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Sparing of the familiarity component of recognition memory in a patient with hippocampal pathology

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Abstract

Subject KN has a persistent anterograde amnesia as a result of brain injury following meningitis in 1993. MRI scans reveal a bilateral decrease in the volume of his hippocampal region (dentate gyrus, CA1–4, subicular cortices) of approximately 45% in both the right and left hemispheres, although the volume of his perirhinal cortex appears normal. Aside from some changes to his occipital lobe and bilateral shrinkage of the amygdala, the rest of his brain appears normal on recent quantitative MRI scans. A striking feature of his memory loss is his ability to perform at normal levels on some tests of recognition, despite his consistent deficit on tests of recall. Two tests designed specifically to distinguish performance of two putative divisions of recognition memory (the Remember/Know procedure and the use of receiver operating characteristics to distinguish familiarity and recollection), provide evidence for a selective sparing of the familiarity component of recognition. The dissociation within recognition memory supports dual-process models of recognition, and also supports proposals that anatomically linked regions within the medial temporal lobe make qualitatively different contributions to recognition.

Keywords: Amnesia; Recognition memory; Familiarity; Perirhinal cortex; Hippocampus

1. Introduction

In recent years two opposing views have emerged concerning the relative importance of the hippocampus for recognition memory and for the recall of episodic information. One proposal is that the hippocampus is just as essential for recognition as it is for recall. This view arises from the notion that both aspects of memory are intrinsic components of the same class of memory (Knowlton & Squire, 1995). A second proposal is that the hippocampus is only essential for recollective aspects of recognition (Aggleton & Shaw, 1996; O'Reilly, Norman, & McClelland, 1998; Yonelinas et al., 2002). This second view can be traced back to dual-process models of recognition memory (Mandler, 1980), and assumes that recognition is composed of two kinds of memory. One kind reflects a feeling of familiarity (sometimes called 'knowing') while the other reflects the actual recollection of events associated with the previously encountered stimulus (sometimes called 'remembering') (Gardiner, 1988; Yonelinas, 2002). It is supposed that each of these two forms of memory depend on one or more processes unique to it (Yonelinas, 2002). This second view predicts that patients with selective hippocampal pathology can still use their intact familiarity information to guide recognition and so show a relative sparing on many tests of recognition (Aggleton & Brown, 1999).

As the two proposals make very different predictions concerning the outcome of bilateral hippocampal pathology in humans this debate should be relatively straightforward to

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resolve. This has not, however, been the case. One major problem is that a likely source of familiarity information is the perirhinal cortex (Brown & Aggleton, 2001; Meunier, Bachevalier, Mishkin, & Murray, 1993; Murray & Richmond, 2001), which is subadjacent to the hippocampus. For this reason, lesion locus and extent become critical features in any test of these two different views. As a consequence, the most informative cases are those in which the pathology is both restricted to the hippocampus and sufficient to disrupt recall.

Most of the clinical evidence concerning this debate comes from the memory loss associated with hypoxic-induced hippocampal damage. The data on this condition are not, however, consistent. In one series of studies, Squire and his colleagues have repeatedly reported that recall and recognition are equivalently disrupted by hippocampal pathology following hypoxia (Manns, Hopkins, Reed, Kitchener, & Squire, 2003; Manns & Squire, 1999; Reed & Squire, 1997; Stark, Bayley, & Squire, 2002). This series includes three hypoxic cases where post-mortem evidence has confirmed that cell loss is largely confined to the hippocampal formation (Reed, Hamann, Stedanacci, & Squire, 1997). For surviving patients MRI has been used to reveal bilateral hippocampal shrinkage of between 10 and 46% (Manns et al., 2003; Stark et al., 2002), while the parahippocampal gyrus volume is within normal limits (±15%). These cases, who are amnesic, are impaired on a variety of recognition memory tests at levels that seem comparable to their recall deficits (Manns & Squire, 1997; Manns et al., 2003; Reed et al., 1997). Of especial relevance is the report that six hypoxic patients with pathology thought to be primarily limited to the hippocampus, as determined by MRI, fail to show a preservation of familiarity when it is assessed by introspective judgements of 'remember' or 'know' responses in a yes/no recognition task (Manns et al., 2003). The authors conclude that the hippocampus is important for both recollection and familiarity (Manns et al., 2003).

