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How we forget may depend on how we remember

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Recent developments reveal that memories relying on the hippocampus are relatively resistant to interference, but sensitive to decay. The hippocampus is vital to recollection, a form of memory involving reinstatement of a studied item within its spatial–temporal context. An additional form of memory known as familiarity does not involve contextual reinstatement, but a feeling of acquaintance with the studied items. Familiarity depends more on extrahippocampal structures that do not have the properties promoting resistance to interference. These notions led to the novel hypothesis that the causes of forgetting depend on the memories' nature: memories depending on recollection are more vulnerable to decay than interference, whereas for memories depending on familiarity, the reverse is true. This review provides comprehensive evidence for this hypothesis.

> 'Without forgetting it is quite impossible to live at all.'
> Friedrich Nietzsche, On the Advantage and Disadvantage of History for Life

The two faces of remembering and forgetting

Whether or not we feel as positively as Nietzsche regarding forgetting, it is a fact that the vast majority of our life experiences are forgotten. The processes of forgetting are, therefore, at least as crucial to study as those of memory acquisition and retention. We outline a novel hypothesis, based on a recent proposal by Hardt *et al.* [1] on loss of hippocampal memories in rodents, that the characteristics of forgetting may be determined by the nature of the underlying declarative memory representations [While characterizing the mechanisms of forgetting is relevant to other forms of memory (e.g., procedural memory, priming), here we focus only on episodic memory (see Glossary).]

A wealth of research suggests that declarative memory is driven by two processes: recollection and familiarity [2] (Box 1). Recollection is a conscious process which involves reinstatement of an event from memory along with contextual details and an accompanying sense of self. In contrast, familiarity does not involve reinstatement of contextual details, but is accompanied by the feeling that an item had been previously encountered. The dual-process account has provided a comprehensive framework for studying memory for the past forty years. It is thus surprising that little is known about the forgetting patterns associated with these two processes. Importantly, although recollection and familiarity are retrieval processes, in this Opinion, terms such as 'memories relying on recollection or familiarity' refer to memories that were encoded in a manner and/or structure that supports their subsequent retrieval via recollection or familiarity, respectively.

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Keywords: forgetting; recollection; familiarity; interference; decay; hippocampus.

1364-6613/$ – see front matter
 http://dx.doi.org/10.1016/j.tics.2013.10.008

Glossary

AB/AC recall paradigm: in this paradigm, subjects study pairs of arbitrary word pairs (AB pairs; e.g., FIRE–DOG) and later learn new word pairs that partially overlap with the initially studied pairs (AC pairs; e.g., FIRE–TREE).
Deese–Roediger–McDermott (DRM) Paradigm: a method of creating high levels of false memories. In DRM studies, a list of words with a strong semantic theme is presented (e.g., words related to SLEEP). The critical lure is typically the word that best represents the semantic theme (e.g., SLEEP). This word is not presented during the study phase, but nevertheless has a particularly high probability of being falsely recognized or recalled during the test phase.
Episodic memory: memory for items or events that occurred within a specific place and time.
Proactive interference (PI): a phenomenon whereby past memories interfere with similar, more recent memories [83]. In a typical PI paradigm, lists of items from a certain semantic category (e.g., fruit) are presented. Interference is demonstrated by reduction in memory performance from one list to the next. 'Release from PI' [84] – namely, memory performance returning to its original level – may occur when the repeated semantic category is replaced with a new one.
Process Dissociation Procedure (PDP): a method of estimating the separate contribution of recollection and familiarity to memory performance [85]. In studies applying the PDP, two study lists, A and B, are typically submitted to a recognition test. The test includes two different tasks: inclusion and exclusion. In the inclusion task, participants are to respond 'yes' whether the presented item appeared in list A or in list B. In the exclusion task, participants are to respond 'yes' only if the item appeared in one of the lists. Exclusion false alarms (i.e., responding 'yes' to an item from the excluded list) are presumably driven by familiarity alone. Thus, such responses reflect retrieval devoid of recollective information regarding the list in which the item had been presented. Using this underlying logic, estimates of familiarity and recollection can be calculated based on the hit and false-alarm rates in each of the tasks.
Receiver Operating Characteristic (ROC) curves: in recognition experiments, ROC curves plot hit rates against false-alarm rates for each of (usually six) levels of subjective confidence. According to the dual-process approach, the degree to which a ROC curve is symmetrical reflects the degree to which recognition decisions are based on familiarity. Presumably, a completely symmetrical ROC curve reflects complete reliance on familiarity and, the more asymmetrical the ROC curve is, the more recognition decisions rely on recollection.
Remember/Know (R/K) paradigm: a paradigm [86] in which participants make a metacognitive judgment regarding each word retrieved. The judgment pertains to whether its retrieval was accompanied by contextual details of the study episode [a 'remember' (R) judgment] or not [a 'know' (K) judgment]. Typically, R responses are taken as indicators of recollection whereas K responses are taken as indicators of familiarity.
Retroactive interference: a phenomenon whereby presentation of stimuli after encoding hinders memory.

Box 1. Single- and dual-process models of recognition

Dual-process accounts [2,87,88] posit that memory is driven by recollection and familiarity. In contrast to this approach, single-process accounts of recognition have also been offered. According to such models, the empirical data regarding recognition can be accounted for by a single process: memory strength [89–91]. Thus, the various items submitted to a memory test are associated with different strength values on a hypothetical continuum. Items whose strength is greater than a certain criterion are perceived as 'old' (i.e., items that had been studied), whereas items whose strength is below that criterion are perceived as 'new' (i.e., items that had not been studied).

