LETTER

Forgetting in the no-think paradigm: Interference or inhibition?

When participants, after learning a set of paired associates (e.g., "plane–doctor"), are repeatedly asked to suppress the target ("doctor") upon presentation of its cue ("plane"), such suppression trials can cause later forgetting of the target information. Whereas the original account attributed such forgetting to inhibition of the suppressed memory (1), Tomlinson et al. (2) suggest that forgetting may arise as a result of interference at test. Tomlinson et al. argue that, although in opposition to task instructions, participants occasionally may sample the target during no-think training and then learn to associate this memory with the no-think response (i.e., "sitting quietly"). Later, at test, the newly learned association interferes with recovery of the target information, causing the observed forgetting.

Tomlinson et al.'s account is in the tradition of global computational memory models, which assume that recall of memory traces occurs by means of a two-stage process, which includes initial sampling and a subsequent recovery stage. Although to date such memory models have placed interference effects alone at the sampling stage of recall, Tomlinson et al. allow interference to affect the recovery process as well. Assuming that the sampled memory of a target is an incomplete representation (e.g., "d_ct_r"), they suggest that, during no-think trials, participants learn to associate the sampled target memory with the no-think response, which then at test compete for conscious recall. Doing so, Tomlinson et al. generalize existing global memory models and provide an elegant, noninhibitory account of forgetting over a wide range of recall conditions.

A crucial assumption of Tomlinson et al.'s interference account is that new associations are built up during no-think trials, suggesting an increase in memory-related activity over repeated suppression trials. This suggestion contrasts with findings from neurocognitive studies reporting evidence for a down-regulation of memory-related neural activity during no-think trials (3, 4). When no-think trials are compared with a no-recall fixation baseline (3), fMRI results show that suppression trials lead to decreases in hippocampal activity. Similarly, a recent event-related potentials study (4) reported a reduction in cued-recall positivity with increasing no-think trial repetitions that predicted the amount of no-think forgetting. Remarkably, memory-related neural activity was already down-regulated in anticipation of a suppression trial, suggesting a role of anticipation for no-think forgetting (5).

When different measures of neural activity (fMRI, EEG) are used, the results from the two neurocognitive studies converge on the view that retrieval success is voluntarily down-regulated during suppression trials. These results support the inhibition view of no-think forgetting but appear incompatible with the suggested interference account, according to which memory-related activities during suppression trials should increase rather than decrease.

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Author contributions: K.-H.T.B. and S.H. wrote the paper.

The authors declare no conflict of interest.

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