

## The neural substrates of recollection and familiarity

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**Abstract:** Aggleton & Brown argue that a hippocampal-anterior thalamic system supports the “recollection” of contextual information about previous events, and that a separate perirhinal-medial dorsal thalamic system supports detection of stimulus “familiarity.” Although there is a growing body of human literature that is in agreement with these claims, when recollection and familiarity have been examined in amnesics using the process dissociation or the remember/know procedures, the results do not seem to provide consistent support. We reexamine these studies and describe the results of an additional experiment using a receiver operating characteristic (ROC) technique. The results of the reanalysis and the ROC experiment are consistent with Aggleton & Brown’s proposal. Patients with damage to both regions exhibit severe deficits in recollection and smaller, but consistent, deficits in familiarity.

Aggleton & Brown (A&B) argue that a hippocampal-anterior thalamic system supports the “recollection” of contextual information about previous events, and that a separate perirhinal-medial dorsal thalamic system supports detection of stimulus “familiarity.” There is a growing body of human literature that is consistent with these claims, showing that recognition memory judgments reflect the separate contributions of recollection and familiarity processes. These two processes are functionally independent (e.g., Atkinson & Juola 1974; Jacoby 1991; Mandler 1980) and they exhibit separate electrophysiological correlates (e.g., Düzel et al. 1997), suggesting that they reflect partially distinct cortical generators. Some studies of human amnesia support A&B’s proposed mapping of these processes to areas within the medial temporal lobes. Other recent reports appear to be in conflict, however. Re-examining the human literature on recollection and familiarity in amnesia finds that the current evidence is, in fact, quite consistent with their proposal.

According to A&B’s proposal, patients with damage to both the hippocampal and perirhinal systems – which includes most of the human amnesic patients that have been studied – should exhibit deficits in both recollection and familiarity. Direct tests of recognition memory provide some support for this prediction in showing that amnesics typically exhibit recognition memory deficits. However, because recognition memory judgments can be based on either recollection or familiarity, these results do not show if the deficits are in both recollection and familiarity, or restricted to a single process. Therefore, it is necessary to look to procedures that provide separate measures of these two processes.

A&B describe a study by Knowlton and Squire (1995) using the remember/know procedure (Tulving 1985a) that found amnesics

were less likely to respond that they “knew” on the basis of familiarity that test items were studied, indicating that familiarity was disrupted. However, A&B also describe a study by Verfaellie and Treadwell (1993) using the process dissociation procedure (Jacoby 1991), that concluded that familiarity was preserved in amnesia. More recent studies using the remember/know procedure have led to different conclusions than either of the earlier studies. For example, Schacter et al. (1996) found that the proportion of knowing responses actually increased significantly for amnesic patients relative to healthy controls, indicating that amnesia was associated with an increase in familiarity. Given that all three of these studies included patients with widespread damage to the medial temporal lobes, if A&B are right, these patients should have exhibited deficits in both recollection and familiarity. On the surface, therefore, human amnesia data does not appear to provide consistent support for their new proposal.

However, a closer examination of these studies finds that the previous interpretations of these results were incomplete in that they did not fully account for response biases or, in the case of the remember/know studies, did not correct their measures to account for the mutual exclusivity between the remember/know responses. That is, in all of the reported studies the amnesics exhibited higher false alarm rates than the controls and this biased the estimates of recollection and familiarity. Moreover, the probability of a “know” response in the remember/know procedure is mathematically constrained by the proportion of remember responses, so that “knowing” responses by themselves do not provide an accurate measure of familiarity. In a recent article (Yonelinas et al. 1998), we reanalyzed the results of these earlier studies using a dual-process signal-detection model (Yonelinas 1994) that allowed for the independent contribution of recollection and familiarity and incorporated signal detection theory to accommodate differences in false alarms. We also tested additional amnesics using a receiver operating characteristics (ROC) analysis to validate the model and to derive estimates of recollection and familiarity in these patients.

