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What is This?

A Theory About Why We Forget What We Once Knew

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ABSTRACT—*Traditional theories of forgetting assume that everyday forgetting is a cue-overload phenomenon, and the primary laboratory method used for investigating that phenomenon has long been the A-B, A-C paired-associates procedure. A great deal of research in psychology, psychopharmacology, and neuroscience suggests that this approach to the study of forgetting may not be very relevant to the kind of interference that induces most forgetting in everyday life. An alternative interference theory holds that recently formed memories that have not yet had a chance to consolidate are vulnerable to the interfering force of mental activity and memory formation, even if the interfering activity does not involve material similar to what was previously learned. This account helps to explain why sleep, alcohol, and benzodiazepines all forestall forgetting of a recently learned list, and it is consistent with recent work on the variables that affect the induction and maintenance of long-term potentiation in the hippocampus.*

KEYWORDS—*forgetting; interference; consolidation; retrograde facilitation; long-term potentiation (LTP)*

In common parlance, the verb "to forget" refers to the inability to remember a prior event whether or not that event was ever committed to memory. Thus, for example, I might claim to forget where I placed my keys, but the truth may be that I neglected to encode the relevant information in the first place. People with amnesia resulting from damage to the brain's medial temporal lobes typically experience this kind of forgetting. That is, they fail to encode new information, so they seem to forget everything quickly.

In the field of psychology, theories of forgetting are typically concerned with the loss of information that was once successfully encoded. Thus, although you once remembered the name

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of your first-grade teacher, thereby proving that the information was encoded, you may now discover that the name has slipped away (forever, perhaps). Why does that happen? Traditionally, the answer has been that forgetting occurs either because memories naturally decay or because they succumb to the forces of interference. The story that most students of psychology learn is that a classic sleep study conducted by Jenkins and Dallenbach (1924) tilted the balance in favor of interference theory. In that study, subjects remembered more of a previously learned list when they slept before taking the memory test than when they remained awake. Because interference is much less likely to be encountered while sleeping, whereas a natural decay process should unfold whether one is asleep or awake, the results pointed to a dominant role for interference. In a recent review of a century of work on forgetting, I argued that the field accurately interpreted Jenkins and Dallenbach's study (i.e., the results suggest a role for interference) but then modeled the interference process in a way that is not likely to be very relevant to ordinary forgetting (Wixted, 2004).

In the early part of the 20th century, two views of interference struggled for supremacy. According to one view, interference consists of new memories degrading previously established memory traces that have not yet had a chance to consolidate. For such interference to occur, the new memories do not need to be especially similar to the ones they impair (Skaggs, 1925). The formation of new memories, per se, is the interfering force. According to the other view, interference consists of what later came to be called a *cue-overload* effect (Robinson, 1920; Watkins & Watkins, 1975). Cue overload occurs when more than one memory is associated with the same retrieval cue. Thus, for example, if I memorize "Learned Hand" as the name of a famous judge, the retrieval cue "famous judge" will become ever less effective in retrieving that name as I memorize the names of more famous judges. From this point of view, similarity of the interfering material is critical because similar items tend to be subserved by the same retrieval cue (whereas dissimilar items tend not to be). Thus, instead of interference being viewed as the degradation of a memory trace, it is viewed as a competition phenomenon that occurs at the time of retrieval.

The study of cue-overload interference is exemplified by the well-known A-B, A-C paired-associates design. In this procedure, the experimental group of subjects learns an A-B list of cue-target word pairs (e.g., *hero-prison*, *water-valley*, *tiger-image*, etc.) followed by a second, A-C, list involving the same cue words but different target words (e.g., *hero-women*, *water-salad*, *tiger-infant*, etc.). A control group learns the A-B list followed by a C-D list, which involves a completely different set of words. Both groups are then tested by giving the A cues and asking for recall of the B targets. The typical finding is that the experimental group performs more poorly than the control group, and this implies that the more items are attached to the A cue, the less likely that cue will be to occasion the retrieval of any one of its associates. Because paired-associates procedures generate powerful effects in the laboratory, the cue-overload view of forgetting came to dominate the trace-interference account during the latter half of the 20th century.

Specific theories of cue-overload interference died an inglorious death in the late 1960s, in part because concepts such as *unlearning* (the idea that forgetting is caused by the unlearning of previously learned associations) and *spontaneous recovery* (the idea that unlearned associations spontaneously reestablish themselves) failed to find much empirical support (Tulving & Madigan, 1970). Still, the general notion that interference is mainly due to cue overload at the time of retrieval lived on, despite the fact that multiple efforts to demonstrate that cue-overload effects apply to everyday forgetting (not just laboratory forgetting) generally suggested just the opposite (Underwood & Ekstrand, 1967). In fact, the only reason to believe that cue-overload effects play any role at all in everyday forgetting is that almost everyone can point to a few examples from their own lives where it surely does. But the mere fact that cue-overload effects sometimes play a role does not mean that the role is a substantial one.