Critically, however, despite the differences between single- and dual-process models, both classes of models currently agree on the basic notion that recognition is driven by both recollection and familiarity. The source of disagreement between the two sides is whether, as dual-process models argue, recollection and familiarity signals can each be directly accessed or whether, as single-process models argue, both signals combine into one so-called 'strength signal' and it is this combined signal that guides recognition performance. However, the very existence of recollective and familiarity components that together contribute to performance is accepted by the vast majority of current recognition models.

Box 2. Neural correlates of recollection and familiarity

The critical role of the hippocampus in episodic memory has been recognized since Scoville and Milner's seminal findings regarding the famous patient H.M., who, as a result of removal of his hippocampus, suffered severe memory deficits [92]. Since then, the mnemonic function of the hippocampus has been more specifically defined and it is now well accepted that the hippocampus is critically involved in recollection of information from episodic memory [93,94] (but see [12]). By contrast, the PRc has been associated with familiarity [10,57,95].

Our brief review of the neuroanatomical correlates of recollection and familiarity begins with findings from animal studies. Recording studies, as well as neurotoxic-lesion studies, have shown that the hippocampus is implicated when recognition judgments cannot simply rely on familiarity, but involve a spatial or associative component [57]. By contrast, object-exploration studies have provided evidence that PRc-lesioned rats are specifically impaired when performance relies on familiarity [57].

Evidence from humans with hippocampal lesions has shown that such lesions have severe detrimental effects on recollection, although often sparing familiarity [57]. Thus, in recognition paradigms, patients with hippocampal lesions are impaired when recollection is required, but not when familiarity is required [74] (but see [96]). In addition, such patients generally show a disproportionate impairment in recall, which relies primarily on recollection, *vis-à-vis* recognition, which can rely also on familiarity [97,98] (but see [77]).

Studies using fMRI provide further illuminating evidence for the crucial role of the hippocampus in recollection. Strikingly, the vast majority of fMRI studies (reviewed in [10]) examining the neural underpinnings of recollection both at encoding and at retrieval have reported hippocampal activity. By contrast, when recognition decisions are based on familiarity, hippocampal activity is very rarely reported, but activity in the PRc is (but see [12]). These studies used a wide variety of paradigms including R/K and source and associative memory versus item memory, all converging on a similar pattern of results.

Most recently, magnetoencephalography (MEG) evidence [99] has also linked the hippocampus with recollection and furthermore provided data regarding the time course of recollective hippocampal activity.

We propose that the representations and structures that mediate recollection and familiarity involve fundamentally different forgetting mechanisms. Although our hypothesis has never been directly investigated in a single empirical study, a review of the memory literature provides striking evidence in favor of it. Thus, we review evidence suggesting that memories relying on recollection are forgotten primarily due to decay over time, but are relatively resistant to interference from irrelevant information. By contrast, memories relying on familiarity are prone to the detrimental effects of interference but show less effect of decay (Figure 1).

A neuroanatomical basis for resistance and proneness to interference

Our proposal stems from recent advances in the study of the circuit architecture of the hippocampus [1,3–5], the primary structure associated with recollection (Box 2 and Figure 2). Such research has shown that whereas much of the neocortex uses overlapping representations to represent similar stimuli, the hippocampus is unique in its ability to assign orthogonal representations to even highly similar stimuli [4,6,7]. Termed 'pattern separation', this property of the hippocampus enables similar memories to be distinguished from one another, such that the encoding of new, incoming experiences is unlikely to interfere with or override similar older memories. Importantly, pattern separation is unique to the hippocampus and is believed to be dependent on processes supported by multiple subfields (such as CA1, CA3, and the dentate gyrus) and their interaction that comprise the unique anatomical structure of the hippocampus. For example, in one biologically plausible neural

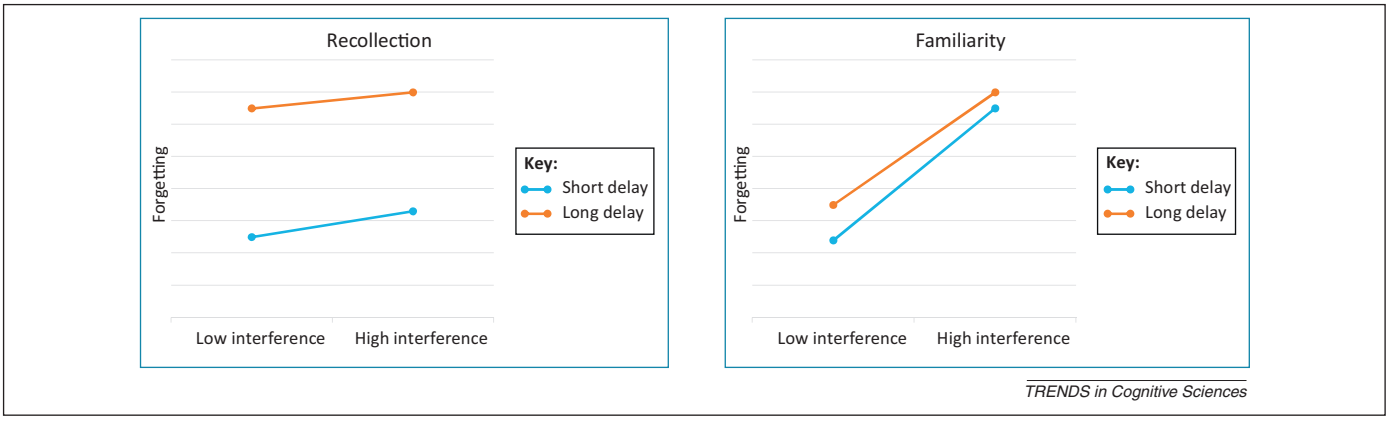

Figure 1. Effects of interference and delay on recollection and familiarity. Hypothesized results illustrating how manipulating interference and delay parameters should affect estimates of recollection and familiarity. Assuming learning involves verbal materials, low interference may include a non-verbal task, whereas high interference is a verbal task that resembles the one that is being targeted.

