Brain and Cognition 75 (2011) 111–118

Contents lists available at ScienceDirect

Brain and Cognition

journal homepage: www.elsevier.com/locate/b&c

False recognition in Lewy-body disease and frontotemporal dementia

C. de Boysson [a,b], S. Belleville [a,b,*], N.A. Phillips [c], E.K. Johns [c], D. Goupil [b], C. Souchay [d], R. Bouchard [e], H. Chertkow [f]

[a] Université de Montréal, Montréal, Canada
[b] Centre de recherche de l'Institut universitaire de gériatrie de Montréal, Montréal, Canada
[c] Department of Psychology/Centre for Research in Human Development, Concordia University, Montreal, Canada
[d] Leeds Memory Group, Institute of Psychological Sciences, University of Leeds, Leeds, UK
[e] Hôpital de l'Enfant-Jésus du CHA de Québec, Montréal, Canada
[f] Lady Davis Institute for Medical Research, Jewish General Hospital, McGill University, Montreal, Canada

ARTICLE INFO

Article history:
Accepted 27 October 2010
Available online 20 November 2010

Keywords:
Frontotemporal dementia
Lewy-body disease
False recognition
DRM
Memory

ABSTRACT

The primary goal of this study was to evaluate the false recognition phenomenon in persons with frontotemporal dementia (FTD) and those with Lewy-body disease (LBD). Patients with LBD ($n = 10$) or FTD ($n = 15$) and their corresponding controls ($n = 30$) were subjected to the Deese–Roediger–McDermott (DRM) paradigm to induce false recognition. Patients were first presented with items semantically related to a nonpresented critical target. The critical target was later included in a word list shown to patients to assess level of recognition. Both groups of patients showed a reduced level of false recognition of the critical target when controlling for their overall level of false alarms. This reduction was greater in persons with LBD than in those with FTD. Correlational analyses of performance on neuropsychological tests and the DRM variables indicated that the reduced DRM effect was associated with inhibition deficits in patients with LBD and with inhibition deficits and verbal memory in those with FTD. Our results support current models suggesting that these cognitive components contribute to the false recognition effect.

1. Introduction

The reconstructive nature of memory is now a well-recognized phenomenon. Not only do people forget events, but they also reconstruct and create new ones on the basis of their fragmented memories. This inherent characteristic of typical memory is known as false memory (Anderson, 1981; Krantz, Luce, & Tversky, 1971). There is ample evidence to suggest that the phenomenon of false memory is robust in healthy young adults who show a high level of false recognition in paradigms that promote memory reconstruction. The effect is modified in Alzheimer's disease (AD) (Budson et al., 2002), with patients showing a reduced level of false recognition compared with healthy controls. However, nothing is known about the false recognition effect in forms of age-related dementia other than AD. Therefore, the goal of this study was to fill this gap by studying false memory in patients with frontotemporal dementia (FTD) and Lewy-body disease (LBD) while attempting to elucidate whether other cognitive processes contribute to possible false memory modifications in these two types of dementia. False memory can be studied through different paradigms. Here, we focus on the Deese–Roediger–McDermott (DRM) false recognition paradigm in which instances of false recognition are induced (Roediger & McDermott, 1995). In this paper, we first explore current models of false memory, along with their empirical support, in an attempt to predict the nature and source of false memories in dementia. We then present a brief overview of the empirical work that has been carried out on AD and a brief description of the dementia typical of LBD and FTD.

1.1. Theories of the level of false recognition

The Deese–Roediger–McDermott paradigm involves the presentation of lists of words that are all related to a nonpresented lure (e.g., presented words: *hot*, *wet*, *ice*, *winter*, etc.; nonpresented lure: *cold*). When participants are later tested for their recognition of the lure, they show a high level of false recognition and report, incorrectly, having learned the nonpresented lure. Different theories have been proposed to account for this robust phenomenon, with each implying that different cognitive components contribute to the effect. The fuzzy-trace theory (Brainerd & Reyna, 1998; Reyna & Brainerd, 1992, 1995) suggests that presenting a list of semantically related words induces the memorization not only of specific

* Corresponding author. Address: Research Center, Institut universitaire de gériatrie de Montréal, 4565 Queen Mary Road, Montreal, Quebec, Canada H3W 1W5. Fax: +1 514 340 3548.
E-mail address: sylvie.belleville@umontreal.ca (S. Belleville).

0278-2626/$ - see front matter © 2010 Elsevier Inc. All rights reserved.
doi:10.1016/j.bandc.2010.10.011

