Behavioural Brain Research 225 (2011) 297–304

Contents lists available at ScienceDirect

Behavioural Brain Research

journal homepage: www.elsevier.com/locate/bbr

Research report

Damage to the lateral prefrontal cortex impairs familiarity but not recollection

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ARTICLE INFO

Article history:
Received 7 December 2010
Received in revised form 19 July 2011
Accepted 22 July 2011
Available online 30 July 2011

Keywords:
Recognition memory
Lateral prefrontal cortex
Recollection
Familiarity
Receiver operating characteristics

ABSTRACT

Frontal lobe lesions impair recognition memory but it is unclear whether the deficits arise from impaired recollection, impaired familiarity, or both. In the current study, recognition memory for verbal materials was examined in patients with damage to the left or right lateral prefrontal cortex. Words were incidentally encoded under semantic or phonological orienting conditions, and recognition memory was tested using a 6-point confidence procedure. Receiver operating characteristics (ROCs) were examined in order to measure the contributions of recollection and familiarity to recognition memory. In both encoding conditions, lateral prefrontal cortex damage led to a deficit in familiarity but not recollection. Similar deficits were observed in left and right hemisphere patients. The results indicate that the lateral prefrontal cortex plays a critical role in the monitoring or decision processes required for accurate familiarity-based recognition responses.

Determining the neural substrates of episodic memory has been the focus of decades of research, beginning with the classic case of H.M., which revealed the importance of the medial temporal lobes [1]. In recent years, the contribution of the prefrontal cortex (PFC) to episodic memory has received growing attention. Frontal lobe lesions lead to a variety of subtle but noticeable memory impairments, particularly in the strategic control of encoding and retrieval [2–7]. Frontal patients perform poorly on many long-term memory tasks, including free recall, cued recall, and source and temporal order memory [see Refs. [2,8–10] for reviews]. They also show increased susceptibility to interference [e.g., Ref. [11]] and have difficulty with strategy implementation at encoding and retrieval, which extends to the organization and monitoring of retrieval from remote memory [12,13].

The body of research on long-term memory deficits in patients with frontal lesions has focused largely on memory tasks that involve some degree of strategy implementation at study, test, or both, and considerable progress has been made in understanding the strategic memory deficits in these patients. What has received less attention is a precise understanding of how the PFC contributes to item recognition, where the demand for strategic retrieval processes is minimized. Although item recognition was initially thought to be preserved in PFC patients [14], it is now apparent that PFC lesions do in fact impair recognition memory [see Ref. [10] for a meta-analysis]; nevertheless, the nature of this impairment is undetermined.

It is widely agreed that two retrieval processes support recognition memory judgments: recollection and familiarity [see Ref. [15] for a review]. Recollection reflects the retrieval of qualitative information about the study episode, such as where or when an event took place, or one's thoughts and feelings at the time. On the other hand, familiarity drives memory performance without any qualitative details coming to mind about where or when the item was encountered before. An extensive body of patient and neuroimaging research has focused on the role of the medial temporal lobes in recollection and familiarity [see Ref. [16] for review]. In the past 10 years, the role of the frontal lobes in recollection and familiarity has received growing attention in neuroimaging studies [Refs. [17–22]; see Ref. [23] for review], but there are only a handful of patient studies that address this issue. The consequence of frontal lobe lesions on recollection and familiarity, therefore, is not well established.

On theoretical grounds, there is good reason to think that the PFC may be important for both recollection and familiarity. Some indirect evidence comes from task comparisons, which show that source memory, which depends heavily on recollection, is impaired in PFC patients, while item memory, which can be supported largely by familiarity, is less impaired [e.g., Ref. [14]]. Moreover, recollection is often characterized as reflecting a controlled or strategic retrieval process, similar to that underlying free recall, whereas familiarity is thought to be a more automatic process. As such, one might predict that the frontal lobes are particularly critical for recollection [e.g., Refs. [24–28]]. On the other hand, familiarity is often characterized as a signal-detection retrieval process, which necessitates both an assessment of memory strength and a decision process, which involves setting response criteria for classifying

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doi:10.1016/j.bbr.2011.07.043

items as old or new [e.g., Refs. [29,30]]. Both of those components of familiarity assessment may depend on the monitoring and evaluation processes supported by the PFC [see Ref. [18]].

We focus here on lateral prefrontal cortex (LPFC), which is richly connected with regions in the medial temporal lobe [see Ref. [31]] and has been directly implicated in item recognition in numerous previous studies [see Ref. [10] for review]. There are only a handful of studies investigating recollection and familiarity in LPFC patients, and these studies have yielded conflicting results. Most of the studies have utilized the remember/know procedure [32] to estimate the contributions of recollection and familiarity. This procedure requires participants to introspect on their memory experience and report whether they consciously 'remember' studying an item, or merely 'know' that an item was studied, without any qualitative details coming to mind about the study event. Most recently, Kishiyama et al. [33] found that recollection and familiarity were reduced in LPFC patients, following intentional encoding of pictures. In contrast, Duarte et al. [34] found that LPFC patients were impaired in familiarity, but not recollection, and the deficit was restricted to pictures presented to the lesioned hemisphere. Left LPFC patients were additionally impaired at remembering the context which items were encoded, suggesting that an objective measure of recollection was impaired in these patients, although self-reports of recollection were intact. Finally, an earlier study found that LPFC patients were not impaired at 'remembering' or 'knowing', although 'know' responses were slightly reduced [35]. Familiarity estimates, however, were not reported.