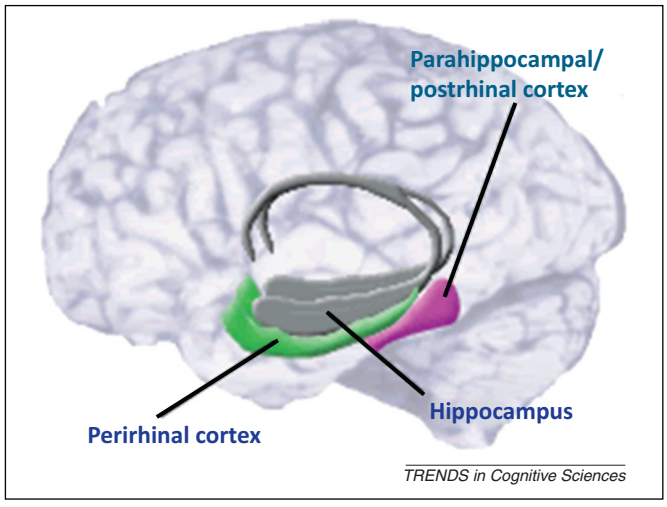

Figure 2. Medial temporal lobe regions supporting recollection and familiarity. Based on abundant evidence, the hippocampus supports recollection. Therefore, memories relying on this structure would be relatively immune to the effects of interference, but susceptible to decay. Because familiarity is supported by the perirhinal cortex (PRc), memories relying on this structure should show the reverse pattern. Reprinted with permission from Dr Andrew Doherty, Centre for Synaptic Plasticity, University of Bristol, Bristol, UK.

network model [the Complimentary Learning Systems (CLS) model] [7], CA3 plays an important role in binding the units of a representation, while simultaneously the dentate gyrus facilitates the orthogonal storage of the complete pattern [8]. During retrieval, recurrent connections in CA3 make it possible to recover an entire representation based on only a partial cue (i.e., 'pattern completion'). Although they are resistant to interference, a recent model provides a plausible mechanism by which hippocampal memory representations decay [9]. According to this model, neurogenesis of hippocampal granule cells results in remodeling of hippocampal circuits. This, in turn, causes forgetting by means of decreasing the likelihood of pattern completion of information previously represented in the hippocampal circuits that had been remodeled. Importantly, this neurogenesis process is ongoing and not dependent on exposure to new information. It is, therefore, more consistent with the notion of decay than interference. (See the supplementary data online for proposed computational implementations of decay and interference.)

Familiarity, by contrast, relies on medial temporal lobe (MTL) structures other than the hippocampus, such as the perirhinal cortex (PRc) (Figure 2) [10,11] (but see [12]). Because neocortical areas lack the complex architecture found in the hippocampus, processes such as pattern separation are typically not implicated in these areas. Instead, neocortical representations have been proposed to be encoded in self-organizing networks reliant on principles such as Hebbian learning [7]. Such learning entails that, for familiarity-based memories, similar experiences are often represented in overlapping networks of neurons. Although such organization has the benefit of allowing familiarity-based representations to accommodate novel representations as a function of their similarity to past experiences, it also renders familiarity-based representations vulnerable to interference, inasmuch as the encoding of novel stimuli will change previously established representations [13,14].

A functional description of the manner by which the hippocampus and PRc differ may also provide valuable insight into their different capacities for handling interference. The hippocampus supports episodic memory by binding the content of a memory to its unique spatial–temporal context [10,15]. In doing so, the hippocampus enables similar pieces of information (e.g., memories of similar conversations) to be distinguished from each other by the different contexts in which they were experienced (e.g., where, when, or with whom the conversations occurred). Such contextualization of memories is a means by which two potentially interfering memories may be encoded in representations that do not interfere with one another. The PRc, by contrast, does not encode the context of a memory, but rather specializes in processing the fine details of a single item [10,13]. Therefore, two episodes including the same item, or two similar items, may not be well distinguished from each other in that they are encoded in overlapping or interfering representations [13,14]. Finally, it has been recently shown [16] that, when encoding novel stimuli, the hippocampus specifically reactivates older, related stimuli. This has been proposed as a mechanism by which the hippocampus limits interference.

Operationalizations and manifestations of forgetting

Interference is typically induced by having participants process both study items and additional materials [17]. These additional materials are typically similar to the study materials and are thus are assumed to involve overlapping cognitive representations. Decay, by contrast, refers to a study–test delay interval that does not include a cognitive task aimed at causing interference. Nevertheless, even when examining decay, a certain level of interference from the external world and/or from internal thoughts and memories is present. The crux of the distinction between interference and decay pertains to whether the retention interval involves processing information similar to that committed to memory.