characteristics of each word (representing the item-specific information) but also of the common characteristics of the words (assumed to be a general gist of the information or the general idea conveyed by the list). Because the critical lure represents the general idea of the list, it seems familiar to the participants and is thus falsely recognized. Empirically, the effect is partly due to the fact that the paradigm involves numerous word lists, which reduces the veridical memory of each item. In this view, the false memory effect is dependent on a competition between episodic memory and semantic memory. By contrast, some investigators have proposed that episodic memory is necessary for participants to encode and memorize the general meaning of the list of related words (Hudon et al., 2006; Verfaellie, Schacter, & Cook, 2002). This view is consistent with findings that indicate that memory-impaired patients exhibit both reduced veridical memory and false memory when correcting for level of false alarms (in AD, Hudon et al., 2006; in amnesic patients, Verfaellie et al., 2002). The activation/monitoring theory (McDermott & Watson, 2001; Roediger & McDermott, 2000) suggests that the false memory effect reflects an automatic process, since all words on the list are part of the same semantic network (Collins & Loftus, 1975). Further, because a similar network is activated by each word in the list, the critical lure is also activated and becomes over-activated as more of the list is presented. In this view, the false recognition effect would depend on the spread of activation within the semantic network. Finally, there is some empirical evidence suggesting that executive functions may contribute to the reduction of the false recognition effect (Budson et al., 2002; Butler, McDaniel, Dornburg, Price, & Roediger, 2004). Budson et al. (2002) showed that, across repetitive trials, patients with frontal lobe lesions displayed increases in their false recognition performances, whereas the controls were able to reduce them. These results suggest that the increasing level of false recognition across trials resulted from an impairment of frontal lobe functions.

To summarize, current accounts of the false recognition (clFR) effect suggest that the phenomenon depends on a balance between episodic memory (hit rate) and semantic memory and that executive functions may contribute to the FR effect by monitoring the balance between these memory processes. Age-related neurodegenerative disorders impair the aforementioned cognitive processes, and this impairment should result in a predictable impact on the nature and the extent of the false memory effect as discussed below.

1.2. Alzheimer's disease

The majority of research on false recognition and dementia has been conducted on individuals suffering from AD. Given that patients with dementia are prone to false alarms (by recognizing unpresented unrelated distractors), the most recent studies of these populations have used corrected scores to control for patients' tendency to produce false alarms. This measure is critical because the presence of a positive response bias will artificially increase the level of recognition, both true and false. When controlling for false alarms, Hudon et al. (2006) reported a reduced false recognition effect in patients with AD compared with the control subjects, which was interpreted as resulting from an impaired ability to memorize the general gist of a word list. On the other hand, Watson, Balota, and Sergent-Marshall (2001), who did not control for the level of false alarms, reported comparable levels of false recognition for nonstudied critical lures in AD relative to healthy aging. Budson et al. (2002) later reported data that could reconcile the two different findings. They reported that the level of false recognition after a single list exposure was lower in AD patients than in controls, but that, across repeated trials, veridical recognition increased and false recognition decreased in controls, whereas false and veridical recognition increased to the same degree in AD patients. The authors suggested that episodic memory serves to reduce the false recognition effect in healthy controls. Because of their impaired memory, this would not be the case for AD patients who would rely on semantic meaning, which would be reinforced across repeated trials.

Overall, evidence supports that false recognition is reduced in AD. Studies have suggested that this reduction could arise from patients' inability to extract and/or memorize the essence of the presented list. Even though executive deficits are present in dementia (Collette, Delrue, Van der linden, & Salmon, 2001; Lambon, Patterson, Graham, Dawson, & Hodges, 2003), their contribution to the reduced false recognition effect in AD is unclear, as it has not been measured directly.

1.3. Lewy-body disease and frontotemporal dementia

Lewy-body disease (see McKeith et al. (2004) for a review; Dieudonné, Marquis, Ergis, & Verny, 2006) is associated with abnormal aggregates of Lewy bodies in the limbic system and neocortical regions. Patients with LBD are known to have parkinsonian motor disturbances and visual hallucinations, with deficits on attention-based tests and in their visuo–spatial ability (Collerton, Burn, McKeith, & O'Brien, 2003; McKeith, 2005; Walker, Ayre, & Cummings, 2000). In addition, in the early stages of the disease, patients with LBD show significant impairment on tests of executive function (Calderon et al., 2001). In a comparison study, Downes et al. (1998–1999) studied patients with LBD and Parkinson's disease (PD) who had similar levels of dementia. The authors observed that, in seven out of eight tasks, LBD patients were significantly more impaired than PD patients, which indicates an important frontal dysfunction. Metzler-Baddeley (2007) conducted a review on LBD patients in comparison with AD and PD patients. Her review shows that LBD patients are generally more impaired than AD patients on visual-perception and construction tasks. Patients with LBD also present deficits in attention and executive functions that appear more severe and more pervasive than those exhibited in AD. Memory impairment is equivalent or more severe in LBD than in AD, though interpretation is complicated by the potential contribution of executive function and attentional deficits to performance in attention-demanding memory tasks.

Frontotemporal dementia describes patients presenting with atrophy of the frontal and temporal lobes associated with the presence of Pick bodies (Neary & Snowden, 1996) or tau pathology (Kirshner, 2010). The condition is a type of frontotemporal lobar degeneration (FTLD), which also includes semantic dementia and nonfluent progressive aphasia. Patients with FTLD present with atrophy of both frontal and temporal regions of the brain. Frontotemporal dementia has also been referred to as a behavioral variant of FTLD, and three different variants of FTD have been identified: a behavioral variant, a frontal variant, and a semantic/progressive aphasia variant. Patients with FTD very often suffer from both semantic and executive deficits (Neary & Snowden, 1996), but they are classified as suffering from the behavioral, frontal, or progressive aphasia variant depending on which deficit prevails (Josephs et al., 2009; Kirshner, 2010). Often, the pathology extends beyond the frontal and temporal lobes, and additional symptoms may be found (Graff-Radford & Woodruff, 2007). In the research cohort we studied, most FTD patients were classified as suffering from the frontal variant based on initial clinical symptoms. In this variant, patients suffer from executive dysfunction (Johns et al., 2009) but often show semantic deficits as well, particularly as the disease progresses (Kertesz, McMonagle, Blair, Davidson, & Munoz, 2005; Lezak, Howieson, & Loring, 2004). Thus, patients with FTD have executive deficits and, to a lesser degree, semantic deficits. Although patients with LBD and FTD are known

to suffer from executive and semantic deficits, no study has assessed the false recognition effect in these clinical populations.