Another way of estimating the contributions of recollection and familiarity to recognition memory is to use receiver operating characteristics (ROCs), and fit the dual process signal detection model to the data [36,37]. This method allows one to estimate recollection and familiarity without relying on the subjective reports of the remember/know technique. The only ROC study with frontal lobe patients found that LPFC patients were impaired at familiarity, but not recollection, for incidentally encoded pictures [38].

Thus, there are some studies suggesting that recollection and familiarity are both intact in LPFC patients [35], other studies indicating that familiarity is impaired but recollection is not [34,38], and yet other studies suggesting that both recollection and familiarity are impaired [[33,34] left PFC patients]. The lack of a consensus among these studies might arise from a number of factors. First, reliance on subjective reports of recollection and familiarity may be complicated by the metamemory deficits in frontal patients [39]. Of note, different measures of familiarity were used in different studies (i.e., 'know' responses versus independence remember/know estimates of familiarity, see Ref. [40]), complicating comparison across studies. Second, the use of intentional encoding conditions [33,34] may lead to different results than incidental encoding [[38]; see also Ref. [35]]. In the former, recognition impairments may be the result of impairments at strategic encoding as well as impairments at retrieval, whereas in the latter case, deficits are likely to be primarily at retrieval. Memory deficits in frontal patients are reduced when encoding conditions are incidental or constrained [e.g., Refs. [41,42]], so it is possible that this contributes to the discrepancies between studies. Finally, high levels of performance in the ROC study [38] complicate the interpretation of those results, because ceiling effects can reduce the reliability of the estimates of recollection and familiarity [15].

The aim of the current study was to use receiver operating characteristics to investigate whether recollection, familiarity, or both are impaired in patients with lesions to the lateral prefrontal cortex. Incidental encoding was used to reduce demands on strategic processing at encoding, and memory was examined at two levels of performance (following shallow and deep encoding) to determine if the memory deficit generalizes across memories of different strength.

1. Method

1.1. Participants

Thirteen patients with unilateral prefrontal cortex lesions (7 left, 6 right) (mean age = 63.4 years, SD = 12.1) and 26 age-matched control participants (mean age = 62.9 years, SD = 11.2) took part in the experiment. Patient characteristics and neuropsychological test scores are shown in Table 1. Each patient was yoked to two age-matched controls. The average age of patients and controls was not different, $t < 1$. However, left LPFC patients ($M = 57.7$ years, SD = 11.0, $n = 7$) were younger on average than right LPFC patients ($M = 70.0$ years, SD = 10.5, $n = 6$), and this difference approached significance, $t(11) = 2.06$, $p = .06$. The control groups for the left and right patients also differed in age (left control group $M = 57.5$ years, SD = 11.0, $n = 14$; right control group $M = 69.1$ years, SD = 9.2, $n = 12$), and this difference was significant, $t(24) = 3.04$, $p = .006$. For this reason, age was used as a covariate in initial analyses, but follow-up tests did not use age as a covariate if there were neither main effects of age nor any interactions. The education levels of the patients ($M = 15.7$ years, SD = 3.6) did not differ from that of the controls ($M = 14.7$ years, SD = 1.84; $t(32) = 1.07$, $p = .29$). Left and right hemisphere patients also did not differ in education, $t < 1$.

Patients were recruited from the Veteran's Administration Northern California Health Care System (VANCHCS) in Martinez, CA and other participating hospitals and clinics. Patients were included if they were at least 6 months post-cerebral vascular accident and had no history of any other medical, neurological or psychiatric disorder. None of the patients were aphasic. The lesions for all patients were the result of middle cerebral artery infarcts. The lesions were centered in the lateral PFC encompassing both dorsolateral PFC (DLPFC; Brodmann's areas (BA) 9 and 46) and ventrolateral PFC (VLPFC; BA 44, 45 and 47) sub-regions with varying degrees of damage in BA 6, 8, and 10. Note that every patient had damage to at least one DLPFC region (BA 9 and 46) and only four patients had damage that did not include at least one VLPFC region (BA 44, 45, and 47). Group lesion overlaps are shown in Fig. 1a for the left LPFC group and Fig. 1b for the right LPFC group. The control participants were recruited from the Davis, Sacramento, and San Francisco Bay Area communities, and they had no history of neurological or psychiatric disorders. Participants were paid for participation and signed consent statements approved by the Institutional Review Boards of the University of California, Davis, the University of California, Berkeley, and the Veterans Administration Research Service.

