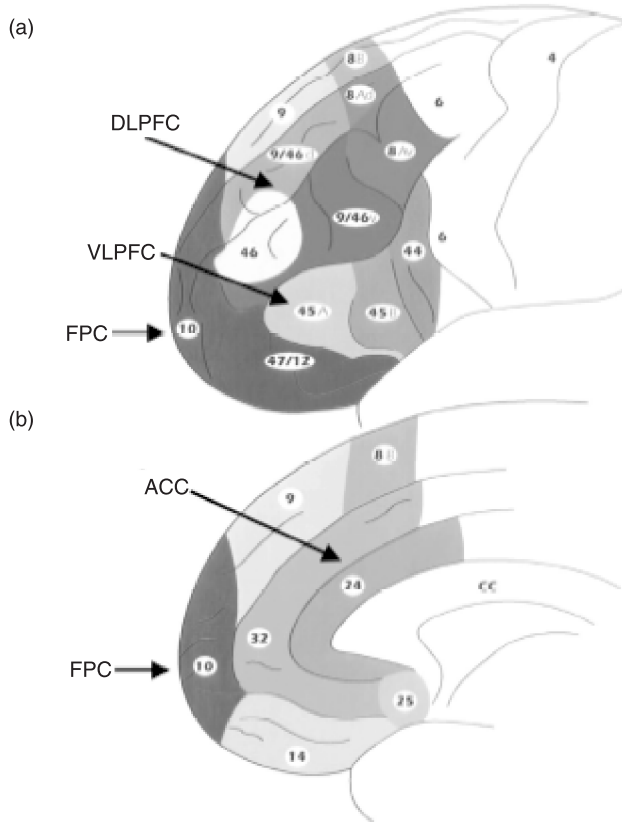

Figure 7.3 Organization of prefrontal cortex. (a) Lateral view of PFC and corresponding cytoarchitectonic areas. DLPFC corresponds to areas 46 and 9/46, while VLPFC corresponds to areas 47/12, 45, and 44. In this review, we highlight functional differences between anterior VLPFC (area 47/12) and mid-VLPFC (area 45). FPC corresponds to area 10. (b) Medial view of PFC. Medial portion of area 10 corresponds to FPC and ACC corresponds to areas 32 and 24. Adapted from Petrides and Pandya (1999).

and transcranial magnetic stimulation studies (Feredoes, Tononi, & Postle, 2006). Consideration of all the foregoing results suggests that left mid-VLPFC is critical for resolving interference across a variety of episodic, semantic, and working memory tasks.

In summary, competition can powerfully impact the likelihood of retrieval success, as inappropriate memories can dominate and preclude retrieval of desired memories. This is most evident in patients with frontal lobe damage, who suffer substantial problems selecting the most appropriate response and instead persevere on prepotent, incorrect responses. In healthy subjects, it is also clear that some instances of forgetting can be explained by mnemonic competition. For example, attempts to recall the name of an actress from a

movie can often be met with frustration as names of other actresses, similar in career history or appearance, leap to mind. Recent neuroimaging work has built on general evidence from lesion studies that underscored the importance of PFC in resolving retrieval competition by specifically implicating mid-VLPFC in resolving interference amongst competing representations. Therefore, some instances of forgetting may be due to a failure to sufficiently engage mid-VLPFC in the face of competition at retrieval.

As a final point, it is worth noting that modern accounts of interference-related forgetting have tended to focus almost exclusively on competition that occurs during retrieval (but see Dolan & Fletcher, 1997; Fletcher et al., 2000; Henson et al., 2002). That is, all learned responses are assumed to be stored in memory and compete for access at the time of test. When the desired response loses the competition, forgetting occurs. However, there is evidence from the classical interference literature that is difficult to explain entirely through retrieval-stage competition. Most notably, Melton and Irwin (1940) reported substantial retroactive interference effects even under conditions where there were few overt intrusions of the interfering material and the frequency of intrusions did not relate in any sensible way to the magnitude of interference. While overt intrusions are an imperfect measure of competition (i.e., subjects could be covertly retrieving competing items), Melton and Irwin (1940) suggested that a second factor, in addition to mnemonic competition, was necessary to explain interference-related forgetting. The second factor they proposed – unlearning of the association between the cue and the interfering response – has not been supported by empirical evidence, but an influential idea that arose from their proposal is that competition elicits a second process that actively reduces competition (e.g., Anderson, 2003; Osgood, 1949; Postman, Stark, & Fraser, 1968). In the following section, we will describe the modern descendant of this idea and show how this secondary mechanism can also produce forgetting.

