Human recognition memory: a cognitive neuroscience perspective

Michael D. Rugg[1] and Andrew P. Yonelinas[2]

[1]Institute of Cognitive Neuroscience and Department of Psychology, University College London, 17 Queen Square, London, WC1N 3AR, UK
[2]Department of Psychology, University of California, Davis, California 95616, USA

For many years the cognitive processes underlying recognition memory have been the subject of considerable interest in experimental psychology. To account for a broad range of behavioral findings, psychologists have put forward a variety of 'dual-process' models, all of which propose that recognition memory is supported by two forms of memory – familiarity and recollection – that differ in their speed of operation and the specificity of the retrieved information. More recently, the dual-process framework has been extended to encompass findings from studies investigating the neural basis of recognition memory. Results from neuropsychological, ERP and functional neuroimaging studies can be accommodated within the framework, and suggest that familiarity and recollection are supported by distinct neural mechanisms.

We have all had the uncomfortable experience of recognizing a person as familiar, yet being unable to recollect any qualitative information about the person such as their name or where we met the person before. Such experiences suggest that memory judgments can be based on an acontextual sense of familiarity, or on recollection of detailed information about previous events. Motivated in part by these types of experiences, a variety of 'dual-process' models propose that recognition reflects the products of two distinct memory processes (e.g. [1–5]). In general, these models assume that recollection depends on a relatively slow process, similar to that involved in recall tasks, which yields qualitative information about previous study events (e.g. when or where an item was studied). By contrast, familiarity reflects a purely quantitative, 'strength-like' memory signal. The models differ, however, in a variety of important ways. For example, some models focus solely on the functional nature of recollection and familiarity (e.g. [1]), whereas others make specific claims about the neural substrates and mechanisms of the two processes (e.g. [5]). Moreover, some models assume that familiarity and implicit forms of memory such as priming are two expressions of a common process (e.g. [2]).

Various methods have been developed to measure recollection and familiarity (see Box 1), and they have indicated that the two forms of memory can be fully dissociated. Moreover, they have led to a remarkably consistent picture about the functional nature of these processes (for a review see [6]). For example, recollection benefits more than familiarity from elaborative encoding, such as meaningful compared with perceptual processing, or active generation compared with passive reading. Moreover, recollection is slower and requires more attention during both encoding and retrieval than does familiarity. Conversely, familiarity is more sensitive to perceptual changes between study and test, and it alone is influenced by 'fluency' manipulations such as subliminal masked priming. Furthermore, as the study-test retention interval increases up to several minutes, familiarity declines whereas recollection is relatively unaffected. Finally, familiarity is much more sensitive to manipulations that influence how liberal or conservative subjects are in making their recognition responses.

The behavioral studies demonstrating that recollection and familiarity can be dissociated provide strong evidence that they reflect distinct forms of memory. However, alternative 'single-process' models have been proposed in which recollection is assumed to reflect the retrieval of strong, content-rich memories whereas familiarity is associated with weaker, less specific memories (e.g. [7]). Although such models have difficulty accounting for all of the reported behavioral dissociations [6], their apparent parsimony makes them important competitors of dual-process approaches.

The cognitive neuroscience perspective

Cognitive neuroscience, in which the methods of experimental psychology and neuroscience are combined, can complement purely behavioral studies of recognition memory in several ways. First, results showing that recollection and familiarity rely on different brain regions or neural mechanisms would provide an important source of convergent evidence that the two processes are qualitatively distinct; if familiarity simply reflects a weak form of recollection, then it should have similar neural correlates that differ only quantitatively from those related to recollection. Second, investigating the neural basis of recollection and familiarity would be particularly useful in directly testing the predictions of various dual-process models. For example, based primarily on research with non-human primates (see Box 2), some models have proposed that the hippocampus is critical for recollection, whereas familiarity is supported by adjacent medial temporal cortex [5,8]. This can be contrasted with the

Corresponding author: Michael D. Rugg (m.rugg@ucl.ac.uk).

 1364-6613/03/$ - see front matter doi:10.1016/S1364-6613(03)00131-1

Box 1. Methods used to assess recollection and familiarity

One approach to assessing recollection and familiarity is to examine performance under test conditions expected to rely to a greater extent on one process than the other. For example, because familiarity is expected to be a faster process than recollection, performance when subjects are required to make recognition decisions very quickly should rely more on familiarity than on recollection [64]. By contrast, because only recollection provides qualitative information about the previous study event, *relational* recognition tests, in which subjects must retrieve a specific aspect of the study event or its context, such as the sensory modality or spatial location in which the item was presented, or whether a word was initially paired with another context word, should rely mainly on recollection [65,66]. Similarly, because familiarity contributes little or nothing to free recall, performance in this task should also rely primarily on recollection [2].

In addition, several modeling approaches have been developed to derive quantitative estimates for the contribution of recollection and familiarity to overall recognition performance. One such method is the **process-dissociation procedure** [67], which is based on the premise that if a subject can recollect a given item, then they should be able to determine when or where it was initially studied, whereas familiarity should not support such a discrimination. Thus, recollection is estimated as the ability to make an accurate relational recognition judgment, and familiarity is estimated as the conditional probability of recognizing an item given that it was not recollected.