1.2. Materials

Four-hundred and eighty nouns, adjectives, and verbs were selected from the Toronto word pool. The words ranged from 3 to 7 letters in length ($M = 5.4$, SD = 1.3) and from 1 to 3 syllables. Kucera–Francis frequency [43] ranged from 11 to 39 ($M = 20.7$, SD = 8.3). The words were randomly divided into two sets to serve in sessions 1 and 2. Each set was randomly divided into three lists. List 1 served as the first study list, list 2 served as the second study list, and list 3 served as non-studied lure items. The test list consisted of a random mixture of all the items from the three lists.

1.3. Design and procedure

Four PFC patients were tested in two 1-h sessions, and the remaining nine patients participated in one 1-h session. Controls yoked to the patients were matched to the patients for number of trials. Participants heard one list of words under deep encoding conditions (i.e., make an abstract/concrete judgment about each word), and then a second list of words under shallow encoding conditions (i.e., count the number of syllables in each word). For participants who took part in two sessions, the order of the encoding conditions was reversed for the second session. Participants were then read a test list and were required to make recognition memory judgments using a 6-point confidence scale from 'certain it was new' (1) to 'certain it was old' (6). The study and test phases were participant-paced. The participants responded verbally and the experimenter recorded their responses. Participants were instructed to spread their responses across the whole range from 1 to 6, and were reminded of this after they made their first few responses. Pilot studies with healthy controls suggested that these instructions were necessary in order to avoid having participants use only high-confidence or low-confidence responses. Failure to use the entire range of response confidence would lead to ROCs in which the points were closely clustered together, thus making the assessment of the function difficult.

2. Results

The mean proportion of responses in each confidence bin are shown separately for each of the patient and control groups in Table 2. In order to estimate the contributions of recollection and familiarity to recognition performance, confidence responses were used to plot receiver operating characteristics for each patient and control. The proportion of correct recognitions (hits) was plotted

Table 1
Patient characteristics and neuropsychological test scores. WAIS-R scores are for the digit symbol subtest, number correct and incorrect in 90 s. Trail making test scores are times in seconds for Part A (numbers) and part B (switching). The Stroop test was a modified version from the Delis–Kaplan executive function system (D-KEFS, [74]). Patients had 60 s for each of the color naming and interference conditions. Stroop scores are color correct/color errors/interference correct/interference errors.

Patient	Hemisphere of lesion	Sex	Education	Age at test	WAIS-R: digit symbol	Trail making test	Stroop	WRAT-4: word reading
L1	Left	M	Unknown	70	N/A	N/A	N/A	N/A
L2	Left	M	Unknown	67	N/A	N/A	N/A	N/A
L3	Left	M	Ph.D.	46	56/0	27/83	44/0/42/0	44
L4	Left	M	11	63	15/0	54/193	38/5/22/0	21
L5	Left	F	14	65	27/0	45/172	27/0/17/0	36
L6	Left	F	DO (MD equivalent)	49	45/0	38/81	43/0/28/0	49
L7	Left	M	MBA	44	43/0	23/96	25/0/21/0	41
R1	Right	F	Unknown	79	N/A	N/A	N/A	N/A
R2	Right	F	Unknown	82	N/A	N/A	N/A	N/A
R3	Right	M	13	72	19/1	93/260	60/0/22/2	43
R4	Right	M	12	72	24/0	59/265	42/0/17/0	44
R5	Right	M	HS, 2.5 yrs college	59	41/1	35/139	115/2/53/2	36
R6	Right	F	Master's Degree	56	36/0	31/53	83/0/56/0	49

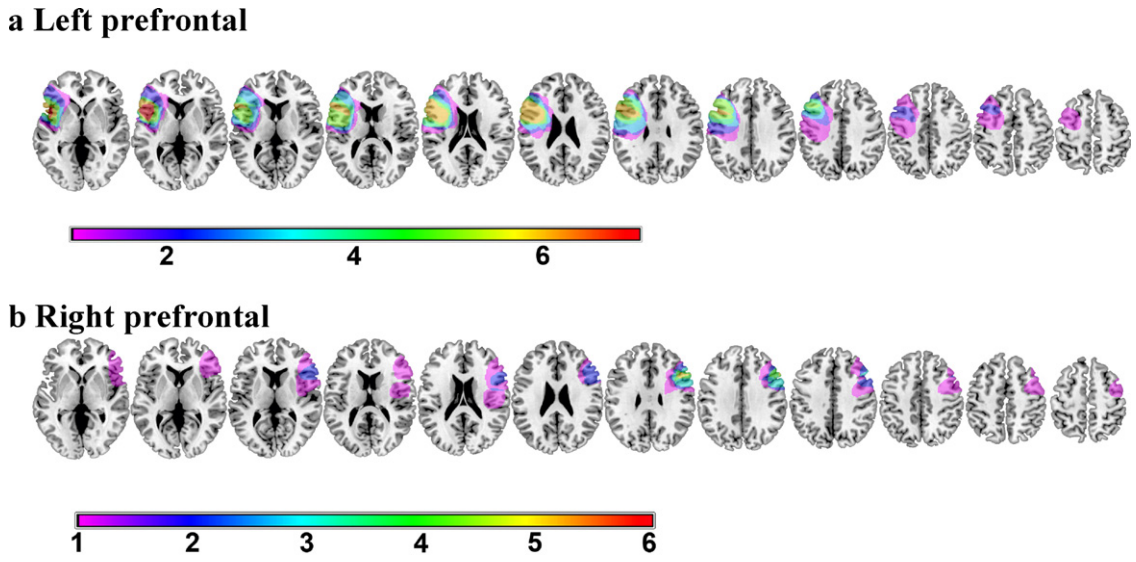

Fig. 1. Overlay of lesion reconstructions for the seven left (a) and six right (b) lateral prefrontal patients.