Forgetting as a consequence of resolving competition

While retrieval is often thwarted by strong, irrelevant memories that block access to a currently desired memory, this interference can be overcome, allowing retrieval of the initially obscured information. One account of cognitive control during retrieval has suggested that this form of conflict resolution is achieved by inhibitory processes that weaken the representations of prepotent competitors, making them less interfering and thus allowing goal-directed control over retrieval (for reviews see Anderson, 2003; Levy & Anderson, 2002). This form of control does not produce forgetting at the time of the initial retrieval – in fact, it counteracts retrieval competition and thus promotes successful remembering. Rather, the inhibition of competing memories lingers and produces forgetting later when those items become goal-relevant and thus need to be recalled. From this perspective, some instances of forgetting reflect the consequence of having resolved retrieval

competition in the past. Such inhibitory processes have now been implicated in at least two distinct situations: when we wish to selectively retrieve a particular memory amongst competing alternatives; and when there is an explicit attempt to prevent a specific memory from being retrieved.

Selective retrieval

The idea that inhibition may be involved in achieving control during competitive retrieval situations has been explored in the retrieval practice paradigm (Anderson, Bjork, & Bjork, 1994), a procedure which bears many similarities to the classic retroactive interference paradigm. In a typical experiment, subjects study category–exemplar word pairs (e.g., fruit–apple, fruit–banana, drink–whiskey, drink–rum) and then engage in selective retrieval practice of some of the items from some of the categories (e.g., "fruit–a____" might be given as a cue to recall "apple"). After a delay, subjects are then asked to recall all of the exemplars they studied earlier. As would be expected, the items that were practiced during the selective retrieval practice phase (referred to as RP+ items) are recalled more often than baseline items, which were exemplars from categories that were not tested at all during the selective retrieval phase (e.g., "whiskey" or "rum", referred to as NRP items). More interestingly, items from the practiced categories that were not practiced themselves (referred to as RP– items) are recalled less often than the baseline (NRP) items (see Figure 7.4). Thus, selectively retrieving associates of a cue strengthens those items, but also weakens other unpracticed associates related to that cue. This finding, that selective retrieval can cause forgetting of competing memories, has been referred to as retrieval-induced forgetting (RIF), and it has been interpreted as evidence that inhibition is engaged

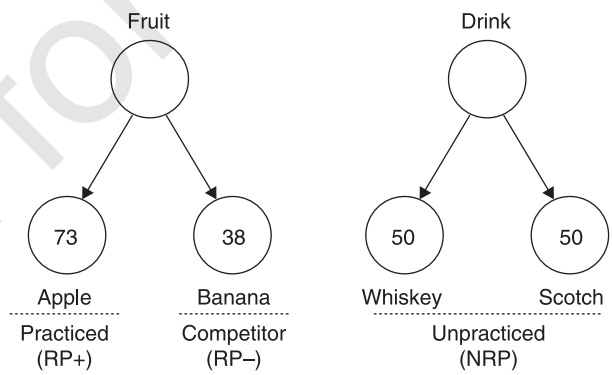

Figure 7.4 Schematic of retrieval-induced forgetting. Practiced items (RP+) are typically better remembered than baseline (NRP) or competing (RP–) items (numbers reflect percentage recall). Critically, RP– items are typically more poorly recalled than NRP items. The recall impairment for RP– items, relative to NRP items, reflects the magnitude of RIF.

during selective retrieval in order to dampen the interference from competing representations. This inhibition putatively promotes successful retrieval and indirectly produces later forgetting.