1.4. Objectives

One of the general objectives of this study was to contribute to a better understanding of the similarities and differences between the memory deficits that characterize different age-related neurodegenerative disorders. The false recognition effect has been studied extensively in AD patients; therefore, the goal of this study was to examine the false recognition effect in persons with other types of dementia: Lewy-body disease and frontotemporal dementia. Accordingly, we relied on the classical semantic Deese–Roediger–McDermott paradigm in which participants are asked to learn lists of items that are semantically related to a nonpresented critical target. To favor encoding and to control for the possibility of a floor effect in memory-impaired patients, we provided participants with two study trials, both comprising a study phase and a recognition phase. We measured several variables: hit rate (HR; correct recognition of the previously learned words), false alarms (unFA; positive answers for unrelated lures), false recognition of weakly-related lures (wrFR; positive answers to lures weakly related to the list), and false recognition of critical lure (clFR; positive answers to the item highly related to the list). The scores for HR, wrFR, and clFR were statistically controlled for the overall level of false alarms to nonpresented unrelated lures (unFA) because the presence of a response bias toward positive responses, which would result in a high level of false alarms to all lures, would also artificially inflate the level of false recognition of the critical lure.

Because LBD and FTD patients are demented and thus suffer from a range of cognitive impairments, a second goal was to assess whether other cognitive deficits were associated with and/or contributed to the false recognition effect. Such knowledge is important because clFR has been noted to depend on a number of cognitive systems including episodic memory, semantic memory, and executive functions. The clFR scores were therefore correlated with neuropsychological measures of naming, verbal memory, and executive functions. It was expected that, in LBD, the impaired level of clFR would be positively correlated with impairment on the executive task, but that, in FTD, it would be correlated with the executive and/or naming deficit. To evaluate the aforementioned relations, the hit rate (correct recognition of the learned items) and false alarms to unrelated lures were also correlated with neuropsychological measures of these cognitive components.

2. Materials and methods

2.1. Participants

This study was part of a larger research program supported by the *Fonds de la recherche en santé du Québec* (FRSQ) and the Quebec Research Network on Aging (Cognition axis). The goal was to create a patient registry for memory impairment across the province of Quebec, Canada. Patients from the registry were recruited from different memory clinics and academic clinical centers within Quebec. To be included in the registry, patients had to undergo a common assessment protocol of clinical, cognitive, and imaging measurements. This study used the registry data collected from patients who suffer from FTD or LBD. The data presented here include the results for 10 patients with LBD and 15 with FTD, 11 of whom had frontal alterations (frontal variant or progressive nonfluent aphasia, PNFA). Participating physicians met with each patient as part of their standard clinical assessment. During this first appointment, physicians administered physical and mental status evaluations, including the Mini-Mental State Examination (MMSE) (Folstein, Folstein, & McHugh, 1975), to all participants and the Montreal Cognitive Assessment (MoCA) (Nasreddine et al., 2005), to the control group. Patients were then seen over two sessions for a more thorough neuropsychological assessment that included tests of memory (Grober and Buschke test: Grober & Buschke, 1987), language (Boston Naming Test: Kaplan, Goodglass, & Weintraub, 1983), and executive functions (Stroop Victoria test: Spreen & Strauss, 1998; Hayling test: Burgess & Shallice, 1997). Subjects with FTD met the Neary, Snowden, and Mann (2005) diagnosis criteria, and subjects with LBD met the McKeith et al. (2004) diagnosis criteria.

A group of healthy older adults were used as control participants. They were recruited using poster advertising and through visits to senior centers in the same communities as the patients. They were matched for age to each group of patients.

Exclusion criteria are as follows and were applied to all participants: other serious health problems, chronic psychiatric disorders as well as other classic excluding dysfunctions (cerebrovascular disease, head trauma, cerebral infection, metabolic dysfunction, thyroid dysfunction, B12/folic deficiency, epilepsy psychosis, schizophrenia, intoxication or alcohol abuse). Information on the patients' medical condition was obtained during the physical examinations and, in the case of the control participants, from a self-reported questionnaire. In addition to the general exclusion criteria, healthy older adults who fell below the MMSE cut-offs (adjusted for age and education) for dementia were also excluded.

Participants were either francophone or anglophone, since both languages are used in Quebec. They were tested in their primary language (the proportion of primary language subjects in each group is shown in Table 1). Patients were tested in the referral clinic. Healthy controls were tested either at Concordia University (anglophones) or at the *Institut universitaire de gériatrie de Montréal* (francophones).