Importantly, forgetting, whether due to decay or interference, does not necessarily entail that a memory is lost. Rather, a memory trace may endure substantial decline in its accessibility or coherence. For example, a memory trace may be easily accessible immediately after learning, but at a later point may be recovered only with the aid of a specific cue, due to its decline in accessibility. This example highlights the importance of cues in providing access to memories. Namely, a memory trace may be inaccessible at times because the relevant cues, which could have otherwise triggered the memory, cannot be reinstated at the time of retrieval [18]. Thus, whereas forgetting may be defined as 'the inability to recall something now that could be recalled on an earlier occasion' [18], some views of memory posit that forgetting is, in fact, the inability to recall something now that could be recalled on a later occasion. Indeed, it has been demonstrated that information that was not accessible using a particular cue was accessible later when subjects were exposed to a different cue (e.g., [19]).

Notwithstanding this issue, here we do not address the question of whether forgetting entails memory loss or inaccessibility, but rather focus on the mechanisms underlying the empirical manifestation of forgetting. The model we

Table 1. Details of interference and delay studies[a]

Studies employing an interference manipulation				
Study	***Interference details***	***Recollection/ familiarity measurement***	***Detrimental effect of interference on recollection measure***	***Detrimental effect of interference on familiarity measure***
Hockley, 1992 [17]	Continuous recognition paradigm; retroactive interference was manipulated by varying the lag	Single words vs paired associates	×	✓
Murdock and Hockley, 1989 [20]	Continuous recognition paradigm; retroactive interference was manipulated by varying the lag	Single words vs paired associates	×	✓
Hockley, 1992 [17]	Multiple study–test sessions followed by a final recognition test; retroactive interference of an encoded list was caused by the additional study–test sessions that followed the encoded list	Single words vs paired associates	×	✓
Yonelinas and Levy, 2002 [24]	Continuous recognition paradigm; retroactive interference was manipulated by varying the lag	Item vs source memory	×	✓
Öztekin and McElree, 2007 [26]	PI task	Retrieval time course of false alarms, with familiarity being characterized by early and fast responses and recollection by slower response times	×	✓
Jacoby et al., 2001 [25]	PI task	PDP	×	✓
Winocur et al., 1996 [28]	AB/AC PI design	Induction of higher reliance on familiarity via an implicit memory test	No information	✓
Cary and Reder, 2003 [30]	Retroactive interference manipulated by list length (longer lists causing more interference)	R/K paradigm	✓	✓
Studies employing a delay manipulation				
Study	***Delay details***	***Recollection/ familiarity measurement***	***Detrimental effect of delay on recollection measure***	***Detrimental effect of delay on familiarity measure***
Brubaker and Naveh-Benjamin, 2013 [37]	24-h delay	Item vs associative memory	✓	×
Hockley and Consoli, 1999 [38]	30-min, 1-day, 2-day, and 1-week delays; for the 30-min delay, the retention interval was filled with a non-interfering task	R/K paradigm	✓	Proportion of K responses increased
Hockley and Consoli, 1999 [38] (Exp. 2 only)	2 days and 1 week	Item vs associative memory (R responses only)	✓	Smaller decrease for item memory
Sharot and Yonelinas [39]	Decline in memory after a 24-h interval compared with after a 5-min interval	R/K and item/source memory	Source memory declined with retention interval	K responses were not affected by retention duration
Brown and Halliday, 1991 [40]	Decline in memory after 1 week compared with immediately after study	Item/source memory	✓	Item memory declined to a lesser extent than source memory
Bornstein and Lecompte, 1995 [41]	Decline in memory from immediately after study, half an hour, 48 and 168 h later	Item/source memory	✓	✓
Gardiner, 1988 [43]	Comparison of memory 1 h after study with 1 week after study	R/K paradigm	✓	K responses were less affected than R responses
Gardiner and Java, 1991 [44] (Exp. 1)	Comparison of memory 1 min after study with 1 week after study	R/K paradigm	✓	×

Table 1 (*Continued*)

Studies employing a delay manipulation				
Study	***Delay details***	***Recollection/ familiarity measurement***	***Detrimental effect of delay on recollection measure***	***Detrimental effect of delay on familiarity measure***
Gardiner and Java, 1991 [44] (Exp. 2)	Comparison of memory 1 week after study with 6 months after study	R/K paradigm	✓	✓
Barber et al., 2008 [50]	Comparison of memory immediately after study with 48 h after study	R/K paradigm	✓	Proportion of K responses increased
Knowlton and Squire, 1995 [46]	Comparison of memory 10 min after study with 1 week after study	R/K paradigm	✓	×
Harand et al., 2012 [45]	Comparison of memory 3 days after study with 3 months after study	R/K paradigm	✓	×
Carr et al., 2009 [47]	Comparison of memory 10 min after study with 1 week after study	R/K paradigm	✓	Proportion of K responses increased
Tunney, 2010 [48]	Comparison of memory 10 min after study with 1 week and 2 weeks after study	R/K paradigm and ROC curves	✓ Effect only for the 10 min vs 1 week comparison	Familiarity increased between 10 min and 1 week but remained unchanged between 1 week and 2 weeks
Viskontas et al., 2009 [49]	Comparison of memory 10 min after study with 1 week after study	R/K paradigm and fMRI	✓ In addition, after 1 week hippocampal activity for items that were no longer recollected decreased compared with hippocampal activity of items that were still recollected	Proportion of K responses increased
Petrican et al., 2010 [51]	Decay as a function of time elapsed since the occurrence of a public historical event	R/K paradigm	✓	Familiarity measures decreased with time to a lesser extent than recollection measures
Wais et al., 2006 [52]	Comparison of memory 1 h, 1 day, 1 week, 2 weeks, and 8 weeks after study	ROC curves	✓	Familiarity measures decreased with time to a lesser extent than recollection measures
Wolk et al., 2006 [53]	Comparison of memory 39 min and 24 h after study	R/K paradigm and ERP estimates of recollection and familiarity	✓	×
Stark and Squire, 2000 [55]	Comparison of forgetting of line-figure stimuli over various delays (1 h, 1 day, and 1 week)	fMRI	✓ Reduced right anterior hippocampal activity	Not reported
Talamini and Gorree, 2012 [56]	Comparison of memory decline from 5 min to 3 months (memory tested five times in total)	Memory for an object vs memory for an object's contextual features	✓	Familiarity decayed at a slower rate than recollection

[a]✓, effect present; ×, no effect present.

propose does suggest, however, that items that resemble the target, or reinstate the context, would serve as good retrieval cues for recollection as a result of pattern completion, but may cause interference for memories based on familiarity.