Another modeling method is based on the **'remember/know' procedure** [68], in which subjects are required to introspect about the basis of their recognition memory judgments and report whether they recognize items on the basis of remembering (i.e. recollection of qualitative information about the study event) or knowing (i.e. the item is familiar in the absence of recollection). Because subjects are instructed to respond 'remember' whenever they recollect a test item, the probability of a 'remember' response can be used as an index of recollection, whereas the probability that an item is familiar is equal to the conditional probability that it received a 'know' response given it was not recollected [69].

A third modeling method is the **'receiver operating characteristic' (ROC) procedure** [4]. In this procedure, subjects are required to rate the confidence of their recognition memory judgments which are then used to plot an ROC – the function that relates hits (i.e. proportion of studied items that are correctly recognized) to false alarms (i.e. the proportion of non-studied items that are incorrectly recognized) as a function of response confidence [70]. A model-based equation is then fitted to the observed empirical ROC to estimate the contribution of recollection and familiarity. The method is similar to that used when conducting a linear regression. In the present case, the function is curvilinear, and the two parameters that are estimated correspond to recollection and familiarity.

Each of the procedures just described relies on assumptions that can be questioned (e.g. the modeling methods assume that the two processes are statistically independent). Indeed, no single experiment, nor any single method, could by itself provide definitive evidence about the nature of the processes underlying recognition memory. Taken together, however, the convergent pattern of results obtained across a wide variety of different behavioral studies and measurement methods attests to the validity of the dual-process framework, and provides a strong foundation for developing a full understanding of the two memory processes.

view that the hippocampus supports a form of memory that underlies both recollection and familiarity (e.g. [9]). Here, we examine the recent contributions that cognitive neuroscience has made in advancing our understanding of recollection and familiarity.

Findings from amnesia

Neuropsychological evidence relevant to the dual-process framework has come largely from the study of patients with global amnesia – patients who, following neurological insult, demonstrate abnormally low scores on neuropsychological test batteries such as the Wechsler Memory Scale in the face of otherwise normal cognitive function. A prediction of one early dual-process model is that amnesic patients (regardless of aetiology) should suffer a selective deficit in recollection (e.g. [2]). This prediction is based on two findings. First, amnesics perform normally on most tests of implicit memory, such as perceptual identification [10]. If implicit memory and familiarity are equivalent, as has sometimes been assumed [2], then amnesics should exhibit preserved familiarity. Second, amnesic patients are excessively prone to confuse recently presented items with frequently presented items [11,12], as would be expected if their performance were based primarily on a strength-like index such as familiarity.

Current evidence does not support the view that, in general, familiarity is preserved in amnesic patients, or that implicit memory and familiarity are functionally equivalent. Amnesic patients (not selected by aetiology) have been reported to show greater deficits on tests of relational recognition than item recognition [13,14], as expected if recollection is disproportionately disrupted. Disproportionate deficits in free recall compared with recognition have also been reported in some studies [15–18], again consistent with greater impairment in recollection. However, in other studies equivalent levels of recall and recognition impairment have been reported [19–21] (but see [22]). Results from studies that used the process-dissociation [23], 'remember/know' [24–26], and receiver operating characteristic (ROC) procedures [27] also indicate that the memory deficit in amnesic patients unselected by aetiology is not confined to recollection. The results do suggest, however, that although familiarity is impaired, recollection is disrupted to a greater extent (for review see [27]). Importantly, inasmuch as implicit memory was preserved in the amnesic subjects tested in these studies, the findings suggest that familiarity and

Box 2. Recognition memory in animals

Distinctions similar to that embodied in the dual-process framework have been proposed in the context of research on animal memory [8,71]. One focus of this research has been whether different parts of the primate medial temporal lobe (MTL) – a region that includes the hippocampal formation and the parahippocampal, entorhinal and perirhinal cortices – have dissociable mnemonic functions. The finding that performance on recognition memory tasks such as 'delayed non-matching to sample' is profoundly impaired by lesions confined to perirhinal cortex [72,73], together with the observation that a significant proportion of perirhinal neurons demonstrate lower firing rates for recently experienced than for experimentally novel objects [74], have motivated the proposal that the perirhinal region plays a crucial role in familiarity-based recognition [8]. In a similar vein, reports that lesions affecting the hippocampal formation severely impair memory for complex associations, such as those between a stimulus event and its context (see [75] for review), have led to the suggestion that the hippocampus plays a crucial role in memory for specific episodes – recollection, in the present terminology. This research has brought the question of the neural substrates of human recognition memory into sharp focus, and has motivated efforts to determine whether different regions of the human MTL contribute differentially to recollection and familiarity.

implicit memory are not two manifestations of a single process (see also [28]).

Recognition memory and the hippocampus

More recent research has focused on whether recollection and familiarity can be dissociated in patients with selective lesions of the medial temporal lobe (MTL). One motivation for these studies is the proposal – which has not gone unchallenged [29] – that in non-human primates the hippocampus and the perirhinal cortex support processes akin to recollection and familiarity, respectively (Box 2). Relevant evidence comes primarily from patients with damage apparently restricted to the hippocampus. Almost invariably, these are patients who developed memory difficulties following a period of cerebral hypoxia (oxygen deprivation). The hippocampus is particularly vulnerable to hypoxic damage, and post-mortem studies have demonstrated that in some cases neuronal loss can be confined largely to this structure [30–32].