against the proportion of incorrect recognitions (false alarms) as a function of confidence, with the left-most point indicating the most confidently recognized items. The dual-process signal detection (DPSD) model was then fit to the observed ROCs to derive estimates of recollection and familiarity [36,37]. Recollection was measured as the *y*-intercept of the ROC, and familiarity as the degree of curvilinearity of the ROC. Aggregate ROCs and mean parameter estimates of recollection and familiarity for patients and

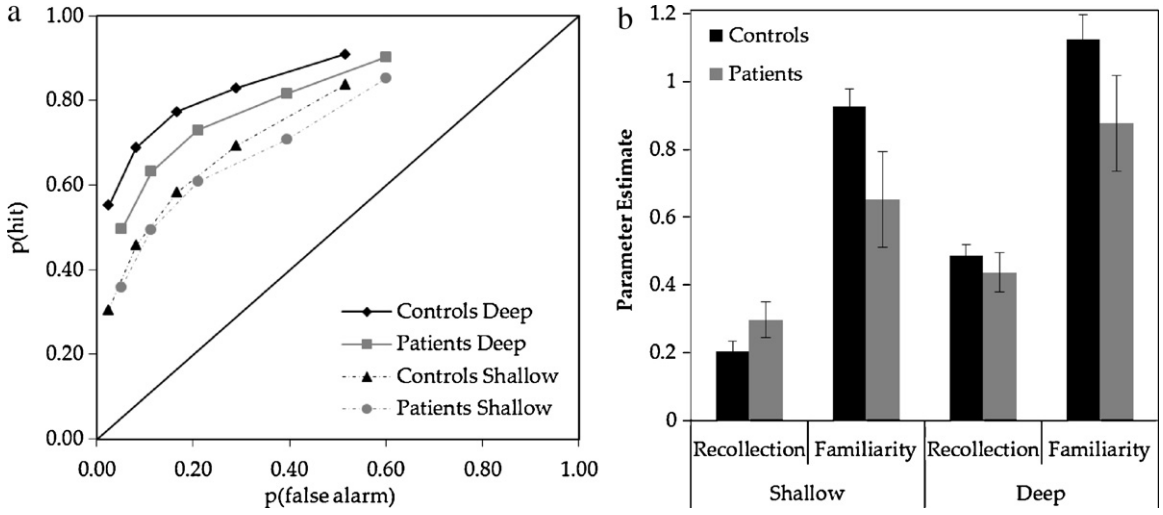

Fig. 2. Aggregate ROCs for the deep and shallow encoding conditions, for patients and controls (a). Average estimates of recollection and familiarity from the ROC analysis (b).

Table 2
Mean proportion of responses for each confidence bin, for new, old shallowly encoded items, and old deeply encoded items, separately for the left and right LPFC patients and their respective control groups. Standard deviations are in parentheses.

	New						Old (Shallow)						Old (Deep)					
	1	2	5	4	5	6	1	2	5	4	5	6	1	2	5	4	5	6
L Controls	.43 (.14)	.25 (.08)	.15 (.06)	.09 (.04)	.06 (.03)	.02 (.02)	.11 (.10)	.12 (.03)	.11 (.06)	.13 (.06)	.16 (.05)	.36 (.13)	.05 (.04)	.07 (.05)	.06 (.04)	.09 (.04)	.14 (.06)	.59 (.17)
LLPFC	.44 (.18)	.20 (.10)	.16 (.08)	.09 (.07)	.07 (.07)	.04 (.06)	.16 (.10)	.13 (.11)	.08 (.04)	.11 (.05)	.13 (.06)	.40 (.21)	.11 (.08)	.07 (.06)	.07 (.03)	.09 (.05)	.12 (.07)	.54 (.16)
R Controls	.55 (.20)	.19 (.11)	.09 (.09)	.08 (.04)	.06 (.05)	.03 (.03)	.23 (.11)	.17 (.10)	.10 (.06)	.12 (.05)	.15 (.08)	.23 (.14)	.14 (.07)	.08 (.08)	.05 (.04)	.08 (.04)	.14 (.05)	.51 (.14)
RLPFC	.35 (.22)	.21 (.11)	.22 (.11)	.10 (.08)	.06 (.03)	.07 (.10)	.13 (.09)	.16 (.10)	.12 (.05)	.12 (.05)	.14 (.07)	.32 (.20)	.09 (.05)	.10 (.09)	.11 (.07)	.11 (.08)	.15 (.11)	.44 (.30)

Table 3
Recollection and familiarity estimates in the shallow and deep encoding conditions for the left and right LPFC patients and their respective control groups.