The basic RIF effect – forgetting of unpracticed items from practiced categories – is not uniquely diagnostic of inhibition. Increased retrieval competition could explain such forgetting because the practiced items are strengthened and should therefore cause even greater competition when the nonpracticed competitors are to be recalled during the final test. Several findings argue against such an interpretation, however, and support the inhibition explanation. First, RIF occurs even when items are tested with retrieval cues that were not studied earlier (e.g., "monkey–b_____" for "banana"; Anderson & Bell, 2001; Anderson, Green, & McCulloch, 2000; Anderson & Spellman, 1995; Aslan, Bäuml, & Pastötter, 2007; Camp, Pecher, & Schmidt, 2005; Johnson & Anderson, 2004; Levy, McVeigh, Marful, & Anderson, 2007; MacLeod & Saunders, 2005; Saunders & MacLeod, 2006). This is inconsistent with a pure retrieval-competition explanation as there is no reason to think that the practiced items should provide competition in this situation (e.g., presenting "monkey" as a retrieval cue should not make subjects think of "apple"). Further evidence of the cue-independent nature of RIF comes from reports that memory for RP– items is also impaired on tests of recognition memory (Hicks & Starns, 2004; Spitzer & Bäuml, 2007; Starns & Hicks, 2004; Verde, 2004) and implicit lexical decision (Veling & van Knippenberg, 2004). Thus, it appears that the forgetting occurs due to weakening of the competitors, rather than simply strengthening of alternative representations. Second, RIF is strength-independent, such that the magnitude of forgetting does not depend on the degree of strengthening of the practiced memories. This directly challenges the retrieval competition account, which predicts that forgetting arises because the practiced memories are strengthened, blocking later access to the subsequently relevant competitors. This decoupling between strengthening of initial targets and impairment of competitors can be observed in situations where targets are strengthened without a corresponding impairment for competitors (Anderson et al., 1994; Bäuml, 1996, 1997; Bäuml, & Hartinger, 2002; Ciranni & Shimamura, 1999), and in situations where competitors are forgotten without clear evidence of targets being strengthened (Storm, Bjork, Bjork, & Nestojko, 2006). Third, RIF is stronger for competitors that provide more interference during initial selective retrieval (e.g., "banana" is more likely to be forgotten than "kiwi"; Anderson et al., 1994; Bäuml, 1998). This finding suggests that RIF is interference-dependent, challenging the response competition account that predicts that strong and weak competitors alike should be influenced. Taken together, these results strongly support the inhibitory account of RIF.

As discussed earlier, neuroimaging data indicate that lateral PFC is engaged when competition must be resolved during selective retrieval. On the one hand, PFC could be engaged in response to the presence of conflict or in

service of resolving competition in some noninhibitory manner. On the other hand, frontal regions – or perhaps a subset of them – may directly mediate the inhibitory process that is measured by the behavioral RIF effect. Two recent fMRI studies have explored this relationship between PFC activity, retrieval competition, and inhibition (Kuhl, Dudukovic, Kahn, & Wagner, 2007; Wimber, Rutschmann, Greenlee, & Bäuml, 2009). Kuhl et al. (2007) predicted that inhibition should cause competitors to be less interfering with subsequent retrieval practice and thus successive acts of selective retrieval should require less control (i.e., recalling "apple" should make it easier to recall "apple" later due to inhibition of "banana"). Consistent with this prediction, Kuhl et al. found that lateral and medial PFC showed a pattern of decreasing activation across repeated retrieval practice trials. While intriguing, this pattern alone would be expected even from a purely noninhibitory response competition account, as successive trials should lead to strengthening of the target and therefore less control would be needed with each subsequent attempt (i.e., recalling "apple" gets easier simply because "apple" is strengthened). A second analysis, however, directly tested for a relationship between the decreases in PFC engagement and the weakening of competitor (RP–) items. This analysis revealed that two subregions within PFC – ACC and right anterior VLPFC – exhibited decreases in activation in proportion to the forgetting that competing memories suffered (see Figure 7.5). The authors argued that these decreases reflected the reduced engagement of control processes that are engaged in relation to the strength of competing memories.

The relationship between selective retrieval and competitor forgetting was also addressed by Wimber et al. (2009), in a study that directly contrasted selective retrieval with a nonselective condition where the word pairs were simply re-presented. This re-presentation condition is known to produce comparable strengthening of the practiced items yet no inhibition of competitors (Bäuml, 1996, 1997; Bäuml, & Hartinger, 2002; Ciranni & Shimamura, 1999). Since both conditions are similar in terms of strengthening, Wimber et al. reasoned that additional activity observed in the selective retrieval condition should reflect, at least in part, processes involved in inhibiting competitors. Indeed, this contrast (retrieval > re-presentation) revealed activity within lateral and medial PFC, presumably reflecting the engagement of control processes that are needed to a greater extent in the selective retrieval condition. Moreover, Wimber et al. found that the difference in activation during selective retrieval vs. re-presentation in several PFC regions – specifically, ACC and DLPFC – was correlated with behavioral evidence of competitor forgetting. The localization within ACC was highly consistent with the ACC region that Kuhl et al. found to be correlated with competitor forgetting (see Figure 7.5).