Some post-hypoxic patients do appear to exhibit selective deficits in recollection, whereas other seemingly similar patients appear to have impairments in both processes. For example, normal or near-normal performance on item recognition tasks, but very poor recall, has been reported both in a group of patients with hippocampal damage following hypoxia in early childhood [33] (see [34] for further investigation of a single case), and in an adult-onset patient with MRI evidence of selective hippocampal damage [35]. A disproportionate recall impairment has also been reported in a large group study of the effects of mild hypoxia that is discussed in more detail below [36]. Studies of other post-hypoxic patients with MRI evidence of damage limited primarily to the hippocampus have, however, failed to reveal a relative sparing of recognition memory [37–40]. Notably, on the same standardized test (the 'Doors and People Test') for which Baddeley *et al.* [34] and Mayes *et al.* [35] reported a large discrepancy between recall and recognition in their patients, no discrepancy was found either by Manns and Squire [38] in a group study, or in the single case study of Cipolotti *et al.* [39].

Relational memory in post-hypoxic patients has been assessed using tests of 'associative recognition' – tests that require pairs of items to be discriminated according to whether their members were paired with each other, or with different items, at the time they were studied. Dissociations between item and associative recognition have been reported both in the early-onset amnesic patients mentioned above [33], and the adult-onset case reported by Mayes and colleagues [33,41,42], but only for test items belonging to different stimulus classes (e.g. faces and names, or objects and locations) [33,43]. Very different findings were reported, however, in two other studies that contrasted item and associative recognition in patients with MRI evidence of hippocampal damage [44,45]; the patients showing no evidence of a relative sparing of item recognition.

Comparison of performance on tests of recall versus recognition, or item versus relational memory, provides only indirect evidence about the relative integrity of recollection and familiarity. Unfortunately, few studies of patients with selective hippocampal pathology have thus far reported results from process estimation methods (Box 1). One study examined the covariation between recall, recognition and severity of hypoxia (as indexed by coma duration) in a large sample of post-hypoxic patients [36]. Structural equation modeling showed that hypoxic severity predicted the degree to which recollection, but not familiarity, was impaired (Fig. 1). Furthermore, in a subgroup of these patients, estimates of recollection and familiarity derived from both remember/know and ROC methods (Box 1) supported a selective impairment in recollection. These findings provide strong evidence that recollection and familiarity can be dissociated in some post-hypoxic individuals. If it is assumed that the effects of hypoxia were mediated through damage to the hippocampus (MRI could not performed on these patients), the findings support the proposal that this structure is more important for recollection. In contrast, a recent study by Manns *et al.* [9] reported that recollection and familiarity, as assessed by the remember/know procedure, were both impaired in a group of post-hypoxic patients with MRI evidence of hippocampal damage.

In sum, whereas some studies have found that the hippocampus is more important for recollection than familiarity, others have not. The reasons for these divergent findings are unclear. In the case of the patients who sustained their lesions early in life [33], it is possible that their spared memory reflects, at least partially, developmental functional reorganization. Another factor might be neuropathological differences between patients, with those who exhibit familiarity impairment having extra-hippocampal damage not identified by MRI (as in case WH of Rempel-Clower *et al.* [31]). A third possibility is that patients with familiarity deficits have more complete hippocampal dysfunction than those who do not (but see [42]). By this last argument, recollection and familiarity both rely on the hippocampus, but familiarity is less likely to be affected by a subtotal lesion.

The study of amnesia has shown that at least some patients exhibit selective deficits in recollection, indicating that recollection can be dissociated from familiarity.

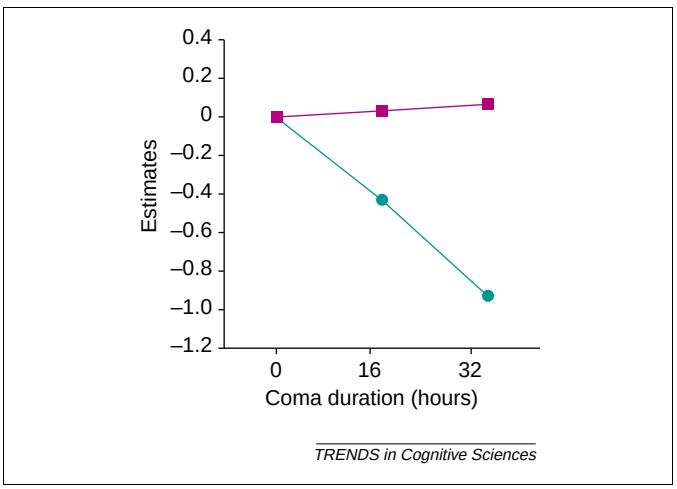

Fig. 1. Estimates of recollection (green symbols) and familiarity (purple symbols) plotted as a function of hypoxic severity as measured by coma duration. Estimates were derived using the regression coefficients from a structural equation model that linked coma duration to recall and recognition measures. Hypoxic severity was related to a decrease in recollection and no change in familiarity. Data redrawn from Ref. [36].