Contradictory findings have, however, come from different cohorts of cases with hypoxic-induced pathology (Turriziana, Fadda, Caltragirone, & Carlesimo, 2004; Yonelinas et al., 2002). Here, recall is more disrupted than recognition. In addition, tests on a subgroup of hypoxic cases by Yonelinas et al. (2002) found impaired recollective aspects of recognition while familiarity-based responses appeared intact. Furthermore, analysis of recall and recognition performance in 56 hypoxic cases using covariance structural modelling found that the best fit for the data involved dual, independent processes within recognition, and that a single factor explanation for recall and recognition was insufficient (Quamme, Yonelinas, Widaman, Kroll, & Sauvė, 2004). Related to these findings are studies of people who suffered hippocampal damage at a young age, again due to hypoxia. These cases of 'developmental amnesia' can also show a relative sparing of recognition (Vargha-Khadem et al., 1997), which has been most detailed in the case Jon (Baddeley, Vargha-Khadem, & Mishkin, 2001). Unfortunately, attempts to compare levels of 'remember' or 'know' responses in Jon have proved problematic as he does not seem to have a typical concept of what constitutes 'remember' (Baddeley et al., 2001). It has also been suggested that the early age of hippocampal pathology in cases like Jon could lead to compensation during development (Manns & Squire, 1999).

The apparent lack of consistency between studies of hypoxia, coupled with an unresolved debate over whether hypoxia might induce more generalised neural dysfunction (Bachevalier & Meunier, 1996; Caine & Watson, 2002; but see Squire & Zola, 1996), underlines the need for neuropsychological data from other causes of restricted hippocampal pathology. For these reasons case YR is especially interesting as she has an estimated bilateral volume decrease of 46% in her hippocampi while her entorhinal and perirhinal cortices appear intact and are of normal volume (Holdstock et al., 2002; Mayes, Holdstock, Isaac, Hunkin, & Roberts, 2002). While her recall performance is consistently impaired, her recognition scores across a wide array of tasks are markedly better and usually fall in the normal range (Holdstock et al., 2002; Mayes et al., 2002). Thus, of 43 item recognition tests, on only four tests did her scores fall more than 1.96 S.D. below the mean score of her control group. This pattern of results has been interpreted as reflecting a sparing of familiarity memory, a view that receives some support from the 'Remember/Know' procedure (Holdstock et al., 2002). Although the onset of YR's amnesia coincided with the administration of an opiate drug, which may have led to an ischemic incident, there is some uncertainty about the precise aetiology of her pathology.

The present study concerns a man (KN) who suffered temporal lobe damage as a result of meningitis. This pathology occurred in 1993 when KN was 34. His MRI scans show evidence of bilateral hippocampal pathology while much of the remainder of his temporal lobes appear normal. The most obvious cognitive consequence of his pathology is severe anterograde amnesia, although he is able to learn and retain some new semantic knowledge (McKenna & Gerhand, 2002). As KN provides a rare opportunity to test the two opposing views concerning the effects of hippocampal pathology on recognition, the first goal was to compare more formally his performance on tests of recall and recognition. The second goal was to measure his performance on tests specifically designed to distinguish the putative 'recollective' and 'familiarity' components of recognition memory (Yonelinas & Kroll, 1998). In parallel with these cognitive tests we sought to quantify more precisely the locus and extent of his medial temporal lobe pathology. In describing his pathology, the term 'hippocampal region' is used throughout this text to refer to the dentate gyrus, hippocampal fields CA1–4, and the subicular cortices.

2. Methods

2.1. Participants

Approval for this study was provided by a Multi-Centre Research Ethics Committee (MREC). All partici-