Recollective memories are more resistant to interference than familiarity-based memories

Several studies support the hypothesis that recollective memories are more resistant to interference than familiarity-based memories [17,20] (Table 1). Examining memory for items versus paired associates is an established means of teasing apart the effects of familiarity and recollection [21]. In associative memory paradigms, both items of a pair previously presented at study are paired at test either with each other or with other studied items. Thus, whereas item memory may rely on familiarity, the correct association of two items can presumably be mediated only by recollection of the study episode in which both

items appeared together. (An exception to this notion is cases in which the items in a pair are unitized to one concept. In such cases, item-pair memory relies strongly on familiarity [22] and is thus prone to interference [23].)

Hockley [17] compared forgetting of single words and paired associates using a continuous-recognition paradigm where study and test materials were intermixed such that study items reappeared as test probes after a lag of several intervening items. This paradigm manipulates the degree of interference by varying the number of intervening items. Whereas associative memory remained stable across varying lags [17,20], item memory deteriorated as the lag increased. These results were replicated in an additional interference paradigm where multiple study–test sessions were presented, followed by a final recognition test of items and item pairs from all lists [17].

An effect of interference was found in familiarity but not in recollection when comparing item memory and source memory (i.e., 'was the word presented in a red or green font?'), with the former indexing familiarity and the latter recollection [24]. Converging results were found when interference was operationalized using a proactive interference task [25,26]. There, proactive interference affected estimates of familiarity, but not of recollection (these estimates were derived using methods such as the Process Dissociation Procedure [PDP]). Using the AB/AC recall paradigm [27–29] as a mean of employing proactive interference (PI), Winocur *et al.* [28] demonstrated that difficulties in learning AC word pairs are observed when participants are induced (via an implicit memory test) to rely more heavily on familiarity than recollection.

When single items are studied, recollection and familiarity can be indexed by the Remember/Know (R/K) procedure, in which individuals judge whether they can recollect the context in which the items were presented (R) or whether the items are merely familiar or known (K). Cary and Reder [30] used this paradigm to investigate whether manipulating the length of the study list affects recollection and familiarity differentially. They found that corrected recognition decreased as list length increased. More importantly, false alarms judged as familiar increased with list length – a finding that speaks to the detrimental effects of interference on familiarity.

A caveat of some studies employing interference is that the interference stage necessarily includes a time element, which, as we show in the next section, affects recollection more than familiarity. A concern regarding our hypothesis, therefore, is that the effects of interference and delay may be confounded. This concern is addressed, and countered, when we review findings from patient populations. Briefly, in studies examining a certain population of patients, memory performance after an interfering task is compared with performance after a delay in which there is no interference but only decay. Critically, in these studies, the duration of interference and decay are equated and, therefore, the possible effect of interference, beyond that of delay, can be measured. In other patient studies, interference and decay are not confounded because only interference is manipulated and compared among two populations of patients – one presumably more sensitive to interference than the other.

The converging findings of a condition that detrimentally affects familiarity more than recollection is all the more telling because, to date, the opposite effects have typically been reported. This pertains to experimentally induced conditions, such as divided attention, and to naturally occurring conditions, such as memory loss due to brain damage or degeneration [2,31] (but see [32,33] for exceptions).

Recollective memories decay more than familiarity-based memories over time

In contrast to the prevailing notion that forgetting occurs primarily due to interference [34–36], it has been suggested that memories relying on the hippocampus decay over time due to weakening of synaptic potentiation [1] and/or ongoing neurogenesis [9]. The plausibility of this idea is supported by neurobiological findings regarding the molecular mechanisms of long-term memory maintenance in the hippocampus [1,9]. Extrapolating from these results, we speculate that recollective memories, although relatively resistant to interference, may be forgotten primarily due to decay of hippocampal traces. By contrast, because familiarity-based memories do not rely on the hippocampus but on other MTL structures that may not share the same molecular properties promoting decay [10,11], they may be less prone to decay over time than recollection-based memories. This jibes with our experiences, as we often recognize an individual face we have not seen for a while as familiar, without recollecting the context pertaining to that person. Do the empirical data support this intuition?

Indeed, a substantial number of studies have demonstrated that, compared with familiarity, recollection is more affected by the passage of time, usually days to weeks (Table 1). Over delays of 1 day to 1 week, associative memory (e.g., memory for face–scene pairs [37]) declines to a larger extent than item memory [37,38]. Similarly, source memory appears to decrease over a delay of 1 day [39] and 1 week, whereas item memory decreases less [40] (but see [41]).