	Shallow encoding		Deep encoding	
	Recollection	Familiarity	Recollection	Familiarity
L Controls	0.26(0.04)	1.02(0.08)	0.50(0.05)	1.28(0.11)
L LPFC	0.36(0.08)	0.71(0.21)	0.50(0.07)	0.95(0.18)
R Controls	0.14(0.04)	0.82(0.06)	0.47(0.04)	0.94(0.08)
R LPFC	0.22(0.05)	0.59(0.21)	0.37(0.10)	0.78(0.23)

controls are shown in Fig. 2 and in Table 3. In order to characterize the patients' performance, separate analyses of variance were conducted for recollection and familiarity, using the recollection and familiarity estimates for each patient and control.

2.1. Familiarity

In order to determine if left LPFC and right LPFC patients differed significantly in estimates of familiarity, patients were compared in the shallow and deep encoding conditions. Age was used as a covariate because of the age difference between the patient groups. The main effects of age and hemisphere were not significant, all *p*s > .22. Thus, left and right LPFC groups were collapsed in subsequent analyses and treated as a single patient group.

Familiarity estimates for each patient and control were entered into a 2 (group: patient or control) by 2 (levels of processing: deep or shallow) mixed model analysis of variance, with repeated measures on levels of processing. The ANOVA led to a main effect of group, $F(1,37) = 4.67$, $p = .037$, and a main effect of levels of processing, $F(1,37) = 13.16$, $p = .001$. The group by levels of processing interaction was not significant, $F < 1$. Follow-up planned *t*-tests confirmed that the patients were impaired at familiarity relative to controls in both the shallow, $t(37) = 2.22$, $p = .016$, and deep, $t(37) = 1.73$, $p = .046$ encoding conditions.

To see if there was a trend for a difference in the magnitude of impairment for the left and right LPFC patients, descriptive statistics for the left and right patient groups were compared to their respective control group. Estimates of familiarity in the shallow encoding condition were reduced by 30% and 28% in the left and right LPFC groups, respectively, compared to each of their age-matched control groups. In the deep encoding condition, estimates of familiarity were reduced by 25% for the left LPFC group and 17% for the right LPFC group. Thus, there was a slightly larger numerical impairment for the left LPFC group in the deep encoding condition, but there was no difference in impairments for the different patient groups in the shallow encoding condition, and no statistically significant difference between the left and right LPFC groups.

In order to further investigate the impairments, *z*-scores were calculated for each left- and right-LPFC patient relative to the respective left or right control group. The familiarity impairments for left- and right-LPFC patients were then compared with the non-parametric Mann–Whitney *U* test. The impairments were not different for the left and right LPFC patients ($U = 19$, $p = .84$ for shallow encoding and $U = 21$, $p = .99$ for deep encoding).

We are cautious about interpreting the lack of a difference between the left and right patient groups, due to the small sample sizes. With larger sample sizes, it is possible that differences would arise. Nevertheless, in the current experiment, the patients were impaired relative to controls on estimates of familiarity, and there was no evidence for differences in the degree of impairment.

2.2. Recollection

In order to determine if left and right LPFC patients could again be treated as a single patient group, estimates of recollection were

compared in the shallow and deep conditions, using age as a covariate. The main effects of age and hemisphere were not significant, all $ps > .21$. Left and right LPFC groups were therefore collapsed in subsequent analyses as a single patient group.

A 2 (group: patient or control) by 2 (levels of processing: deep or shallow) mixed model analysis of variance on estimates of recollection resulted in a main effect of levels of processing, $F(1,37) = 57.00$, $p < .0001$, and a group by levels of processing interaction, $F(1,37) = 6.47$, $p = .015$. There was no main effect of group, $F < 1$. The group by levels of processing interaction arose because the patients were numerically higher than controls for estimates of recollection following shallow encoding, but the controls were numerically higher than the patients for estimates of recollection following deep encoding. Neither of these differences was significant, however, $ps > .11$. Additionally, the higher recollection estimate for patients compared to controls in the shallow encoding condition was driven by a left LPFC patient who had a recollection estimate greater than 0.7, which is more than 3 standard deviations greater than the mean for controls. Given this, and the non-significant pair-wise comparisons, we are cautious about interpreting the interaction in the recollection estimates.

Thus, the patients were not impaired at recollection compared to controls, and left and right lesion patients were not significantly different.