Informed consent was obtained for all participants either from themselves or a family member. Ethical approval for this study was obtained by each participating clinic or institute.

2.2. Deese–Roediger–McDermott task

Twelve study lists of 12 semantically related words were created for the study phase. Lists were taken from Roediger and McDermott (1999) for the English version and from Belleville, Caza, and Peretz (2003) and Hudon et al. (2006) for the French version. Each list comprised words semantically related to a critical target. The list corresponding to each target was obtained by taking the first 12 semantic associates of the target listed by word association. The words on each list were presented in decreasing order of association, starting with the word most closely associated with the target.

Table 1
Demographic and clinical variables in frontotemporal dementia (FTD), Lewy-body disease (LBD), and controls.

Variable	Controls		LBD		FTD		Group effect
	M	*SE*	*M*	*SE*	*M*	*SE*	significance level
N	30		10		15		
Age	72.00	0.99	73.9	1.95	67.86	1.99	$p = .044$
Education	13.20	0.42	9.7*	1.12	10.93	0.93	$p = .003$
Sex[a]	40	–	70	–	66.67	–	n.s.
Language[b]	60	–	80	–	66.67	–	n.s.
MMSE	29.03	0.55	23.7*	1.00	24.28*	0.80	$p < .001$
MOCA	27.07	0.35	–	–	–	–	–

Note 1: MMSE = Mini-Mental State Examination; MOCA = Montreal Cognitive Assessment.

[a] Sex is given as percent male.

[b] Language is given as percent French.

* $p < .05$ for pairwise comparisons; relative to controls.

For the recognition phase, a single list of 36 studied and 36 nonstudied items was given to the participants. The 36 studied items consisted of three words from each of the 12 presented lists. Since the words on each list were classified according to the degree of their semantic link to the critical target, we chose words in the same position on each of the list. For an example referring to the target word *cold*, the word in the first position would be *hot*, the word in the sixth position would be *wet*, and the word in the eight position would be *chilly*. The 36 nonstudied items were of three types: 12 critical targets (e.g., *cold*), 12 weakly related words (e.g., *frost*), and 12 unrelated words (e.g., *suit*). The weakly related items of the semantic condition were taken from each association list and situated in position 13 of the presented list (an even weaker link than the presented words). The unrelated words were not related semantically to any of the critical targets. The 72 selected items were divided into two sets that were used for two recognition trials. Each set comprised 18 studied items, six critical targets, six weakly related nonstudied items, and six unrelated nonstudied items.

To promote encoding, the paradigm consisted of two study trials. In Trial 1, participants were instructed to listen carefully to the 12 lists of words and told that their memory would be tested subsequently. The lists were recorded on a CD by a female voice and played to the participants. Words were read at a rate of about 1.5 s per word. The presentation of the 12 lists was followed by an interference task lasting 30 s during which participants were asked to count backward starting from 100. Then, the recognition phase of Trial 1 began using one recognition set. Participants were instructed to decide whether the words they had heard were studied or unstudied items. Recognition lists were recorded on a CD by the same female voice used for encoding. Responses were given verbally and were recorded by the examiner. There was no time limit for responding to each word of the recognition phase. Trial 2 began immediately after the recognition phase of Trial 1 using the other recognition set. In this trial, participants were presented with the same 12 study lists, which were once again followed by the interference task. The participant's recognition was tested using the second recognition set to avoid testing two presentations of lures during the recognition phase as well as to minimize study-test sources of confusion (Budson et al., 2002). The order in which the two recognition sets were presented was counterbalanced across participants.

Five variables were analyzed: (1) the percentage of positive responses to items that were previously learned (HR); (2) the percentage of positive responses to a word that was not presented and that bore no relation to the list (unFA); (3) hit rate corrected by the level of false alarms (HR-unFA); (4) the percentage of positive responses to the critical target, that is, to a word that was not presented but that was highly related to the theme of the list (clFR); and (5) the percentage of positive responses to a word that was not presented but that was weakly related to the theme of the lists (wrFR).

2.3. Neuropsychological measures of semantic memory, episodic memory, and executive functions

To assess the relationship between performance on the DRM paradigm and cognitive functions, patients were tested with neuropsychological tests reflecting semantic knowledge, executive functions, and episodic memory.

The 15-item version of the Boston Naming Test (Kaplan et al., 1983) is associated with verbal ability and often used in dementia assessment as a sensitive indicator of semantic integrity (Goodglass, Kaplan, & Barresi, 2001; Kaplan et al., 1983). In the short version of the Boston Naming Test, participants are asked to name aloud a subset of 15 line drawings of common objects (fruits and vegetables or animals). The short version is part of the Consortium to Establish a Registry for Alzheimer's Disease (CERAD) battery, which is used to assess patients with AD (Lezak et al., 2004). The score is based on the number of items named correctly without cues. The test has been shown to be a valid and reliable measure of naming capacities in brain-damaged patients (Lezak et al., 2004). The French version has been validated by Thuillard Colombo and Assal (1992).