The involvement of PFC during selective retrieval is also supported by an event-related potential (ERP) study. Using a procedure similar to the one employed by Wimber et al. (2009), Johansson, Aslan, Bäuml, Gabel, and

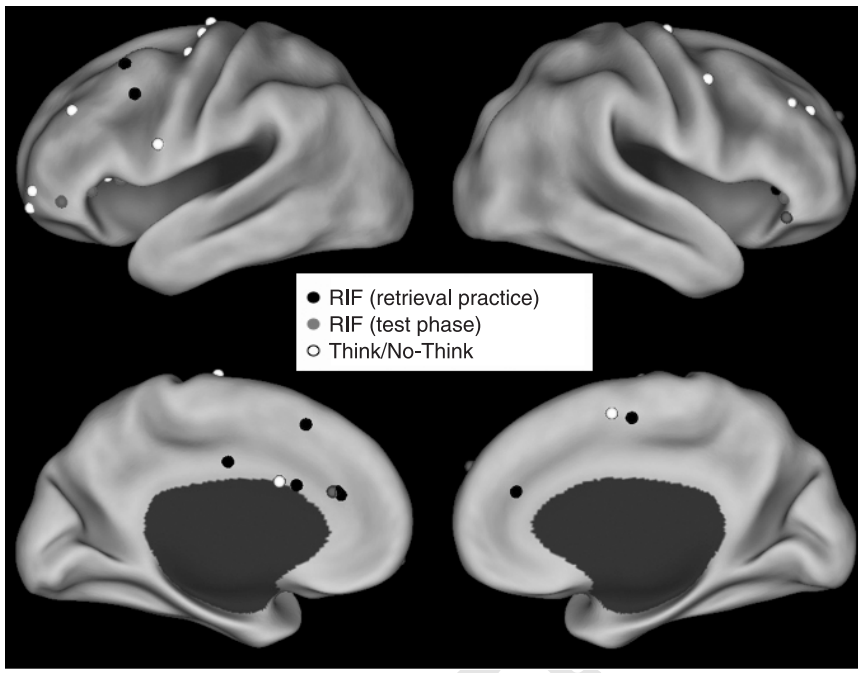

Figure 7.5 PFC regions that predict behavioral inhibition. Plotted here are the peak activations that showed a positive between-subject correlation with behavioral inhibition from six fMRI studies of inhibitory control in memory (Anderson et al., 2004; Depue et al., 2007; Kuhl et al., 2007, 2008; Wimber, Bäuml, Bergström, Markoponlos, Heinze, & Richardson-Klavehn, 2008, Wimber et al., 2009). The magnitude of behavioral inhibition was calculated for each subject based upon the difference between recall for baseline items and the putatively inhibited items (RP– items in the RIF studies and NT items in the TNT studies). This behavioral inhibition score was then regressed upon the main contrast in the study, to reveal regions which were more active for subjects who more successfully inhibited. The black foci represent correlations from the retrieval practice phase of RIF studies, while the grey foci represent correlations from the test phase of RIF studies. The white foci are from TNT phase data. In general, the RIF results tend to converge in ventral regions, with noticeable clustering in anterior VLPFC and ACC. By contrast, TNT results tend to appear more in DLPFC and frontopolar cortex. However, there are only a few studies of each type displayed here and there is considerable variability in the location of these peaks, suggesting that further work will be needed to clearly localize these effects.

Mecklinger (2007) found that selective retrieval produced an enhanced positive component, relative to the re-presentation condition, over frontal electrode sites. Importantly, this enhanced activity did not reflect strengthening of the practiced items because the two conditions yielded comparable facilitation. Rather, the magnitude of this positive frontal component during

retrieval practice predicted how much forgetting subjects experienced for the competitor (RP–) items. While localization of the source of ERP components is difficult, the frontal effect observed in these studies corresponds generally with the prior fMRI findings on the involvement of PFC during selective retrieval and suggests again that the degree to which these regions are engaged relates to subsequent forgetting.