Additional evidence comes from studies in which recollection and familiarity were indexed by the R/K paradigm [42]. In several studies, R/K judgments were collected in at least two testing sessions – the first soon after study and the other between 1 day and 3 months later. These studies found that, although the proportion of R responses greatly decreased over these durations, the proportion of K responses did not decrease as much [43], remained unchanged [39,44–46], or even increased [47–50]. (It should be noted, however, that a caveat of some of these studies was that the same set of words was tested, and given R and K judgments, in the early and later test sessions. It is likely, therefore – and often reported – that the change in R and K proportions with time was due in part to the fact that some of the R judgments transformed into K judgments.) Notably, at longer retention intervals (4–6 months [44] and 2 weeks [48]), R and K responses no longer differed in their forgetting rates. This raises the question of whether memories that persist beyond a certain time frame are no longer sensitive to decay. (Box 3). A similar pattern was also reported when R/K decisions were given regarding memory

Box 3. Outstanding questions

- Is there a particular time frame (i.e., perhaps shortly after learning) during which familiarity-based memories are especially sensitive to interference? Does recollection also decay quickly if the comparison is with tests that occur immediately after encoding an item? What is the major cause of interference affecting familiarity? Do proactive and retroactive interference have different effects in magnitude and/or nature? Perhaps proactive interference affects mostly the encoding process and retroactive interference the consolidation process.
- Does immediate post-learning interference impair consolidation even for recollection?
- What are the parameters that render a task interfering? Is the similarity of the materials to the study items the most important factor? Which matters more, the similarity in the nature of the stimuli or in the type of response? Can interfering materials decrease familiarity even if they are not attended?
- Is there a particular time frame during which the decay of recollection generally occurs? Do memories that persist beyond this time frame never fade?
- Does the decay in recollection entail a conversion to reliance on familiarity or complete forgetting?
- Do certain aspects of contextual information (e.g., spatial information, temporal information) decay faster than others?
- Recent evidence suggests that memory consolidation is enhanced when learning is followed by a period of rest or sleep (see the supplementary data online). In addition, sleep promotes integration of episodic memories into semantic-memory networks. Taken together, do these findings conform with our ideas that, during the delay introduced by rest or sleep, recollection decays as recollective memories transform to familiarity-based semantic memories?
- Is the possible dissociation between forgetting patterns of recollection and familiarity confined to recognition memory or does it also extend to recall?
- Are forgetting rates due to interference associated with decreases in perirhinal activation and those with decay with decreases in hippocampal activation?
- What patterns of forgetting will be observed in patients with semantic dementia whose disorder is characterized by early atrophy to the anterior temporal lobe, including the PRc?
- Will it be possible to show, using multivoxel pattern analysis (MVPA), that interfering stimuli are still distinguishable from one another in the hippocampus, but not in extrahippocampal structures?

of public historical events [51]. Decay as a function of the time elapsed since the public event had occurred was found to be significantly more prominent for recollection than for familiarity.

An additional method often used to estimate measures of recollection and familiarity is Receiver Operating Characteristic (ROC) curves. Wais *et al.* [52] used this method to compare recollection and familiarity estimates at five intervals following study: 1 h; 1 day; 1 week; 2 weeks; and 8 weeks. In line with our hypothesis, they found that recollection estimates decrease with time to a greater degree than familiarity estimates.

Using event-related potentials (ERPs), Wolk *et al.* [53] found that the ERP familiarity signal (i.e., the mid-frontal positivity [54] associated with 'know' responses to old items) did not differ between the short and long delays. However, the ERP recollective signal (i.e., the late left-parietal positivity [54] associated with 'remember' responses) declined in the long delay. These results, however, should be viewed with caution, because the ERP signals for familiarity and recollection for correct rejections (i.e., baseline) also changed across delays. Evidence from functional MRI (fMRI) studies of young individuals also support the notion that recollection is particularly sensitive to decay [49,55]. These studies found decreased recollection-related activation in the hippocampus as a function of delay (see also the supplementary data online).

A final piece of evidence comes from a study by Talamini and Gorree [56] in which memory for the context in which an object was presented (presumably indexing recollection) was compared with memory only for the object itself (presumably indexing familiarity). Recollection was found to decay faster than familiarity over 3 months and especially during the first week.

It is noteworthy that the studies reviewed in this section typically employed retention intervals of days or weeks, whereas the interference studies reviewed in the previous section included retention intervals of minutes or even seconds. This supports the idea mentioned in the previous section that interference and decay may exert their effects on different timescales. Indeed, according to the neurobiological model of decay due to neurogenesis previously mentioned (see 'A neuroanatomical basis for resistance and proneness to interference'), the forgetting process occurs over a timescale of several weeks [9]. Still, another possibility is that the distinct forgetting patterns of recollection and familiarity may not pertain to the differential effects of interference and decay, but to differences in the timescales of the retention intervals. However, this possibility does not seem likely considering findings from patient populations in which the timescales are equated when comparing the effects of interference and decay.

Increased interference following damage to the hippocampus

Individuals with memory disorders due to hippocampal damage or atrophy exhibit profoundly impaired recollection [57,58]. (Some of the studies cited below examined patients whose lesions are primarily, or exclusively, in the hippocampus, whereas in others the lesion probably also includes extrahippocampal MTL regions. Here we discuss both classes of lesions collectively because both should detrimentally affect interference, as long as the hippocampus is affected.) It follows that the mnemonic performance of individuals with hippocampal lesions should be more compromised by interference, because they can rely less on hippocampally mediated recollection to distinguish between relevant and interfering memories. Indeed, it has been found that disrupting hippocampal function by surgical lesions [59] or preventing hippocampal neurogenesis through irradiation [8] renders rodents more susceptible to interference.