The Hayling test (Burgess & Shallice, 1997, for the English version; Belleville, Rouleau, & Van der Linden, 2006, for the French version) reflects frontal lobe function, particularly the inhibition of potent semantic responses. In the Hayling test, sentences missing their last word (e.g., "The prisoners escaped from the…") are read aloud to patients, who must then complete the sentence. In the automatic condition, patients are asked to provide the correct missing word (in the above example, *prison*). In the inhibition condition, they are asked to provide not the appropriate word but one that is completely irrelevant to the context (in the above example, e.g., *chocolate*). In both conditions, the response time for each sentence is measured with a stopwatch and recorded. In this study, the total time taken to complete the sentences in each condition was used as the main dependent variable for the correlational analyses. We also recorded and ranked errors in the inhibition condition, which were of two types: Related errors (type A errors), words that fit perfectly with the meaning of the sentence (e.g., *prison* in the previous example), are assigned three points. Somewhat-related errors (type B errors), words linked to the semantic context of the sentence (e.g., *police* in the previous example), are assigned one point. Correct answers are words completely unrelated to the sentence and are assigned zero points. An overall score is computed by adding up all the scores obtained in the Inhibition condition.

The Stroop Victoria test (Spreen & Strauss, 1998) is a well-known test that gives a reliable assessment of executive functions, particularly of inhibition. In this test, participants are asked to name the color of 24 items (four different colors) printed on glossy paper arranged in three different sets. In the first set (Dots condition), participants have to identify the color of the printed dots; in the second set (Words condition), they name the color of a given word on the page (e.g., "*when*" printed in blue; answer = blue), and in the last set (Color condition), they identify the color in which the name of a color is printed (a noncongruent color, e.g., the word "*red*" printed in blue; answer = blue). For this test, a score was computed by subtracting the variable for the Words condition from those for the Color condition to identify the effect of inhibition (Color–Words score).

Finally, the Buschke Free and Cued Recall test (Grober & Buschke, 1987) was used to diagnose verbal episodic memory deficits. In this test, participants must memorize a set of 16 pictures with and without semantic retrieval cues. Pictures are visually presented four at a time on a sheet of paper, and encoding is guided by the examiner who provides patients with semantic orientation cues (e.g., if the image to be encoded was a shoe, the examiner would ask the patient to indicate and name "the piece of clothing"). This step is followed by immediate cued retrieval of the four images. When participants successfully encode and retrieve all 16 pictures, they are asked to count backwards for 20 s. They are then asked to recall all the presented pictures in any order (free recall) and then with a given cue (cued recall) for missed items. The same procedure (interference task, free recall, and cued recall) was repeated for three other trials. The dependent variable used here is the cued recall of the fourth trial.

3. Results

3.1. Demographic data and preliminary analyses

Demographic data are presented in Table 1. A chi-square analysis indicated that the groups were comparable in terms of

distribution of gender, $\chi^2 = 4.316$, $p = .116$, and language, $\chi^2 = 1.340$, $p = .512$. A one-way analysis of variance (ANOVA) indicated a main effect of Age, $F(2, 54) = 3.326$, $p = .044$, and a main effect of Education, $F(2, 54) = 6.590$, $p = .003$. Bonferroni post hoc mean comparisons indicated that persons with LBD were less educated than healthy controls. However, when comparing groups on Age, Bonferroni post hoc mean comparisons did not reveal significant differences in spite of a general Age effect. Because of overall group effects on Age and Education, all analyses were run with the two factors as covariates to assess if the general Age and Education effects modified the outcomes.

Statistical analyses were also computed on scores on the MMSE to assess the disease severity of the two groups of patients. Results indicated a significant group effect on the MMSE, $F(2, 53) = 18.132$, $p < .001$; Bonferroni post hoc analysis indicated that the two groups of patients were significantly different from the controls, although they were not different from each other.

Table 2 shows statistics for the neuropsychological tests evaluating the different cognitive functions proposed to be involved in the false recognition effect: the Boston Naming Test, the Hayling test, the Stroop Victoria test, and the Grober and Buschke test. Data indicate that both groups of patients were impaired when compared with controls on the Boston Naming and the Grober and Buschke measures. Only FTD patients were impaired relative to controls on the automatic condition of the Hayling test ($p < .001$), and both FTD ($p = .02$) and LBD ($p < .001$) patients were impaired in the inhibition condition. When completion time in the inhibition condition was examined, while controlling for completion time in the automatic condition with an analysis of covariance (ANCOVA), only the LBD group was different from controls ($p < .01$). On the Stroop Victoria test (Color–Words score), both groups of patients differed from controls only in terms of the number of errors ($p = .02$).

Because two different word sets (study trials) were used, a preliminary analysis was carried out to examine whether there was a set effect on the main false recognition variable (clFR). An ANOVA with Group as a between-subject variable and Set as a within-subject variable indicated no significant Set effect, $F(1, 52) = 0.951$, $p = .334$, and no Set $\times$ Group interaction, $F(2, 52) = 0.907$, $p = .410$. Consequently, all the following statistics were computed by combining the results of the two sets. Given that participants were tested in their mother tongue (English or French), another preliminary analysis was done to examine the effect of language on the main variable. Group and Language were included as between-subject variables in the ANOVA. There was no significant main effect, nor a significant interaction of Language by Group, indicating that the language used for testing had no effect on performance. Thus, we collapsed all the data together.