When considered alongside the retrieval competition literature, these studies suggest a tentative model of PFC functioning during selective retrieval. Left mid-VLPFC is activated during situations that feature mnemonic competition, but, to date, there is little evidence that the mechanisms subserved by this region correlate with later forgetting of competitors. This suggests that left mid-VLPFC plays a direct role in resolving competition, but not in a manner that is related to subsequent inhibition of the nonselected items. This is consistent with the idea that left mid-VLPFC is engaged post-retrieval to select amongst multiple active representations (Badre & Wagner, 2007).

In contrast to left mid-VLPFC, there is accumulating evidence that activity in DLPFC, anterior-VLPFC, and ACC are related to the forgetting that competing memories suffer (a putative result of inhibition). One interpretation of these relationships is that lateral PFC mechanisms (e.g., DLPFC and right anterior-VLPFC) guide attention toward task-relevant representations. This orienting of attention then indirectly produces inhibition of the competitors, consistent with a biased competition account (e.g., Miller & Cohen, 2001). Interestingly, a recent computational model of RIF has suggested that the weakening of competing representations could occur entirely locally within the MTL, suggesting that the role of PFC may only be involved in selecting representations and not directly involved in inhibition (Norman, Newman, & Detre, 2007). Alternatively, lateral PFC regions may implement a form of inhibitory control that directly weakens the competing representation (see Levy & Anderson, 2002). While distinguishing between these accounts is difficult, it is worth emphasizing that both accounts predict that lateral PFC regions should be engaged in relation to the strength of competing memories. Interestingly, two recent studies (Kuhl, Kahn, Dudukovic, & Wagner, 2008; Wimber et al., 2008) reported that when initially selected-against competing memories are subsequently retrieved (i.e., when they later become retrieval targets), activation is observed in anterior-VLPFC that specifically relates to the magnitude of weakening that competitors suffered. Thus, consistent with evidence from other retrieval contexts, there is strong evidence that anterior VLPFC is sensitive to the strength of information being retrieved (Badre et al., 2005; Badre & Wagner, 2007; Danker et al., 2008; Wagner, Maril, Bjork, & Schacter, 2001). The relationship between ACC and competitor forgetting is potentially consistent with other findings that implicate ACC in the detection of conflict (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Braver, Barch, Gray, Molfese, & Snyder, 2001; MacDonald, Cohen, Stenger, & Carter, 2000; van Veen & Carter, 2002). That is, in the

retrieval practice paradigm, competing memories may elicit conflict that is detected by ACC; as competitors are weakened, responses in ACC should decrease correspondingly. Importantly, lateral PFC may be engaged in response to ACC conflict detection, thus supporting successful target retrieval (e.g., Badre & Wagner, 2004; Bunge, Burrows, & Wagner, 2004). While this hypothesis is speculative, it is consistent with theories regarding the roles of ACC and lateral PFC in cognitive control.

Perhaps challenging the conclusions of the foregoing section, neuropsychological evidence suggests that RIF can occur even when lateral PFC functioning is compromised. Specifically, the retrieval practice paradigm has now been studied in several populations associated with frontal functional impairments, including patients with frontal lobe damage (Conway & Fthenaki, 2003), Alzheimer's patients (Moulin, Perfect, Conway, North, Jones, & James, 2002), and healthy older adults (Aslan et al., 2007; Hogge, Adam, & Collette, 2008; Moulin et al., 2002). In each study, the "frontally-impaired" group showed normal RIF, suggesting that this form of inhibition may not depend upon intact frontal functioning. However, a difficulty arises in interpreting these studies because all but one (Aslan et al., 2007) relied solely on the studied categories as cues at test. As described earlier, that type of test does not distinguish between forgetting that is produced by inhibition during the earlier retrieval practice or by retrieval competition during the final test (i.e., is the forgetting due to strengthening of "apple" or weakening of "banana"?). In fact, populations with impaired PFC function are likely to be even more vulnerable to response competition – as we discussed above – and may therefore display very robust forgetting without any contribution of inhibition per se (Anderson & Levy, 2007). Aslan et al. (2007), however, found preserved RIF in older adults using independent probes, suggesting that RIF may actually be preserved in healthy aging. It is difficult to interpret this study, however, with respect to the involvement of PFC in RIF because Aslan et al. did not ascertain whether these older adults were experiencing any frontal lobe dysfunction – indeed, their retrieval performance, in general, did not suggest any deficits. Given this limitation and the fact that earlier studies were unable to disentangle response competition from inhibition, it remains uncertain whether normal PFC functioning is a prerequisite for RIF to occur.