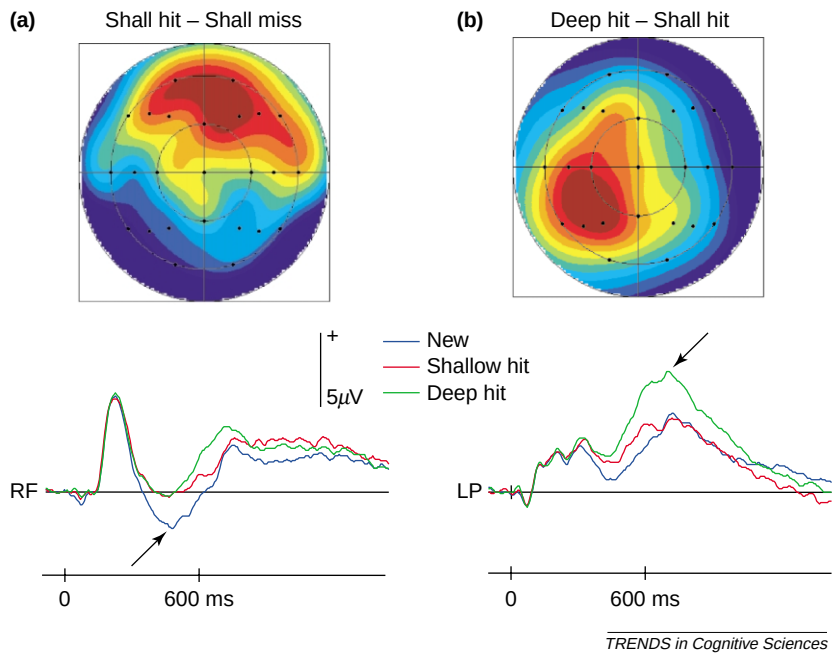

Fig. 2. ERP waveforms from (a) right frontal (RF), and (b) left parietal (LP) electrodes in the study of Rugg *et al.* [56], illustrating putative ERP correlates of familiarity and recollection respectively (arrows; see text for further details). Above each waveform are the scalp topographies of the effects, as revealed by the ERP voltage differences. For familiarity (a) the voltage difference is between words subjected to shallow study and subsequently recognized (Shallow hits) or misclassified as 'New' (Shallow misses), and has a latency in the range 300–500 ms; for recollection (b), it is between Deep hits and Shallow hits and has a latency in the range 500–800 ms.

However, evidence that the hippocampus plays a selective role in recollection is equivocal. Further progress on this issue would benefit from increasing the range of neuroimaging techniques and analysis methods to characterize neuropathology than has typically been the case thus far (e.g. [46]).

An important limitation of current evidence from amnesia is that, at best, it takes the form of a 'single' dissociation. In the absence of reports of patients demonstrating the complementary dissociation– intact recollection and impaired familiarity – it is difficult to reject the possibility that measures of recollection are just more sensitive to memory impairment than measures of familiarity. Because the cortex of the parahippocampal gyrus supplies the hippocampus with most of its input [47], even if the hypothesis that familiarity is a parahippcampal function (e.g. [36]) is correct, a selective familiarity deficit might be difficult to find; lesions that impair familiarity might inevitably deafferent the hippocampus sufficiently to affect recollection.

Event-related potential studies

ERPs have been used in several studies relevant to the dual-process framework. Although ERPs lack the spatial resolution necessary to address questions about the neural substrates of different processes, they are valuable for determining whether neural correlates of recollection and familiarity differ qualitatively (indexed by ERP effects that differ in scalp distribution rather than simply in magnitude), as would be expected if the two forms of memory have different neural substrates. A number of ERP studies have addressed this question by comparing ERPs elicited by test items recognized on the basis of familiarity or recollection. Although the results of initial studies (e.g. [48–50]) offered limited evidence that recollection and familiarity have distinct neural correlates (see [51] for review), later findings suggest that the ERP correlates of familiarity and recollection can be dissociated on a combination of functional, temporal, and neuroanatomical grounds. For example, Rugg *et al.* [52] contrasted ERPs to items recognized predominantly on the basis of familiarity (following exposure in a 'shallow' study task), with ERPs when the probability of familiarity- and recollection-based recognition were both high (after 'deep' study). As illustrated in Fig. 2, both classes of recognition judgment were associated with an early, frontal effect, whereas only deeply studied items elicited a subsequent, left-lateralized, parietal positivity. Importantly, the early frontal effect was absent for old items misclassified as new, suggesting that the effect is a correlate of processes associated with a positive recognition judgment rather than, say, priming. These findings can be explained by assuming that the frontal effect common to both classes of recognized item is a correlate of familiarity, whereas the later parietal effect indexes recollection.

Dissociations between frontal and parietal ERP memory effects have also been described by Curran [53,54]. Curran used a recognition memory test in which some of the new items were similar to studied items (e.g. SWAMPS → SWAMP) [53]. These items, which attract a high proportion of false alarms, are held to give rise to strong familiarity in the absence of recollection [55]. ERPs associated with these false alarms showed an early, frontal positivity relative to items correctly judged new, but did not exhibit the later parietal effect (see also [56]). By contrast, ERPs elicited by recognized old items elicited both effects. Similar findings were reported in a subsequent study using pictures [54].