Studies have shown that when encoding is followed by an interfering task, memory-impaired patients with hippocampal lesions exhibit more profound forgetting than patients with frontal-lobe lesions [60] (but see [61,62] for proactive interference effects) and Korsakoff patients [63], both of whom often have less hippocampal damage or none at all. By contrast, when the study–test interval was not filled with interfering activity, no differences between forgetting rates of diencephalic and MTL amnesia patients was found [64] (but see [65]). Additional evidence comes

from a study [28] in which interference was manipulated using the AB/AC paradigm. Patients with lesions that included the hippocampus had abnormal difficulty learning AC word pairs after learning AB word pairs, whereas patients with frontal lesions did not.

Perhaps the most compelling evidence regarding interference in amnesia comes from studies that compared mnemonic performance when the study–test delay included processing of potentially interfering materials with when it did not [66–68] (for a review, see [1]). Significantly more forgetting was found when encoding was followed by interference rather than by rest. Similar results were found in a study that compared memory performance after rest with performance after a delay in which interfering materials were presented, although they did not resemble the target [69]. It may be that patients' susceptibility to interference is so pronounced that even dissimilar materials can interfere with learning, perhaps by disrupting consolidation [1,69]. It could also be that, because the task introduced during the study–test interval was engaging enough to distract participants, it prevented rehearsal or continuous processing of the target materials. One caveat with regard to this set of studies is that inclusion of participants was made based on behavioral measures of memory, so that some participants with memory loss may not have had hippocampal lesions.

A study examining accelerated long-term forgetting in patients with temporal-lobe epileptic foci [70] found that abnormal forgetting rates after 24 h, which are typical of such patients, became equivalent to those of controls when encoding was followed by a 12-h sleep. Here, too, one could argue that even the minimal interference caused by engaging in daily activities is reduced by sleep in patients with lesions that include the hippocampus. In healthy individuals whose hippocampi are intact, sleep can lead to loss of recollection accompanied by transformation of the memories to those that are familiarity based (see the supplementary data online).

Although rare, studies of patients with selective hippocampal lesions may provide particularly strong evidence for the role of the hippocampus in resisting interference. Indeed, it has been reported [71] that selective hippocampal damage impairs yes/no recognition but only when target and lures were very similar, hence interfering with one another. By comparison, forced-choice recognition, which depends more on familiarity, was not compromised. The authors' interpretation of this pattern, which we endorse, is that in such cases familiarity can support recognition in forced-choice but not in yes/no recognition. For forced-choice recognition, familiarity decisions are based on a comparison between targets and relatively similar lures. The relative familiarity of the targets is higher than that of lures, so decisions based on familiarity can be made using a relatively high criterion for distinguishing between targets and lures. However, for yes/no recognition, each recognition trial involves a comparison of the presented stimulus relative to all other stimuli presented at test. When targets and lures are similar, the familiarity distribution of targets and lures may overlap highly, whereas the distribution of targets' familiarity may not overlap as much. In this case, a high-familiarity criterion would lead to many misses, whereas a low criterion would lead to many false alarms. Therefore, recollection is needed to differentiate between the highly similar targets and lures in yes/no recognition.

More rapid decay of recollection-based memories in patients with MTL lesions

The notion that recollection is particularly sensitive to decay over time gains support from patient studies. Accelerated long-term forgetting patterns of patients with temporal-lobe epilepsy were found to be different from those of patients with epileptic foci outside the temporal lobes [72]. Namely, although both groups of patients forget lists of words more than controls after 1 day and 1 week, the patterns of forgetting of the two patient groups are different. Whereas most of the forgetting of the temporal-lobe epilepsy group, whose recollection is likely to be impaired, occurs after 1 day, the other patient group, who presumably relies more on recollection, shows more gradual forgetting, with most of the forgetting occurring after 1 week. This study demonstrates that decay over relatively long durations is a more prominent cause of forgetting among patients with intact MTLs, whereas more rapid forgetting is a hallmark of MTL damage.

When performance relies predominantly on familiarity, there should not be much of an effect of decay, because performance will be mediated by structures that are not particularly prone to decay (i.e., extrahippocampal structures, probably the PRc for words and objects [13] and perhaps the parahippocampal cortex for scenes [73]). Such is the case with patient Y.R. who has selective hippocampal damage [74]. Y.R.'s memory performance was tested in numerous tests that differed substantially in the study–test interval, at delays ranging from 0 s to 30 days. Performance in these tests could be guided by familiarity, which indeed was not impaired. Strikingly, when examining *z*-scores reflecting Y.R.'s performance compared with that of controls, no effect of delay was found. Note that no effect for list length was found either. However, the failure to find this effect may be due to limited power of the list-length analysis compared with the delay analysis.

The question concerning the effects of decay on memory of patients with lesions that include not only the hippocampus but other regions of the MTL is more difficult to assess. This issue was debated vigorously in the 1970s and 1980s, without being resolved to everyone's satisfaction. It may be worthwhile to revisit some of the major experiments and view them in the context of this paper's discussion. A long-held belief is that medial temporal damage is accompanied by rapid forgetting. However, when care is taken to ensure that patients' memory is equated with that of controls at encoding by testing their memory immediately after learning, rapid forgetting occurs under some circumstances but not others. To anticipate, it appears that decay is faster when memory relies more on recollection than familiarity and furthermore that the rate of decay may depend on the extent of hippocampal damage.