3.2. Deese–Roediger–McDermott analyses

3.2.1. Hit rate (HR)

Percentage of hit rate was analyzed (Table 3) by an ANOVA using group (healthy controls, LBD, and FTD) as a between-subject variable. This analysis showed a significant effect of Group, $F(2, 52) = 3.281$, $p = .046$, $n^2 = 0.112$, power = .599. Only the LBD group had a lower level of hit rate ($p = .041$). The Bonferroni tests indicated that only the patients with LBD recognized fewer studied words than did healthy controls, $p < .001$. The LBD and FTD groups differed from one another, $p = .05$, but when Age and Education were included as covariates, the effect was no longer significant.

3.2.2. False alarms (unFA)

The proportion of false alarms is shown in Table 3. A significant group effect $F(2, 54) = 15.877$, $p < .001$ was found. More false alarms were produced in patients with LBD and FTD than in healthy controls $p < .001$, as indicated by post hoc comparisons. Similar results were obtained when Age and Education were included as covariates, $F(2, 50) = 8.786$, $p = .001$. Therefore, we used ANCOVAs with the rate of false alarms as a covariate in the analyses of false recognition effects for both clFR and wrFR.

3.3. Corrected hit rate (HR-unFA)

Corrected hit rate was obtained by subtracting the proportion of false alarms for unrelated lures from the proportion of hit rate for studied words (HR-unFA). Levels of corrected hit rate are presented in Table 3. A one-way ANOVA using Group (healthy controls, LBD, and FTD) as a between-subject variable, $F(2, 54) = 29.104$, $p < .001$, $n^2 = 0.528$, confirmed that both groups of patients performed at a lower level than the healthy older adults. The Bonferroni tests indicated that both patients with LBD and those with FTD recognized fewer studied words than did healthy controls, $p < .001$. Similar results were obtained when using Age and Education as covariates.

3.4. False recognition of critical lure (clFR)

Table 3 shows the level of false recognition for the critical lure (clFR) in the three groups of participants. The ANCOVA using Group (healthy controls, LBD, and FTD) as a between-subject variable and False Alarms (unFA) as a covariate indicated a significant group effect, $F(2, 54) = 17.802$, $p < .001$, $n^2 = 0.411$, power = 1.000. Bonferroni tests revealed that the two patient groups produced a lower level of false recognition than did healthy controls, $p < .001$ in both cases. Patients with LBD showed a lower level of clFR than those with FTD, but the effect was marginally significant, $p = .059$.

Table 2
Performance on tests by LBD and FTD patients and normal older adult controls.

Tests		Controls		LBD		FTD		Group differences ($p < .05$)
		M	*SE*	*M*	*SE*	*M*	*SE*	
Boston Naming Test		13.8	0.42	10.60*	0.73	8.53*	0.60	All patients ≠ controls
Hayling test								
	Time part 1 (automatic condition)	9.071	8.23	48.55	14.26	85.52*	14.26	FTD ≠ controls
	Time part 2 (Inhibition condition)	41.19	17.25	182.61*	29.88	146.12*	29.88	LBD ≠ controls when controlling for time on part 1
	Type A errors (related)	0.20	0.52	7.3*	0.90	7.9*	0.90	All patients ≠ controls
	Type B errors (somewhat related)	2.67	0.40	4.5	0.69	3.9	0.69	No group difference
Stroop Victoria task (Color–Words)								
	Time	7.85	5.01	33.22	9.14	10.18	7.33	No group difference
	Error	0.47	0.71	10.09*	1.23	5.14*	1.04	All patients ≠ controls
Buschke	Cued recall 4	3.9	0.46	9.3*	0.80	7.00*	0.67	All patients ≠ controls

* $p < .05$ for pairwise comparisons; relative to controls.

Table 3
Percentage of hit rate (true recognition) and false alarms and percentage of recognition for true, false critical, and weakly-related lures when covaried with percentage of false alarms.

Mean (SD)	Groups of participants		
	Controls	LBD	FTD
Hit rate (HR)	71.48 (3.17)	55.28* (5.50)	68.52 (4.49)
False alarm (unFA)	5.56 (4.34)	46.67* (7.51)	37.78* (6.14)
HR-unFA	65.93 (8.61)	8.61* (7.10)	30.74* (5.80)
False recognition of critical lures	81.94 (3.31)	41.45* (5.59)	57.39* (4.41)
False recognition of weakly-related lures	31.87 (3.05)	26.84 (5.16)	32.80 (4.07)

For the HR-unFA, LBD and FTD were also different from one another ($p = .050$).
* $p < .05$ for pairwise comparisons; relative to controls.

Results were unchanged when Age and Education were considered covariates.

3.5. False recognition of weakly-related lures (wrFR)

As was found in previous studies (Roediger & McDermott, 1995), the level of false recognition for weakly-related lures (wrFR) was higher than that for unrelated lures (or false alarms) but lower than the false recognition levels for critical lures (clFR) for the healthy controls only. The ANCOVA using Group (healthy controls, LBD, and FTD) as a between-subject variable and FA as a covariate on frWR indicated no group effect, $F(2, 54) = 0.499$, $p = .061$, $n^2 = 0.019$, power = .127. Similar results were found when Age and Education were added as covariates.