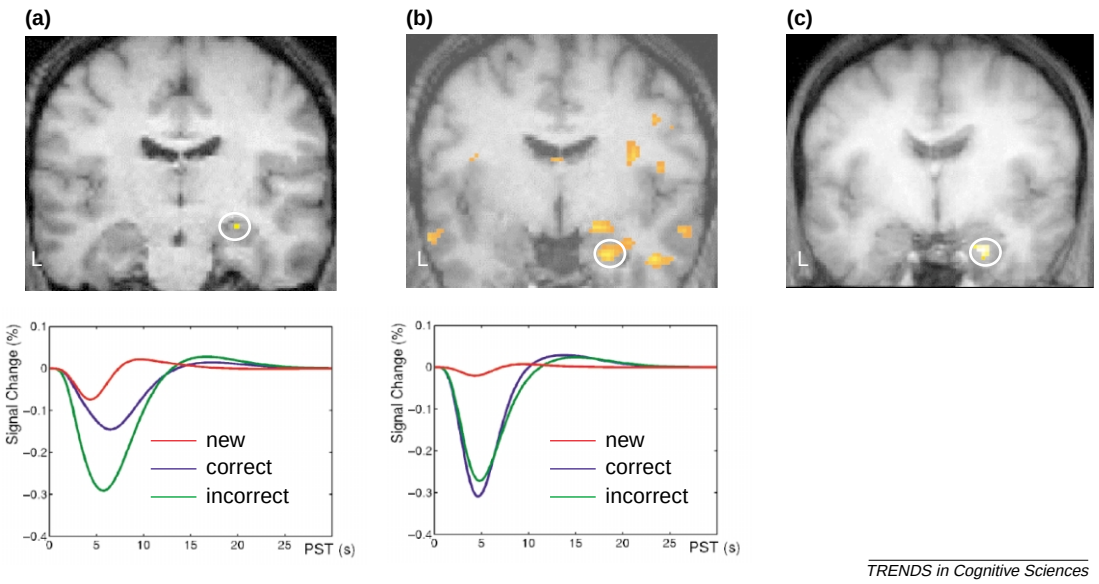

Fig. 3. (a) fMRI findings from the study of Cansino *et al.* [60], rendered on to a single subject's magnetic resonance image and illustrating the right hippocampal region in which activity was greater for recognized test items accorded correct vs. incorrect source judgments. Below the image are the time-courses of the best fitting responses for these two classes of judgment, along with those for correctly classified new items. (b) Data from the region of right anterior medial temporal lobe (MTL) cortex in Ref. [60], where recognized test items elicited smaller responses than did new items, regardless of the accuracy of the associated source judgment. Time courses of the best-fitting responses illustrated below. (c) Data from the meta-analysis of Henson *et al.* [62], illustrating the common right anterior MTL region where responses elicited by new items exceeded those to old items in four different studies. Data rendered onto the average of the magnetic resonance images of a representative subject from each study.

fMRI correlates of recollection and familiarity

The ERP findings described above suggest that recollection and familiarity depend on neural substrates that are at least partially non-overlapping, but give no direct information about the specific brain regions that are differentially active during recollection- and familiarity-based recognition. In principle, such information can be obtained by analogous studies using fMRI. At the present time, however, relevant evidence from fMRI studies is sparse; whereas contrasts between activity elicited by old and new items have been reported in more than a dozen studies (for review see [57]), only a handful have used procedures for separating familiarity- and recollection-based recognition. The experimental effects obtained in these studies are sometimes hard to interpret, since the low temporal resolution of fMRI means that the timing of an experimental effect cannot be used as a guide to its likely functional significance. Thus, it is not always easy to distinguish between neural activity engaged during the initial retrieval of information, and activity supporting processes – such as evaluation and decision-making – that operate 'downstream' of retrieval.

Two fMRI studies contrasted the activity elicited by test words subjected to remember/know judgments [58,59]. In both cases greater activity was found in left lateral parietal cortex (Brodmann Areas 39/40) for old items endorsed as 'remembered' rather than 'known'. Together with findings from ERP studies suggesting a link between enhanced parietal positivity and recollection (Fig. 2), these fMRI results suggest the lateral parietal region plays a role in this form of memory. A second finding, reported only by Eldridge *et al.* [59], was of greater activity in the hippocampus and adjacent MTL cortex for items endorsed as 'remembered' than for items classified as 'known' or 'new'. In a third study [60], which used pictures as test items, the test task required subjects to classify the items as 'new' or, if 'old', to signal the location where they had been presented during study. Recollection was operationalized as the contrast between items correctly judged 'old' according to the accuracy of the location judgment. As illustrated in Fig. 3a, this contrast revealed greater activity in the right hippocampal region for 'recollected' than 'unrecollected' items. A similar finding was reported by Dobbins *et al.* [61]. In this study subjects encoded words in one of two study tasks, and subsequently judged which of two old words had been encoded in a given task. Correct judgments were associated with more activity in the hippocampus and adjacent cortex than were incorrect judgments.

On the assumption that recollection and familiarity are independent, a significant proportion of items recognized on the basis of recollection will also be familiar. Thus, the neural correlates of familiarity should be evident in differences in activity common to the contrasts between recollected vs. new items on the one hand, and familiar vs. new items on the other (cf. the aforementioned early frontal ERP effect). Henson *et al.* [58] reported findings relevant to this comparison. An anterior region of the MTL – identified by the authors as the amygdala – exhibited greater activity for new items than for items attracting either 'remember' or 'know' judgments. According to the logic just outlined, this difference in activity is a putative correlate of familiarity. A recent meta-analysis of four additional studies [62] (including Cansino *et al.* [60]) adds weight to these findings, in that old items were consistently found to elicit less MTL activity than new items. The effects varied in their spatial extent and degree of lateralization, but included a common region identified as right anterior entorhinal/perirhinal cortex (Fig. 3c), near to one of the areas showing the analogous effect in

Questions for future research

- What is the relationship between familiarity and different kinds of implicit memory, such as those that underlie perceptual versus conceptual priming?
- Is there more than one kind of familiarity, and under what conditions does each kind influence performance?
- Which regions of the medial temporal lobe (MTL) contribute to familiarity?
- What role do regions other than the MTL – for example, parietal cortex – play in familiarity- and recollection-based recognition?