Stopping retrieval

Another situation that requires control over memory is when we desire to prevent a memory from coming to mind. For example, when confronted with a reminder of something upsetting (e.g., seeing someone who recently witnessed you doing something embarrassing) we often wish to avoid thinking about the unpleasant thoughts associated with that event. Similarly, the ability to focus cognition in a goal-directed manner relies on the ability to selectively prevent task-irrelevant memories from entering awareness. In these situations the focus is not on selectively retrieving alternative memories; rather, the

desire is to simply stop the retrieval process itself. Recent research using the Think/No-Think (TNT) paradigm suggests that this situation also relies on inhibitory control that weakens the to-be-avoided memory, rendering it less intrusive. In a typical TNT study, participants learn a list of cue-target word pairs (e.g., ordeal–roach) and are then presented with some of the studied cue words (e.g., ordeal) and asked to either think of the associated word (roach) or prevent that word from coming to mind. After seeing these "Think" and "No-Think" cues multiple times, subjects are then asked to recall all of the words they studied earlier. If subjects are able to recruit control mechanisms to inhibit the unwanted memories on No-Think trials and if this suppression lingers, then these words should be less accessible later.

Unsurprisingly, when subjects were instructed to remember (i.e., Think condition), reminders enhanced later memory relative to baseline word pairs, which were studied initially but whose cues were not seen again during the TNT phase (see Figure 7.6). In contrast, when people try to prevent an associate from coming to mind (i.e., No-Think condition), subjects have more difficulty recalling these items than baseline items (Anderson & Green, 2001; Anderson et al., 2004; Depue, Banich, & Curran, 2006; Depue, Curran, &

(a)

	Study	TNT Phase	Test Phase: Same Probe	Test Phase: Independent Probe
No-Think	Ordeal–Roach	Ordeal	Ordeal	Insect r___
Think	Steam–Train	Steam	Steam	Vehicle t___
Baseline	Jaw–Gum		Jaw	Candy g___

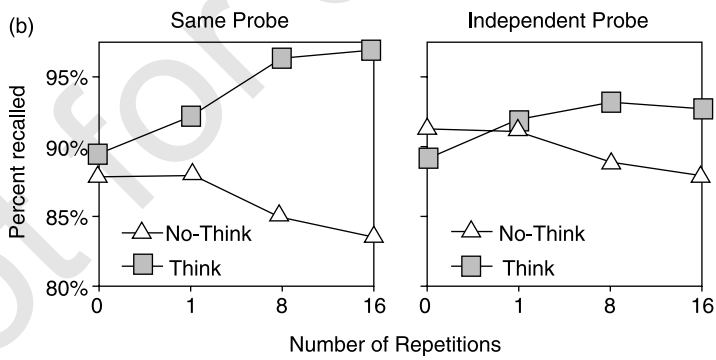

Figure 7.6 The Think/No-Think paradigm. (a) During the TNT phase, subjects are cued to think of the corresponding associate for Think items, but to avoid thinking of the response for No-Think items. (b) Final recall performance. Memory for the Think items increases as a function of repetition, while recall of the No-Think items decreases as a function of repetition. The No-Think impairment is apparent both in the Same Probe and Independent Probe tests.

Banich, 2007; Hertel & Calcaterra, 2005; Joorman, Hertel, Brozovitch, & Gotlib, 2005; Wessel, Wetzels, Jelicic, & Merckelbach, 2005; although, see Bulevich, Roediger, Balota, & Butler, 2006). Thus, avoiding a memory makes it harder to recall later even when it is desired, and this impairment is a function of the number of times that the thought has been avoided (Anderson & Green, 2001; Depue et al., 2006).

As was the case with RIF, the basic TNT forgetting effect is compatible with either an inhibitory process or a noninhibitory retrieval competition explanation. For example, subjects might generate diversionary thoughts when they see the No-Think cues. Subsequently, when presented with the same cues on the final memory test, the strengthened diversionary thoughts may come to mind and block retrieval of the original representation. Arguing against a pure noninhibitory account, however, is evidence that increased forgetting is observed even when subjects are provided with novel, extralist items as retrieval cues on the final test (e.g., "insect–r_____" for "roach"; Anderson & Green, 2001). This finding of cue-independent forgetting suggests that retrieval competition from diversionary thoughts cannot account for the observed memory impairments. While this result supports the inhibitory account, it is still unclear exactly how these avoided memories are inhibited as it is compatible with at least two distinct inhibitory mechanisms. First, as described earlier, subjects may generate diversionary thoughts as a means of preventing the original word from coming to mind (Hertel & Calcaterra, 2005). Then when No-Think cues are presented again, subjects may retrieve these earlier diversionary thoughts, creating a selective retrieval situation where the original learned words suffer from RIF. Alternatively, when confronted with a reminder of an unwanted memory, subjects may engage control processes that directly target the to-be-avoided memory and inhibit this representation. At present, it is unclear which of these two inhibitory accounts best describes forgetting in the TNT paradigm.