3.6. Repetition effect

Because participants viewed the study lists twice, we studied whether this repetition had an effect on the level of false recognition. A repeated measures ANOVA was used on the clFR variable using Study trial (1, 2) as a within-subject factor and Group (LBD, FTD, and controls) as a between-subject factor. The analysis indicated a significant Group × Study trial interaction, $p = .032$, $F(2, 52) = 3.673$. As shown in Table 4, healthy controls showed decreased levels of clFR on Study trial 2 relative to Study trial 1, whereas FTD patients showed increased clFR levels. Persons with LBD showed no Study trial effect. Patients with FTD had lower levels of clFR on Study trial 1 but did not differ from controls on Study trial 2.

3.7. Correlations

As seen in Table 5, there are no observable negative correlations between the proportion of clFR and hit rate. This finding indicates that memory capacity did not impact the performance of any group of participants in this paradigm.

In order to assess whether other cognitive processes (semantic memory, executive functions, and/or episodic memory) were associated with the loss of the false recognition effect in each patient group, we computed correlations between selected neuropsychological tests and the level of clFR and HR. The neuropsychological variables used were the total correct responses in the Boston Naming Test, the time to complete the automatic and inhibition conditions of the Hayling test, the time and number of errors of the Stroop Victoria test using a score that reflects performance on the interference/inhibition condition, while taking into account performance on the Color and Words conditions (Color–Words), and the fourth cued recall of the Grober and Buschke test. We also correlated performance with a more general measure of disease severity (the MMSE). In all cases, partial correlations were used to control for the level of unFA.

Table 6 shows results of the partial correlations for the patients with LBD. A significant negative correlation was found between clFR and the Stroop score, $r = -0.725$, $p = .042$, indicating that patients with more difficulties completing the Stroop Victoria test (those who took more time) produced fewer clFR responses. Unsurprisingly, there was also a positive correlation between HR and score on the MMSE, $r = 0.846$, $p = .008$, indicating that a more severe overall cognitive impairment is associated with a lower level of hit rate.

Correlations in persons with FTD are shown in Table 7. Here, clFR was positively related to the fourth cued recall of the Grober and Buschke task, $r = 0.633$, $p = .020$, and negatively related to the inhibition time of the Hayling test, $r = -0.632$, $p = .050$. The direction of the correlations indicates that, for this group, a lower false recognition effect was associated with a larger inhibition deficit on the Hayling and lower cued (or free) recall tests.

Table 4
Level of clFR at Set 1 and Set 2 for each group.

Mean (SD)	Sets	
	1	2
LBD	55.00 (7.78)	53.33 (8.10)
FTD	54.44 (6.35)	76.67 (6.61)
Controls	78.33 (4.49)	68.89 (4.67)

Table 5
Partial correlations between proportion of hit rate (HR) and false recognition to critical lures (clFR).

Groups		clFR
LBD	HR	.557
FTD	HR	.719*
Controls	HR	.221

* $p < .05$.

4. Discussion and conclusion

Our results are fairly straightforward: Both patients with LBD and those with FTD show a reduced false recognition effect compared with controls. That is, both patient groups produced a lower percentage of false recognition for the lures that were highly associated with the list. Although both groups showed a reduced false recognition effect relative to controls, this reduction was larger in patients with LBD than in those with FTD. Importantly, the results we received for healthy older adults are consistent with those found in the literature. Older adults falsely recognized the critical lures, and their proportion of false recognition is similar to previously published results (Budson et al., 2002; Hudon et al., 2006).

Table 6
Partial correlations between neuropsychological tests and hit rate (HR) and false recognition of critical lures (clFR) for LBD (FA used as covariate).

	clFR	HR
Boston Naming Test (semantic)	0.333	−0.066
Hayling time automatic	−0.157	−0.042
Hayling time inhibition	−0.307	0.272
Buschke cued recall 4	−0.492	−0.032
Stroop Color-Words Time	−0.725*	−0.229
Stroop Color-Words Score	0.281	0.444
MMSE	0.577	0.846*

* $p < .05$.

Table 7
Partial correlations between neuropsychological tests and hit rate (HR) and false recognition of critical lures (clFR) for FTD (FA used as covariate).

	clFR	HR
Boston Naming Test (semantic)	0.410	0.011
Hayling time automatic ($n = 10$)	−0.380	−0. 238
Hayling time inhibition ($n = 10$)	−0.632*	−0.291
Buschke cued recall 4	0.633*	0.330
Stroop Color-Words Time	0.067	−0.082
Stroop Color-Words Score	−0.338	−0.273
MMSE ($n = 14$)	−0.274	−0.264

* $p < .05$.

This correspondence indicates that the procedure we followed to measure this classical effect is sound.

In order to evaluate whether impaired cognitive processes caused these low false recognition effects in the patient groups, we computed partial correlations between the level of false recognition for the critical lure, hit rate, and performance on target neuropsychological measures. In both patients with LBD and those with FTD, lower inhibition capacities were associated with a lower false recognition effect. In the FTD patients, an additional correlation was found with the Grober and Buschke memory test, indicating that a lower cued recall of the fourth trial was associated with a lower false recognition effect.