Henson *et al.* [58]. Importantly, the data from Cansino *et al.* [60] demonstrated equivalent response reductions for recognized items regardless of the accuracy of the associated source judgment, thus demonstrating a dissociation between hippocampal and perirhinal retrieval effects (cf. Figs 3a and 3b). Consistent with these findings, levels of hippocampal and perirhinal activity elicited by items during study have been reported to predict subsequent source and recognition memory performance, respectively [63].

The foregoing findings [59–61] suggest that the hippocampus is more active when recognition is accompanied by recollection than when recognition is based on familiarity alone. The findings are consistent with the idea that this structure plays a selective role in recollection, but do not necessitate this conclusion; the alternative possibility that hippocampal activity is correlated with the amount, rather than the nature, of the information that is retrieved from memory cannot be discounted at present. Complementing the results for the hippocampus, differences in activity observed in the anterior MTL for old and new test items [62] are suggestive of a neural correlate of familiarity, but again it remains necessary to rule out alternative possibilities, such as a priming account. The direction of these differences in activity is intriguing, however, in light of reports that the anterior MTL in the monkey contains neurons in which the firing rate is inversely proportional to an object's relative familiarity (Box 2). In summary, although the fMRI findings can be interpreted as supporting both the dual-process framework, and the proposal that the distinction between recollection and familiarity is maintained within the MTL [8,36], further research is required to rule out alternative interpretations.

Concluding comments

Although developed initially as functional accounts of behavioral findings in healthy young adults, dual-process models provide a useful framework for integrating these findings with the results of neuropsychological, ERP and fMRI studies of recognition memory. Together, the results of these studies support the idea that recognition is supported by functionally dissociable processes, and suggest that these depend upon qualitatively distinct neural mechanisms. In addition, the results strongly support the view that the hippocampus plays a crucial role in recollection, and argue for a less important role for this structure in familiarity. Further work is necessary, however, to determine the extent of the role of the hippocampus in familiarity-driven recognition, and to identify other regions, both within and outside the MTL, that play a selective role in recollection or familiarity (see also Questions for future research).

Acknowledgements

M.D.R. is supported by the Wellcome Trust and UK Medical Research Council. A.P.Y. is supported by the National Institute of Mental Health, USA.

References

1 Atkinson, R.C. and Juola, J.F. (1974) Search and decision processes in recognition memory. In *Contemporary Developments in Mathematical Psychology* In *Learning, Memory and Thinking* (Vol.1) (Krantz, D.H., ed.), pp. 242–293, W.H. Freeman
2 Mandler, G. (1980) Recognizing: the judgement of previous occurrence. *Psychol. Rev.* 87, 252–271
3 Jacoby, L.L. and Dallas, M. (1981) On the relationship between autobiographical memory and perceptual learning. *J. Exp. Psychol. Gen.* 110, 306–340
4 Yonelinas, A.P. (1994) Receiver-operating characteristics in recognition memory: evidence for a dual-process model. *J. Exp. Psychol. Learn. Mem. Cogn.* 20, 1341–1354
5 O'Reilly, R.C. and Norman, K.A. (2002) Hippocampal and neocortical contributions to memory: advances in the complementary learning systems framework. *Trends Cogn. Sci.* 6, 505–510
6 Yonelinas, A.P. (2002) The nature of recollection and familiarity: a review of 30 years of research. *J. Mem. Lang.* 46, 441–517
7 Donaldson, W. (1996) The role of decision processes in remembering and knowing. *Mem. Cogn.* 24, 523–533
8 Brown, M.W. and Aggleton, J.P. (2001) Recognition memory: what are the roles of the perirhinal cortex and hippocampus? *Nat. Rev. Neurosci.* 2, 51–61
9 Manns, J.R. *et al.* (2003) Recognition memory and the human hippocampus. *Neuron* 37, 171–180
10 Moscovitch, M. *et al.* (1993) Implicit tests of memory in patients with focal lesions or degenerative brain disorders. *Handbook of Neuropsychology* (Vol. 8) (Boller, F., Grafman, J., *et al.* eds), pp. 133–173, Elsevier
11 Huppert, F.A. and Piercy, M. (1978) The role of trace strength in recency and frequency judgments by amnesic and control subjects. *Q. J. Exp. Psychol.* 30, 347–354
12 Mayes, A.R. *et al.* (1989) Why are amnesic judgments of recency and frequency made in a qualitatively different way from those of normal people? *Cortex* 25, 479–488
13 Downes, J.J. *et al.* (2002) Temporal order memory in patients with Korsakoff's syndrome and medial temporal amnesia. *Neuropsychologia* 40, 853–861
14 Pickering, A.D. *et al.* (1989) Amnesia and memory for modality information. *Neuropsychologia* 27, 1249–1259
15 Hirst, W. *et al.* (1986) Recognition and recall in amnesics. *J. Exp. Psychol. Learn. Mem. Cogn.* 12, 445–451
16 Hirst, W. *et al.* (1988) More on recognition and recall in amnesics. *J. Exp. Psychol. Learn. Mem. Cogn.* 14, 758–762
17 Isaac, C.L. and Mayes, A.R. (1999) Rate of forgetting in amnesia: I. Recall and recognition of prose. *J. Exp. Psychol. Learn. Mem. Cogn.* 25, 942–962
18 Volpe, B.T. *et al.* (1986) Further characterization of patients with amnesia after cardiac arrest: preserved recognition memory. *Neurology* 36, 408–411
19 Haist, F. *et al.* (1992) On the relationship between recall and recognition memory. *J. Exp. Psychol. Learn. Mem. Cogn.* 18, 691–702
20 MacAndrew, S.B. *et al.* (1994) No selective deficit in recall in amnesia. *Memory* 2, 241–254
21 Kopelman, M.D. and Stanhope, N. (1998) Recall and recognition memory in patients with focal frontal, temporal lobe, and diencephalic lesions. *Neuropsychologia* 36, 785–795
22 Giovanello, K.S. and Verfaellie, M. (2001) The relationship between