2.3. Comparison of recollection and familiarity

To confirm that the LPFC patients were impaired at familiarity but not recollection, a 2 (process: recollection or familiarity) by 2 (group: patient or control) by 2 (levels of processing: shallow or deep) mixed-model analysis of variance was conducted on the parameter estimates from the ROC analyses, using age as a covariate. Since familiarity is measured in d' and recollection is measured as a probability, familiarity estimates were first converted into probabilities to eliminate the difference in scale. Although other methods are available to facilitate comparison of scores (e.g., conversion to z-scores or converting recollection to a d' value), this method was used for consistency with previous patient studies [e.g., Ref. [44]] as well as reviews of the recognition memory literature [15]. To obtain familiarity as a probability, the hit rate was found for each participant that would yield that participant's d' for familiarity, using the average false alarm rate for controls as the false alarm rate. The average false alarm rate was then subtracted from the participant's obtained hit rate to yield an estimate of familiarity as a probability. Note that the use of the average false alarm rate for controls is arbitrary and does not affect any group differences, since the same false alarm rate is used for all individuals, and it is subtracted out at the end to yield the familiarity estimates.

There was a main effect of levels of processing, $F(1,36) = 7.26$, $p = .011$, and no levels of processing by process interaction, $p = .18$, indicating that deeper processing had similar beneficial effects on recollection and familiarity. Most importantly, there was a significant group by process interaction, $F(1,36) = 4.30$, $p = .045$. This interaction arose because patients were significantly impaired relative to controls on familiarity, $t(37) = 2.21$, $p = .021$, but not recollection, $t < 1$. No other main effects or interactions were significant. These results therefore converge with the preceding analyses and confirm that LPFC patients were impaired at familiarity but not recollection.

2.4. Analyses based on d′

The ROC data could also be analysed using an equal variance signal detection approach, which characterizes performance based on a single parameter, d'. The ROC data was analysed using this approach to determine if similar conclusions would result. For each confidence point, d' was calculated, based on the hit and false alarm rate at that confidence level. In the deep encoding condition, patients were not significantly different from controls at the '6', '5', or '4' confidence levels, all $ps > .12$. They were significantly impaired, however, at the '3' and '2' confidence levels, $t(37) = 2.05$ and $t(37) = 2.02$ for the '3' and '2' points respectively, both $ps < .05$. A similar trend was observed in the shallow condition, but this did not reach significance ($p = .07$ and $p = .08$ for the '3' and '2' points, respectively, one-tailed). Inasmuch as both recollection and familiarity can lead to high confidence responses, while the lower confidence responses are based on familiarity [30] these analyses are consistent with the dual-process interpretation that familiarity, but not recollection, is impaired in frontal patients.

3. Discussion

In the past few decades, an extensive body of research has shown that the frontal lobes have important mnemonic functions that are distinct from the contributions of the medial temporal lobes. Specifically, the frontal lobes are important for organizing, evaluating, and monitoring the encoding and retrieval operations of the medial temporal lobes [see Ref. [45] for review]. As a result, damage to the frontal lobes leads to deficits on a variety of memory tasks that put demands on effortful or strategic encoding and/or retrieval [see Refs. [2,6,9] for review]. It was initially thought that performance on a non-strategic memory task, item recognition, was preserved in patients with PFC lesions. However, it became apparent that there was in fact a reliable recognition deficit in PFC patients [10]. What was unknown was whether the recognition impairment resulted from impaired recollection, impaired familiarity, or both.

Given the memory deficits in these patients on tasks that require controlled and effortful retrieval, a reasonable prediction is that recollection would be impaired. Further, inasmuch as familiarity assessments depend on one's ability to judge memory strength and set decision criteria for old/new decisions, it is also reasonable to predict that the frontal lobes play a role in familiarity. Indeed, neuroimaging evidence supports a role of the PFC in both recollection and familiarity [[17–20,22], see Refs. [21,23] for reviews]. With respect to familiarity, neuroimaging studies have found lateral PFC activation at both encoding [e.g., Ref. [20]] and retrieval [e.g., Ref. [22]] which tracks recognition confidence, suggesting this region is sensitive to familiarity. The handful of patient studies that investigated this issue, however, led to mixed results [33–35,38], and were complicated by a variety of factors, such as reliance on subjective reports [33–35], high levels of performance [38], or demands on effortful or strategic encoding [33–35]. In the current study, these issues were addressed by using an incidental encoding task, and analysing receiver operating characteristics to estimate the contributions of recollection and familiarity to recognition memory at two levels of performance. We found that LPFC patients' recognition memory was characterized by preserved recollection but impaired familiarity [see also Refs. [34,38]].

3.1. Assessing reasons for discrepant findings

Why has the prior literature been inconsistent with regard to recollection and familiarity impairments in frontal patients? A number of factors seem to contribute. First, to the extent that encoding conditions are demanding or intentional, leading control participants to utilize strategic processes at encoding to optimize performance, LPFC patients may exhibit deficits in both recollection and familiarity (e.g., intentional encoding, [33]; rapid visual presentations and intentional encoding, [34]). The role of the PFC in demanding encoding conditions is supported by two studies reporting reduced recollection and familiarity following rTMS

to the LPFC during encoding [46,47]. When experiments include demanding encoding conditions, the impairment in recollection may not be due to impaired retrieval of contextual information; instead, the impairment may be due to less effective encoding of information that can support recollection. When encoding conditions for patients and controls are incidental, however, deficits are found in familiarity, but not recollection ([38], and the current study).