The ROC analysis showed that the model provided an accurate account of recognition performance. Most important, however, was that all three estimation procedures converged in showing that both recollection and familiarity were disrupted in amnesics. Figure 1 summarizes the results of the remember/know, process dissociation, and ROC experiments. The results provide consis-

tent support for A&B’s proposal, in that patients with damage to both the hippocampal and perirhinal systems exhibit the expected deficits in both recollection and familiarity.

The results are also consistent with previous studies showing that amnesia is associated with a disproportionate disruption of recollection compared to familiarity (e.g., Kroll et al. 1996). Although familiarity was consistently disrupted in the amnesics, recollection was disrupted to a much greater extent. This disproportional deficit was observed in every condition of every experiment we examined. Note that recollection and familiarity are measured on different scales in Figure 1. The disproportional deficit in recollection is also observed, however, when the familiarity  $d'$  scores are converted to probabilities.

These results join a growing body of studies that show recollection and familiarity to be functionally dissociable memory processes, and suggest that they rely on distinct anatomical regions. However, a critical test of A&B’s specific proposal will be to determine whether patients with damage that is restricted to the hippocampal system or to the perirhinal system will exhibit selective deficits of recollection and familiarity.

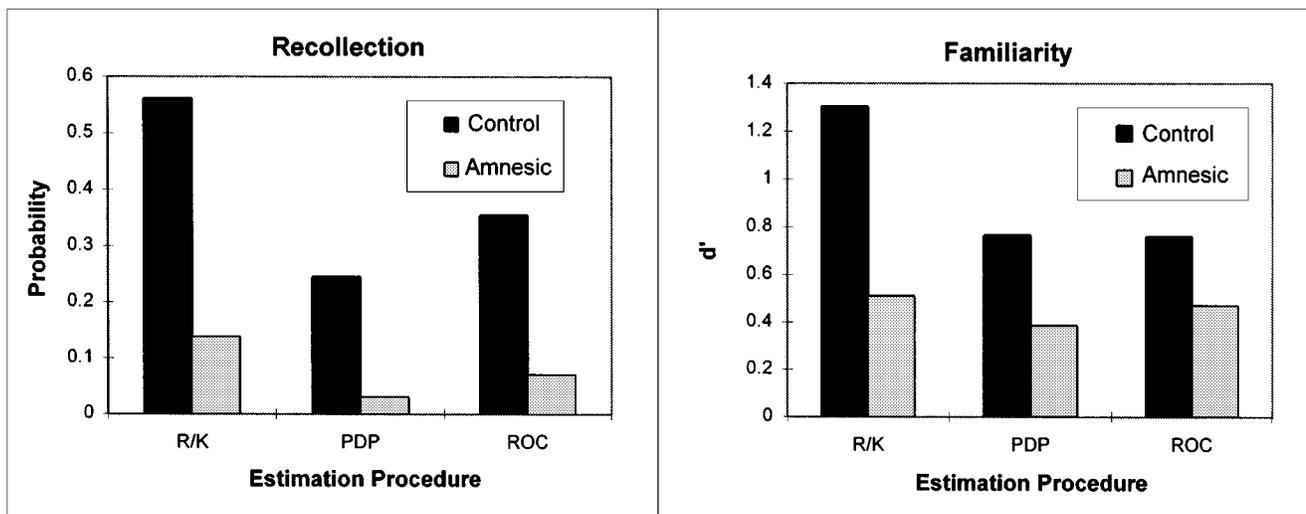


Figure 1 (Yonelinas et al.). Estimates of recollection and familiarity in amnesics and controls derived using the remember/know (R/K), the process dissociation (PDP), and the receiver operating characteristic (ROC) procedures. The R/K data reflect the average estimates derived from Knowlton and Squire (1995), Schacter et al. (1996 and 1997); the PDP data are from Verfaellie and Treadwell (1993); and the ROC data are from Yonelinas et al. (1998). All three procedures showed that amnesics exhibited a reduction in recollection and a smaller but consistent reduction in familiarity.