recall and recognition in amnesia: effects of matching recognition between patients with amnesia and controls. *Neuropsychology* 15, 444–451
23 Verfaellie, M. and Treadwell, J.R. (1993) Status of recognition memory in amnesia. *Neuropsychology* 7, 5–13
24 Knowlton, B.J. and Squire, L.R. (1995) Remembering and knowing: two different expressions of declarative memory. *J. Exp. Psychol. Learn. Mem. Cogn.* 21, 699–710
25 Schacter, D.L. *et al.* (1996) The neuropsychology of memory illusions: false recall and recognition in amnesic patients. *J. Mem. Lang.* 35, 319–334
26 Schacter, D.L. *et al.* (1997) Illusory memories in amnesic patients: conceptual and perceptual false recognition. *Neuropsychology* 11, 331–342
27 Yonelinas, A.P. *et al.* (1998) Recollection and familiarity deficits in amnesia: convergence of remember-know, process dissociation, and receiver operating characteristic data. *Neuropsychology* 12, 323–339
28 Stark, C.E. and Squire, L.R. (2000) Recognition memory and familiarity judgments in severe amnesia: no evidence for a contribution of repetition priming. *Behav. Neurosci.* 114, 459–467
29 Zola, S.M. *et al.* (2000) Impaired recognition memory in monkeys after damage limited to the hippocampal region. *J. Neurosci.* 20, 451–463
30 Zola-Morgan, S. *et al.* (1986) Human amnesia and the medial temporal region: enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus. *J. Neurosci.* 6, 2950–2967
31 Rempel-Clower, N.L. *et al.* (1996) Three cases of enduring memory impairment after bilateral damage limited to the hippocampal formation. *J. Neurosci.* 16, 5233–5255
32 Cummings, J.L. *et al.* (1984) Amnesia with hippocampal lesions after cardiopulmonary arrest. *Neurology* 34, 679–681
33 Vargha-Khadem, F. *et al.* (1997) Differential effects of early hippocampal pathology on episodic and semantic memory. *Science* 277, 376–380
34 Baddeley, A. *et al.* (2001) Preserved recognition in a case of developmental amnesia: implications for the acquisition of semantic memory? *J. Cogn. Neurosci.* 13, 357–369
35 Mayes, A.R. *et al.* (2002) Relative sparing of item recognition memory in a patient with adult-onset damage limited to the hippocampus. *Hippocampus* 12, 325–340
36 Yonelinas, A.P. *et al.* (2002) Effects of extensive temporal lobe damage or mild hypoxia on recollection and familiarity. *Nat. Neurosci.* 5, 1236–1241
37 Reed, J.M. and Squire, L.R. (1997) Impaired recognition memory in patients with lesions limited to the hippocampal formation. *Behav. Neurosci.* 111, 667–675
38 Manns, J.R. and Squire, L.R. (1999) Impaired recognition memory on the doors and people test after damage limited to the hippocampal region. *Hippocampus* 9, 495–499
39 Cipolotti, L. *et al.* (2001) Long-term retrograde amnesia: the crucial role of the hippocampus. *Neuropsychologia* 39, 151–172
40 Verfaellie, M. *et al.* (2000) Acquisition of novel semantic information in amnesia: effects of lesion location. *Neuropsychologia* 38, 484–492
41 Mayes, A.R. *et al.* (1999) What are the functional deficits produced by hippocampal and perirhinal cortex lesions? *Behav. Brain Sci.* 22, 460–461
42 Holdstock, J.S. *et al.* (2002) Under what conditions is recognition spared relative to recall after selective hippocampal damage in humans? *Hippocampus* 12, 341–351
43 Mayes, A.R. *et al.* (2001) Memory for single items, word pairs, and temporal order of different kinds in a patient with selective hippocampal lesions. *Cogn. Neuropsychol.* 18, 97–123
44 Stark, C.E. *et al.* (2002) Recognition memory for single items and for associations is similarly impaired following damage to the hippocampal region. *Learn. Mem.* 9, 238–242
45 Stark, C.E. and Squire, L.R. (2003) Hippocampal damage equally impairs memory for single items and memory for conjunctions. *Hippocampus* 13, 281–292
46 Gadian, D.G. *et al.* (2000) Developmental amnesia associated with early hypoxic-ischaemic injury. *Brain* 123, 499–507
47 Lavenex, P. and Amaral, D.G. (2000) Hippocampal-neocortical interaction: a hierarchy of associativity. *Hippocampus* 10, 420–430
48 Smith, M.E. (1993) Neurophysiological manifestations of recollective experience during recognition memory judgments. *J. Cogn. Neurosci.* 5, 1–13