Another factor that may affect the assessment of recollection and familiarity deficits is how one measures recollection and familiarity. A number of studies have relied on subjective reports using the remember/know procedure [33–35]. Although the remember/know procedure leads to converging results with other process estimation methods used to estimate recollection and familiarity [48] some evidence suggests that the remember/know distinction can be difficult for patients with memory deficits to understand [e.g., Ref. [49]]. Confidence judgments, and the ROC method, may therefore be a useful alternative for deriving recollection and familiarity estimates in patient populations.

A final consideration is that lesion location within the PFC is likely to be critical in determining whether recognition deficits are due to recollection impairments, familiarity impairments, or both. For example, the current results may seem at odds with a study which used ROCs to investigate recollection and familiarity in rats with prefrontal lesions [50] and found impaired recollection and spared familiarity. Importantly, the lesions in the rat study were to the medial PFC, not the lateral PFC as in the current study. The findings from the rat study are consistent with the Wheeler and Stuss [35] finding that patients with medial and polar frontal lesions showed only recollection impairments. Although it is possible that there are differences between the medial frontal regions in the rat and in humans, the results so far suggest that the medial and lateral regions may play different roles in recognition memory. The PFC is known to be a heterogeneous structure, and damage to different subregions of the PFC result in distinct profiles of deficits in attention, memory, and other cognitive functions [see Refs. [51–56]]. It would be surprising if damage to different subregions of the PFC had exactly the same effect on recognition memory. Even if the magnitude of recognition impairments was similar, the impairments may be due to qualitatively different reasons following damage to distinct subregions [see Ref. [52]].

Additionally, neuroimaging evidence suggests that distinct regions of PFC play roles in recollection and familiarity-based recognition [e.g., Refs. [20,22]]. In a review of the neuroimaging data, Skinner and Fernandes [21] proposed that the right dorsolateral PFC is involved in both recollection and familiarity, but recollection additionally involves bilateral anterior and superior frontal regions. Although we did not find that the memory deficits we observed differed for left compared to right hemisphere lesion patients, future studies with larger samples will be useful in assessing this possibility. In addition, future research will be necessary to elucidate whether lesions to distinct PFC subregions have differential effects on recollection and familiarity. For example, as discussed earlier, some evidence suggests that lesions to the frontal poles may impair recollection [35].

Recognition also appears to be disrupted in diseases that affect frontal lobe function, such as Parkinson's disease (PD). PD patients show cognitive deficits arising from fronto-striatal dysfunction. As with PFC patients, the findings with respect to recognition memory impairments in PD have been inconsistent, with some studies reporting sparing and others impairment of recognition memory [see Ref. [57] for a meta-analysis). It has become apparent that there is in fact a recognition deficit in PD, but subsequent research has yielded conflicting findings with respect to whether recollection [58] or familiarity [59,60] is impaired. A recent study found that different encoding tasks lead to either selective familiarity or selective recollection deficits in PD [61]. Thus, as with frontal patients with damage to different regions of the PFC, there may be heterogeneity in recognition memory impairments in PD.

How would the current results be interpreted if examined using a single-process strength model of recognition [e.g., [62], see Ref. [63]]? Single-process models propose that recognition memory is based on a continuous strength signal, as opposed to independent processes of recollection and familiarity. In order to determine how the current findings are accounted for by such a model, we fit the aggregate data to the unequal-variance signal-detection model [62]. This model assumes that recognition memory performance is based on an assessment of overall memory strength, and includes two functionally dissociable parameters: strength (d') and the variance of old items (V_o). We found that V_o was unaffected in the PFC patients (V_o = 1.49 and 1.52 for the patients and controls, respectively), but d' was reduced (d' = 1.42 and 1.67 for the patients and controls, respectively). Thus, both in terms of the dual-process approach and the UVSD single-process approach, one memory process or parameter (recollection, or variance) was unaffected in PFC patients, but the other memory process or parameter (familiarity, or strength) was reduced. So the main conclusions of the current study are not fundamentally different from these two theoretical perspectives.

Frontal lobe lesions often lead to abnormally high false alarms to new items, while hit rates are not necessarily affected [64–68]. Similar to these results in humans, the rat ROC study mentioned previously [50] found that frontal lesions increased false alarms but not hit rates. In the current study, patients consistently showed increased false alarms relative to controls (evidenced by ROCs that are shifted to the right, see Fig. 2a), but they also had lower hit rates for the deep encoding condition relative to controls (evidenced by ROCs that were shifted down), particularly at the higher confidence levels. Thus, while the current study provides additional evidence that false alarm rates are increased in frontal patients, the impairment was not due only to increased false alarms.

Note that the difference in high-confidence responses in patients and controls does not imply that recollection is poorer in patients than controls. Recollection estimates depend on the shape of the entire ROC, not just the hit and false alarm rates at the high-confidence responses. High-confidence responses may include some high-confidence familiarity as well as recollection [30], and familiarity may increase false alarms at the high-confidence points compared to when only recollection contributes to high-confidence responses. Increased false alarm rates at the high-confidence points is therefore consistent with a familiarity impairment in patients, but the raw proportions are less diagnostic than familiarity estimates since they can be influenced by differences in response bias.