Summarizing the state of the art late in his career, Underwood (1983) said: "A relatively few years ago it seemed that a fairly comprehensive theoretical account of forgetting was close at hand, but that has slipped away. Some investigators have lost confidence in interference as a major cause of forgetting, but none of the proposed replacements thus far has created a feeling that things are on a productive new track. But that will surely come" (p. 262).

In search of a productive new track, I recently reviewed a century of research on forgetting and concluded that an idea long ago abandoned by the field—namely, that everyday forgetting is largely the result of trace interference—was ahead of its time (Wixted, 2004). The evidence bearing on that claim derives from the fields of psychology, psychopharmacology, and neuroscience, and I briefly review that evidence next.

PSYCHOLOGY: THE TEMPORAL GRADIENT OF RETROACTIVE INTERFERENCE

An idea that the field of psychology has never fully embraced (even though it has become the "standard story" in the field of neuroscience) holds that memories consolidate for a period of time after they are formed. During the consolidation period, memories are especially vulnerable to disruption. Perhaps the most compelling piece of evidence in favor of consolidation theory is the temporal gradient of retrograde amnesia (Ribot, 1882). The temporal gradient is observed when a brain structure known as the hippocampus is damaged. Bilateral hippocampal damage induces anterograde amnesia (which is the inability to form new memories), but it also impairs, to some degree, previously formed memories, with memories formed just prior to brain injury being more impaired than memories that were formed longer ago (i.e., retrograde amnesia is temporally graded). Older memories are assumed to be relatively spared because they are more completely consolidated than newer memories (and, therefore, are less dependent on the hippocampus).

If memories do consolidate over time, and if the formation of new memories does interfere with previously formed memories, then one should also expect to see a temporal gradient of *retroactive interference*. That is, the encoding of new information should interfere more with recently formed memories than with older memories. This issue was first addressed by Müller and Pilzecker (1900), and they found clear evidence for the predicted temporal gradient (i.e., retroactive interference was more pronounced if the interfering material occurred early rather than late in the retention interval before the memory test). However, most other studies did not. In fact, all reviews of the relevant literature conducted after 1930 concluded that there is little or no evidence for the predicted temporal gradient of retroactive interference. To many psychologists, this meant that the process of consolidation, even if it is real, is a physiological process that has little to do with the psychological processes that are responsible for forgetting (such as unlearning and spontaneous recovery). Such findings may help to explain why the field of psychology has never really embraced the notion of memory consolidation.

I have argued that this interpretation of the literature is inaccurate and that it results, in part, from a failure to distinguish between interference based on trace degradation (a storage phenomenon) and interference based on cue overload (a retrieval phenomenon; Wixted, 2004). No consolidation theory predicts that the retrieval competition induced by cue overload will be more pronounced if the retrieval cue is overloaded early in the retention interval. Even so, manipulating the timing of cue overload was the preferred method of investigation. The few studies that varied the temporal point of memory formation itself against a background of mental quietude (e.g., Skaggs, 1925), which are the kinds of studies that speak more directly to the issue, found evidence for the temporal gradient that is predicted by consolidation theory. In light of such evidence, the long-neglected notion that memories consolidate may be an important part of the story of why we forget.

PSYCHOPHARMACOLOGY: RETROGRADE FACILITATION

Alcohol and benzodiazepines (e.g., Valium, Xanax) both induce temporary anterograde amnesia. Perhaps the best-known example is the alcoholic "blackout," which refers to the individual's complete amnesia for events that occurred while he or she was intoxicated. Also well established, but less well known in the field of psychology, is that these drugs (like sleep) result in retrograde *facilitation*. That is, material learned just prior to taking one of these drugs is, at a later point, remembered better than it otherwise would have been (i.e., less forgetting will have taken place). An obvious explanation for this interesting phenomenon is that the reduced rate of memory formation while one is under the influence of the drug protects recently formed memories during a period of time when they are especially vulnerable. This explanation is sometimes rejected in the psychopharmacology literature because retrograde facilitation is observed in laboratory studies even when no explicit interfering task is arranged for either the subjects who are administered the drug or the control subjects who receive a placebo. According to this way of thinking, if a subject memorizes a list of words and then drinks alcohol, a reduction in interference (relative to a control group) ought to be observed only if a similar interfering task is subsequently arranged. But memory for the list is enhanced even when an interfering task is not presented, and some researchers have taken this to mean that the drug directly enhances the consolidation process.

The idea that an interference effect should be observed only if an explicit (and similar) interfering task is arranged makes sense from the point of view of traditional interference theory. In fact, Underwood (1957) made this very point when he observed that the sleeping subjects in Jenkins and Dallenbach's (1924) classic sleep study did not avoid learning material that was similar to the study list, so avoidance of retroactive interference could not explain why sleep facilitates memory. To Underwood, this reasoning implied that interference must have been proactive in nature (i.e., arising from prior similar learning) and that sleep somehow held the forces of proactive interference at bay. Were he alive today, he might advance the same account of alcohol-induced retrograde facilitation. However, in the absence of any compelling evidence, this unnatural theory seems hard to accept.