49 Düzel, E. *et al.* (1997) Event-related brain potential correlates of two states of conscious awareness in memory. *Proc. Natl. Acad. Sci. U. S. A.* 94, 5973–5978
50 Wilding, E.L. and Rugg, M.D. (1996) An event-related potential study of recognition memory with and without retrieval of source. *Brain* 119, 889–905
51 Rugg, M.D. and Allan, K. (2000) Memory retrieval: an electrophysiological perspective. In *The New Cognitive Neurosciences* (Gazzaniga, M.S., ed.), pp. 805–816, MIT Press
52 Rugg, M.D. *et al.* (1998) Dissociation of the neural correlates of implicit and explicit memory. *Nature* 392, 595–598
53 Curran, T. (2000) Brain potentials of recollection and familiarity. *Mem. Cogn.* 28, 923–938
54 Curran, T. and Cleary, A.M. (2003) Using ERPs to dissociate recollection from familiarity in picture recognition. *Cogn. Brain Res.* 15, 191–205
55 Hintzman, D.L. and Curran, T. (1994) Retrieval dynamics of recognition and frequency judgments: evidence for separate processes of familiarity and recall. *J. Mem. Lang.* 33, 1–18
56 Rugg, M.D. *et al.* (2002) Electrophysiological studies of retrieval processing. *Neuropsychology of Memory*, 3rd edn, (Squire, L.R., Schacter, D.L., *et al.* eds), pp. 154–165, Guilford Press
57 Rugg, M.D. and Henson, R.N.A. (2002) Episodic memory retrieval: an (event-related) functional neuroimaging perspective. In *The Cognitive Neuroscience of Memory Encoding and Retrieval* (Parker, A.E., ed.), pp. 3–37, Psychology Press
58 Henson, R.N.A. *et al.* (1999) Recollection and familiarity in recognition memory: an event-related functional magnetic resonance imaging study. *J. Neurosci.* 19, 3962–3972
59 Eldridge, L.L. *et al.* (2000) Remembering episodes: a selective role for the hippocampus during retrieval. *Nat. Neurosci.* 3, 1149–1152
60 Cansino, S. *et al.* (2002) Brain activity underlying encoding and retrieval of source memory. *Cereb. Cortex* 12, 1048–1056
61 Dobbins, I.G. *et al.* (2003) Memory orientation and success: separable neurocognitive components underlying episodic recognition. *Neuropsychologia* 41, 318–333
62 Henson, R.N.A. *et al.* (2003) A familiarity signal in human anterior medial temporal cortex? *Hippocampus* 13, 301–304
63 Davachi, L. *et al.* Multiple routes to memory: Distinct medial temporal lobe processes build item and source memories. *Proc. Natl. Acad. Sci. U. S. A.* (in press)
64 Hintzman, D.L. *et al.* (1998) Retrieval dynamics in recognition and list discrimination: further evidence of separate processes of familiarity and recall. *Mem. Cogn.* 26, 449–462
65 Hockley, W.E. and Consoli, A. (1999) Familiarity and recollection in item and associative recognition. *Mem. Cogn.* 27, 657–664
66 Yonelinas, A.P. (1999) The contribution of recollection and familiarity to recognition and source-memory judgments: A formal dual-process model and an analysis of receiver operating characteristics. *J. Exp. Psychol. Learn. Mem. Cogn.* 25, 1415–1434
67 Jacoby, L.L. (1991) A process dissociation framework: separating automatic from intentional uses of memory. *J. Mem. Lang.* 30, 513–541
68 Tulving, E. (1985) Memory and consciousness. *Can. J. Psychol.* 26, 1–12
69 Yonelinas, A.P. and Jacoby, L.L. (1995) The relation between remembering and knowing as bases for recognition: effects of size congruency. *J. Mem. Lang.* 34, 622–643
70 Macmillan, N.A. and Creelman, C.D. (1991) *Detection Theory: A User's Guide*, Cambridge University Press
71 Eichenbaum, H. *et al.* (1996) Two functional components of the hippocampal memory system. *Behav. Brain Sci.* 17, 449–472
72 Meunier, M. *et al.* (1993) Effects on visual recognition of combined and separate ablations of the entorhinal and perirhinal cortex in rhesus monkeys. *J. Neurosci.* 13, 5418–5432
73 Buffalo, E.A. *et al.* (1999) Dissociation between the effects of damage to perirhinal cortex and area TE. *Learn. Mem.* 6, 572–599
74 Brown, M.W. and Xiang, J.Z. (1998) Recognition memory: neuronal substrates of the judgement of prior occurrence. *Prog. Neurobiol.* 55, 149–189
75 Eichenbaum, H. (2000) A cortical-hippocampal system for declarative memory. *Nat. Rev. Neurosci.* 1, 41–50