Using visually complex scenes as stimuli and equating performance between amnesic patient H.M. and controls at encoding, it was found that H.M.'s memory was equivalent to that of controls on tests of forced-choice recognition, at

delays of 1, 3, or 7 days [75]. On yes/no recognition, however, his performance was impaired at 3 days. If one considers that forced-choice recognition can rely more on familiarity compared with yes/no recognition, the results are consistent with our hypothesis and suggest that hippocampal damage may increase susceptibility to decay and lead to more rapid forgetting when the stimuli or tests are more dependent on recollection (but see [76]). Consistent with this interpretation are findings showing more rapid forgetting in patients with MTL lesions on tests of recall when performance is equated for recognition [77]. Likewise, using an object-location test that is sensitive to hippocampal damage, more rapid forgetting over a 4-min delay was found when performance was equated at no delay [78]. Interestingly, interpolating a hippocampally sensitive verbal (Hebb digits) or spatial (Corsi blocks) interference task did not exacerbate the deficit, but a similar spatial-location task did [61], again consistent with our hypothesis. The results of this study suggest, furthermore, that the effects of decay may be related not only to the type of memory (recollective) but to the extent of hippocampal damage. It may also be the case that having intact extrahippocampal structures may delay decay for the component of the test that may draw a little on familiarity. Similar patterns of results were obtained with recognition of faces [79] and sentences [60].

It is unfortunate that comparable data are not available for patients with damage restricted to the extrahippocampal structures in the MTL, where we would expect that decay would be normal for tests relying on recollection but perhaps impaired for tests relying on familiarity.

Finally, our hypotheses entail that patients with lesions that encompass the hippocampus as well as the PRc should be particularly sensitive to interference as well as decay. However, such data would not be informative in adjudicating between the specific forgetting patterns of the memory representations supported by each of these two structures. Instead, more experimental evidence for dissociations between the forgetting patterns of patients with hippocampal lesions and patients with PRc lesions is needed.

Concluding remarks

The study of forgetting has a long tradition in memory research dating to the early work of Ebbinghaus [80]. Throughout the years, scholars have sought to identify the causes of forgetting, seesawing between decay and interference [36]. These two causes have often being treated collectively and perhaps even confounded with each other. Therefore, our suggestion that both decay and interference can exert their effects, depending on the memory representation, may place the century-old forgetting research in a new light. (Box 3). If this hypothesis is correct, it would have implications for deriving forgetting curves that would differ depending on the type of representations in memory. This, in turn, may augment many computational models of forgetting that currently apply the same principles to all aspects of forgetting related to episodic memory without regard to its quality [81,82]. Moreover, the theoretical framework we propose could provide a better understanding of forgetting among populations with memory disorders who rely more on familiarity than on recollection. Research on motivated forgetting, conducted in the context of traumatic memories, may also benefit from this framework (Box 4). Finally, it also may help explain why elements of memories that are hippocampally dependent can recombine with elements of other memories to produce new memory combinations without leading to forgetting of the original. This resistance to interference may be one of the major factors that contribute to the flexibility of hippocampally based memories.

Box 4. Motivated forgetting

An aspect of forgetting that was not discussed in this review, which we refer to collectively as motivated forgetting [100], regards paradigms in which forgetting is intentionally induced. In one such paradigm, known as directed forgetting, participants are directly instructed to remember some of the items and forget others, with the former being remembered better than the latter [100–102]. In another paradigm, retrieval-induced forgetting, forgetting is induced by retrieval of some of the items, but not of others [100,103,104]. In a typical retrieval-induced forgetting paradigm, items from certain semantic categories are studied and subsequently some of the items of each category are tested in what is often referred to as a practice stage. In the final test, memory is enhanced for the practiced items and reduced for the unpracticed items from the same semantic category.

Evidence from motivated forgetting studies suggests that these manipulations exert their effects on recollection more so than on familiarity [101,102,104–108] (but see [103]). This pattern has been obtained using various measures of recollection and familiarity, including the R/K paradigm and source memory.

Although motivated forgetting appears orthogonal to the type of passive forgetting discussed in this Opinion, it may be that the above findings can be accommodated within the framework we propose. To do so, we capitalize on the notion that inhibition plays a prominent role in motivated forgetting [109]. According to one inhibition account for directed forgetting, whereas the to-be-remembered items are rehearsed, encoding of the to-be-forgotten items is stopped by blocking processing of those items [110]. For retrieval-induced forgetting, retrieving some of the items during the practice stage causes strengthening of their memory traces and at the same time inhibition of semantically related, interfering items that appeared at encoding [109]. Inhibition thus refers to reduction in the strength of competing, interfering stimuli. The larger effects of motivated forgetting on recollection than on familiarity may be attributed to more efficient inhibition of interfering memory traces. When relying on recollection, one may be better able to separate target and interfering items, thereby strengthening the former while inhibiting the latter. The notion that recollective memory traces are effectively separated from one another may be accounted for by the fact that recollection is supported by the hippocampus, which specializes in pattern separation.

Note that an additional aspect of forgetting that we did not discuss is false memory, in paradigms such as the DRM. Generally, in such studies critical lures are more likely to be falsely recognized and judged as recollected than familiar [111]. This may seem to imply that interference from semantically similar information affects recollection more than familiarity. However, a more plausible interpretation is that false memory for lures is not a form of forgetting, but rather of encoding additional semantically related materials. Namely, it is not the case that critical lures are interfering with other memories, but rather that they are encoded in addition to them.

Acknowledgments

This work was supported by National Science Research Council of Canada Grant A8347 to M.M. J.D.O. was supported by a Postdoctoral Fellowship award from the Natural Sciences and Engineering Research Council of Canada. The authors thank Chen Didi-Barnea, Yonatan Goshen-Gottstein, and Stefan Köhler for insightful discussions.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.tics.2013.10.008.

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