While extant behavioral data suggest an active inhibitory process is engaged in the TNT paradigm, fMRI studies have sought more direct evidence of inhibitory control during attempts to stop retrieval. Using neutral word stimuli, Anderson et al. (2004) found that No-Think trials are associated with elevated activity, relative to Think trials, in several frontal regions, including bilateral DLPFC, VLPFC, and ACC. Depue et al. (2007) extended this study, using negatively valenced photographs (e.g., a photograph of a car crash) as the to-be-avoided memories, and observed increased activation in a similar set of right frontal regions, including DLPFC, anterior VLPFC, and frontopolar cortex. Strikingly, both Anderson et al. (2004) and Depue et al. (2007) found that the magnitude of DLPFC engagement during No-Think trials predicted the amount of behavioral inhibition that subjects displayed on the final memory test (see Figure 7.5). These data suggest that lateral PFC is engaged during attempts to stop retrieval, with DLPFC, in particular, perhaps playing a key role in producing the subsequent forgetting of these avoided memories. Stopping retrieval is, therefore, not simply a failure to engage

retrieval processes; rather, activation of control-related prefrontal regions during No-Think trials suggests that subjects actively engage processes to prevent unwanted memories from coming to mind.

In addition to regions that are engaged by the No-Think task, fMRI studies have also identified regions that are less active during attempts to stop retrieval. In particular, both Anderson et al. (2004) and Depue et al. (2007) observed decreases in MTL activity during No-Think trials relative to Think trials. Decreased MTL activity during No-Think trials is not surprising, as this region is known to be active during conscious recollection (e.g., Eldridge, Knowlton, Furmanski, Bookheimer, & Engel, 2000; Kirwan & Stark, 2004) and the goal of the Think and No-Think tasks, respectively, is to engage and override conscious recollection. This difference, therefore, suggests that subjects are able to phasically regulate the activity of the MTL as necessitated by current goals, but it is unclear whether this difference is due to engagement during Think trials and/or disengagement during No-Think trials. Evidence in support of the latter explanation comes from the finding that the degree of hippocampal activity during No-Think trials is related to behavioral memory inhibition (see Anderson et al., 2004 for a description of this relationship), suggesting that the MTL modulation during No-Think trials is related to processes that produce the subsequent forgetting of the No-Think items. Taken together, it appears that attempts to stop retrieval are associated with increased lateral PFC activity and decreased MTL activity; both of these effects are related to subsequent forgetting.

Recent electrophysiological data suggest similar conclusions. Attempting to stop retrieval is associated with early frontal ERP components (Bergström, de Fockert, & Richardson-Klavehn, 2009; Mecklinger, Parra, & Waldhauser, 2009) that resemble the N2 component observed during the stopping of overt motor responses (Kok, 1986; Kopp, Matler, Goertz, & Rist, 1996). Interestingly, Hanslmayr et al. (2009) found that giving subjects advance warning about an upcoming No-Think trial led to a similar frontal negativity during the warning period, even before the cue word appeared. Critically, the magnitude of this anticipatory effect predicted subsequent forgetting, again linking frontal engagement to successful inhibition. In addition to these early frontal components, a late left parietal component is present selectively on Think trials (Bergström, et al., 2009; Bergström, Velmans, de Fockert, & Richardson-Klavehn, 2007), with the timing and topography of this component being consistent with the parietal old/new episodic memory effect that has been linked to the subjective experience of consciously recollecting a past event (e.g., Friedman & Johnson, 2000; Paller & Kutas, 1992; Rugg & Curran, 2007; Rugg, Schloerscheidt, Doyle, Cox, & Patching, 1996). Because this component is greatly reduced during the No-Think trials, these data suggest that executive control processes that stop retrieval eliminate this parietal retrieval-related component. Together, extant ERP and fMRI evidence suggests that the suppression of competing or avoided memories is associated with lateral PFC function. A fundamental objective for future research will

be to determine whether PFC control processes, including those mediated by DLPFC and VLPFC, implement the stopping of conscious recollection (or the suppression of competitors in the RIF paradigm), or whether these changes in PFC processing demands reflect the benefits of suppression accomplished through other mechanisms (e.g., processes within the MTL).