An alternative possibility is that alcohol and benzodiazepines directly enhance the consolidation process, but this notion is hard to accept as well. Why would a drug that facilitates the consolidation process induce anterograde amnesia? Facilitating the consolidation process should result in anterograde and retrograde facilitation, not anterograde amnesia and retrograde facilitation. A more likely possibility is that these drugs minimize memory formation in general and that, in so doing, they protect recently formed (and, therefore, fragile) memories from the deleterious forces of nonspecific retroactive interference that arise from the process of memory formation itself.

NEUROSCIENCE: A POSSIBLE NEURAL MECHANISM

What is the source of retrograde facilitation? One possibility is that drugs like alcohol and benzodiazepines close the hippocampus to new input, thereby inducing anterograde amnesia without compromising the ability of the hippocampus to consolidate previously formed memories. Because new input is prevented, recently formed (and, therefore, incompletely consolidated) memories are protected from the retroactive interference that they would otherwise encounter. Thus, these drugs may act in the same way that sleep does even though the individual remains conscious. By contrast, hippocampal lesions both prevent new input (resulting in anterograde amnesia) and terminate the hippocampal-dependent consolidation of recently formed memories (resulting in retrograde amnesia as well).

The prevailing view of how memories are initially encoded is that the process involves a change in the probability that postsynaptic neurons in the hippocampus will fire in response to neurotransmitters released from presynaptic neurons. That is, neurons are in a chain of communication, and when a neuron that is earlier in the chain (the presynaptic neuron) releases a substance known as a neurotransmitter, it causes the next neuron in the chain (the postsynaptic neuron) to fire. Memories of an experience may be initially encoded in the brain when that experience causes certain postsynaptic neurons to become more reactive to neurotransmitters released by presynaptic neurons. The laboratory analogue of this theoretical memory mechanism is long-term potentiation (LTP), which is a durable increase in synaptic transmission in response to high-frequency stimulation of presynaptic neurons. This artificially induced increase in synaptic efficacy typically lasts only a few days or weeks, so it presumably does not represent the way in which memories are permanently coded. Still, in their fragile, preconsolidation state, memories may exist by virtue of an LTP-like process taking place in the hippocampus.

Alcohol and benzodiazepines, which interfere with the encoding of new information, both block the induction of LTP in the hippocampus (Givens & McMahon, 1995). However, alcohol does not impair the maintenance of hippocampal LTP induced an hour prior to administration of the drug (Givens & McMahon, 1995). Blocking the induction of LTP without impairing the maintenance of previously established LTP parallels the effects of alcohol on memory. That is, alcohol blocks memory formation without impairing previously formed memories.

Several recent studies show that the induction of LTP in the hippocampus (like the formation of a new memory) serves as an interfering event. Thus, the induction of hippocampal LTP causes partial forgetting of a previously learned task (Brun, Ytterbø, Morris, Moser, & Moser, 2001). It also impairs the maintenance of previously established LTP (Villarreal, Do, Haddad, & Derrick, 2001), which may be the very reason why it induces forgetting of prior learning. Findings like these suggest that the hippocampus may not be an unlimited resource system

when it comes to the induction and maintenance of LTP. If a recently formed memory relies on an LTP-like process that requires biophysical resources to be maintained, then it would be impaired by the subsequent formation of memories that preferentially draw on the same resources. Only after a memory is no longer dependent on the maintenance of an LTP-like process in the hippocampus (i.e., only after it has consolidated) will it be impervious to the interfering effects of retroactive interference.

CONCLUSION

To summarize, my reading of the literature is that forgetting is largely a function of nonspecific retroactive interference acting on memory traces that have not yet had a chance to consolidate. In the early stages of consolidation, memory traces are theoretically maintained by an LTP-like process in the hippocampus. However, the maintenance of these traces is vulnerable to the subsequent induction of LTP-like processes associated with the formation of new memories.

Although it is now possible to tell a story of forgetting that integrates findings from psychology, psychopharmacology, and neuroscience, every part of that story needs to be solidified by further research. In psychology, some very old evidence supports the idea that a temporal gradient of retroactive interference is observed when memories are formed against a background of mental quietude, but that work is far from definitive. Similarly, in psychopharmacology, we know that alcohol and benzodiazepines induce retrograde facilitation, but we do not know if a temporal gradient exists. Thus, for example, memory tested 24 hr after learning should be enhanced to a greater extent if alcohol is consumed immediately after learning than if it is consumed 8 hr after learning. And in neuroscience, a new question concerns whether it is newly formed memories or recently activated memories that undergo a process of consolidation. Further inquiry into these matters is sure to provide a more complete answer to the question of why we forget what we once knew.

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