Our findings of spared recollection in LPFC patients appears to be inconsistent with the literature demonstrating that PFC patients are impaired at source memory, which relies heavily on recollection. One possible account of these results is that the source memory impairments may arise because of the increased monitoring demands in source memory tests. That is, in a standard item recognition task, individuals must make binary old/new judgments at test. In contrast, in a typical source memory test, individuals must first discriminate between old and new items and then discriminate between items from two different encoding sources. These additional monitoring demands may exceed the strategic abilities of patients with frontal lobe damage. The familiarity deficits in the current study may arise because of the more complex monitoring demands placed in the item recognition task used here, in which individuals were required to set criteria to respond at different levels of confidence, and thus must make careful discriminations between items of different levels of familiarity.

3.2. Familiarity and PFC-mediated control

Cognitive neuroscience research on the PFC has shown that this region is involved in many aspects of cognitive control [see Ref. [69] for review]. Since recollection is often characterized as a controlled or strategic retrieval process, whereas familiarity is thought to be a more automatic process [see Ref. [15] for review], the current results suggest that recollection and familiarity cannot be cleanly fractionated along lines of controlled versus automatic processes. Recollection involves both controlled (e.g., strategic search of memory) and automatic (e.g., irrelevant or noncriterial recollection, see Ref. [70]) processes. Similarly, familiarity involves both controlled (e.g., setting response criteria, evaluating weak memories, see Ref. [29,30]) and automatic (e.g., processing fluency, see Ref. [71]) processes.

Familiarity may be particularly impaired in frontal patients in item recognition tasks because the familiarity signal is inherently more ambiguous and thus requires more careful retrieval monitoring than does recollection. Retrieval of qualitative information about the study event is unambiguous in the sense that any recollected detail about the study episode is diagnostic of the item having been studied. In contrast, an item may be familiar because it has been studied or it may be familiar from pre-experimental exposure to that item or some aspect of that item (this is particularly the case for words, which have been encountered frequently and/or recently before the experiment). Moreover, the frontal lobes may be particularly important in evaluating weaker familiarity signals (i.e., this item is familiar because it was studied, or this item is familiar but not from the study phase). In line with this hypothesis, the DLPFC is more active for correct low-confidence recognition judgments than correct high-confidence judgments, suggesting a role of this region in monitoring retrieval of weaker memories [18].

Alternatively, the familiarity impairment might arise from a difficulty in assessing the strength of memories for which detailed information is not available, or an impairment in the decision process involved in setting the criteria to respond at different confidence levels. Both of these interpretations suggest that some degree of monitoring and evaluation is necessary for accurate familiarity-based discrimination, processes known to be impaired in PFC patients.

In support of this idea, one account of medial temporal/prefrontal lobe interactions in memory proposes that hippocampal retrieval is obligatory in the sense that it is automatic and reflexive once an environmental cue is apprehended, but the prefrontal cortex is needed to organize or strategically work with the hippocampal output [3,72]. In item recognition, environmental cues to memory are present and readily apparent, and there are few demands on strategic retrieval or organization. In this case, LPFC patients might show preserved recollection because the hippocampal output is intact, and there is little need for strategic retrieval processes.

This raises the point that deficits in recollection on strategic memory tasks may not necessarily be associated with recollection deficits on non-strategic memory tasks such as item recognition for incidentally encoded stimuli. On the other hand, the monitoring or evaluation of weaker or ambiguous memory signals may depend on intact frontal function, and have an impact on performance on familiarity-based item recognition. The critical point is that recollection *in general* or familiarity *in general* may not be impaired in frontal patients, but that different types of deficits may emerge on different types of memory tests.

Finally, the current findings have implications for the interpretation of recollection and familiarity deficits in medial temporal lobe amnesia. It has been proposed [e.g., Ref. [26]] that recollection deficits in amnesia are not a result of selective MTL damage, but arise from additional prefrontal dysfunction. The current results suggest that the recollection deficits in MTL amnesia are more likely due to the MTL damage (particularly the hippocampus, see Ref. [73] for review) than prefrontal damage, as at least lateral prefrontal dysfunction would be expected to reduce familiarity, and not recollection.

4. Conclusions

Decades of research have established the critical role of the prefrontal cortex in memory, and the impairments following frontal lesions have been better characterized in recent years. The focus has been on the role of the PFC in strategic and effortful control of encoding and retrieval, leading many to propose that the PFC is critical for recollection. It is now apparent that this is not always the case. At least for item recognition, the lateral prefrontal cortex is necessary for familiarity, but not recollection.

Acknowledgments

NIH grant MH59352 to A.P. Yonelinas.
NINDS grant NS21135 to R.T. Knight.
We thank Donatella Scabini for patient recruitment, Jessica Black for help with the patient neuropsychological tests and Clay Clayworth for help with the patient scans and the lesion overlay figure.

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