Forgetting due to ineffective retrieval cues

On some occasions we forget simply because the current retrieval cues are insufficient to bring the desired experience back to mind. This general, but fundamental, observation has been made in a number of different theoretical frameworks, including the encoding specificity principle (Tulving & Thomson, 1973) and context models of memory (e.g., Estes, 1955; Howard & Kahana, 2002; Mensink & Raaijmakers, 1989). According to the encoding specificity principle, the cues present during the encoding experience will be the most effective cues for later retrieving the memory, so a shift in the cues used to guide retrieval away from those present at encoding can cause forgetting. Context models expand this focus on specific cues to explain forgetting as a mismatch between the general context of the encoding situation and that of the retrieval situation, which arises because context varies over time and this constant updating results in a drift between encoding and retrieval (Estes, 1955; Howard & Kahana, 2002; Mensink & Raaijmakers, 1989; Polyn, Norman, & Kahana, 2009). Thus, when we later wish to bring these individual bits of information back to mind, we may fail to retrieve them because the test context is sufficiently different from the original study context so as to poorly cue memory. Common to both of these accounts is the idea that forgetting can be produced when the cues used to guide retrieval are insufficiently related to the desired memory and thus fail to reinstate it. This factor is clearly relevant for understanding forgetting and clearly differs from the other mechanisms advanced here, but as of yet little functional neuroimaging data have been gathered to examine the neural contexts that produce this form of forgetting (although, see Polyn & Kahana, 2008 for a review of early work on this topic).

Conclusions

Here we have argued that forgetting has several distinct causes, rather than being produced by any single mechanism. It seems clear that there are at least five factors that contribute to forgetting of past experiences. First, forgetting can be caused by a failure to encode the initial experience. Ineffective encoding sometimes occurs because of a failure to engage fronto-parietal mechanisms that direct attention to relevant representations for encoding, or because attention is captured by task-irrelevant representations, putatively marked by engagement of ventral parietal engagement, that distract encoding-relevant resources away from to-be-remembered items. Second,

intervening experiences, even those unrelated to the original event, can interfere with the MTL-dependent memory trace before it is fully consolidated. Finally, three other mechanisms focus on the retrieval dynamics created by the relationship between retrieval cues and target memories. When cues are strongly related to competing memories, failures to engage VLPFC can result in strong alternatives blocking retrieval of the desired memory. In situations where we are able to overcome such retrieval competition, however, it appears that the act of interference resolution is accomplished, at least in part, by processes that weaken the alternative memories, causing us to later forget these items. Such memory suppression is associated with activation in anterior VLPFC and DLPFC structures, revealing a relationship between cognitive control and forgetting. Lastly, forgetting can occur when the retrieval cues are simply insufficient to reinstate the desired memory.

Each mechanism proposed here accounts for critical aspects of forgetting, but is unable to explain all the data, suggesting that no one mechanism is sufficient to provide a coherent account of forgetting. It is also clear that while progress has been made in characterizing each of these forms of forgetting, many outstanding questions remain, particularly in terms of the neural mechanisms giving rise to forgetting. For example, it is clear that lateral PFC plays a crucial role during both the encoding of our experiences and during attempts to subsequently remember. Within lateral PFC, future work will need to carefully explore how PFC mechanisms involved in resolving retrieval competition (mediated by left mid-VLPFC) relate to those that correlate with later forgetting as a consequence of resolving competition. We do not wish to suggest, though, that these mechanisms will necessarily be associated with dissociable neural substrates, as many of the differences between them focus on the stage at which they operate (e.g., encoding or retrieval). For example, similar PFC regions may play a role in both failed encoding and failure to resolve interference during retrieval, but at different points in time. Finally, we emphasize that the five mechanisms proposed here likely do not constitute an exclusive list. Nevertheless, the lines of behavioral and functional neuroimaging research described herein hold promise for an increasingly specified account of why we sometimes fail to remember our past.

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