One important question is how these data compare with what has been previously reported on AD. The paradigm used here is very similar to that used in a past study of patients suffering from Alzheimer's disease and persons with mild cognitive impairment (Hudon et al., 2006). In Hudon et al.'s study, the level of FA was corrected by subtracting it from the clFR score. When we applied the same correction, we obtained very similar scores in our group of FTD participants compared with the AD patients examined by Hudon et al. (2006). In turn, persons with LBD produced lower levels of the false recognition effect than both the FTD patients and Hudon's group of AD patients.

Other results are worth mentioning. First, the hit rate level was lower only in LBD patients, but both groups showed a lower level of memory performance when using a score that considers the level of false alarms (H-unFA). Observable group differences were found for level of education and age. However, these differences had little impact on the main variables used here. All false recognition effects remained significant when covarying for age and education. These factors showed an impact only on the hit rate variable.

Another interesting result is the repetition effect. As expected, controls showed a reduction in their level of false recognition effect after two study trials. This finding is coherent with past studies (Budson et al., 2002) and has been suggested to occur because of the balance between veridical and gist memory. Given that veridical memory improves with study trials, participants are better able to identify that the critical lure was not presented after two trials. Most interestingly, persons with FTD showed an increase in their level of false recognition effect with study trials. After two study trials, their false recognition of the critical lure was no longer depleted relative to controls. That is, FTD patients are able to improve the strength of the activation within their semantic network. By contrast, persons with LBD showed no Study trial effect, and the effect remains low even after two study trials, perhaps indicating a more severe and resistant effect in this group.

The results of this study indicate that the false recognition effect measured with the DRM procedure is largely impaired in many types of dementia. The false recognition effect resulting from the DRM paradigm has been suggested to depend on a number of different processes including semantic memory, episodic memory, and executive functions. Because the effect may depend on the integrity of any of the aforementioned processes, this may increase its sensitivity to different types of dementia or cognitive impairment. Yet, the correlational pattern found in LBD and FTD patients indicates that the source of the deficit is quite selective, since we found a relation between impaired DRM effect and inhibition deficits in both groups of patients. Severe executive and inhibition deficits may prevent these types of patients from being able to process the essence of the semantically related lists. Additionally, memory impairment (the fourth cued recall of the Buschke test) was related to a low false recognition effect in persons with FTD. This finding is consistent with the hypothesis that episodic memory is necessary for participants to encode and memorize the general meaning of the list of related words (Hudon et al., 2006; Verfaellie et al., 2002). Another possibility is that semantic difficulties mediate this relation. Semantic difficulties might indeed contribute to lower performance on the Buschke test, as this is a measure that uses categorical support at encoding and retrieval.

Although this study provides interesting data regarding the false recognition effect in dementia, there are some limitations. Most notably, only 10 patients with LBD and 15 with FTD were included in the research cohort. Importantly, however, the critical comparisons were highly significant, and the effect sizes were large to moderate. Moreover, the power analyses indicate that the effect size was not an issue. Examination of the numbers indicates that outliers did not drive the correlations. Another possible limitation is that the LBD patients were recruited exclusively from memory clinics (rather than motor disorder clinics). As such, they may be more likely to demonstrate cognitive deficits than the typical LBD patient. In terms of FTD, it would also be preferable to limit participants to those with a single variant of FTD. It should be noted, however, that reanalyzing the data without considering the patient with a different variant produced no effect on our main results. Next, the presence of a high level of false alarms indicates a major response bias. Although it is crucial to control for this bias, doing so may make the task less sensitive to the false recognition effect. In addition, only two trials were used. It would be interesting to increase the number of repetitive presentation trials (Budson et al., 2002). Finally, in line with the work of Butler et al. (2004), future studies could compare phonologically related word lists and hybrid lists of phonologically and semantically related words. This technique might help to elucidate the implication of semantic memory in the false memory effect of FTD patients.

In summary, the present study is the first to examine the false recognition effect in patients with FTD and LBD. The results clearly indicate the presence of an impairment compared with controls. The false recognition effect is lower than that of controls for both patient groups, though less so for patients with LBD than for those with FTD. By investigating both patients with FTD and those with LBD, this study indicates that the false recognition effect measured with the DRM procedure is a sensitive effect, one largely impaired in many types of dementia. Interestingly, reduced false recognition was associated with an inhibition deficit in both FTD and LBD. From a clinical point of view, this study contributes to a greater understanding of the cognitive profile of patients with FTD and those with LBD by showing that the inhibition deficit in FTD and LBD patients might result in impairment in tasks that are not typically associated with the executive domain.

Acknowledgments

This research was supported by the *Fonds de la recherche en santé du Québec* (FRSQ) and the Quebec Research Network on Aging (Cognition axis) in order to create a patient registry for memory impairment. We would like to acknowledge the teams from the Lady Davis Institute for Medical Research, the *Centre de recherche de l'Institut universitaire de gériatrie de Montréal* and Concordia

University, all of whom contributed to the coordination of this project. We would like to thank Harold Gaboury and Amanda Leonard for their editorial assistance.

References

Anderson, N. H. (1981). *Foundations of information integration theory*. New York: Academic.

Belleville, S., Caza, N., & Peretz, I. (2003). A neuropsychological argument for a processing view of memory. *Journal of Memory and Language, 48*, 686–703.

Belleville, S., Rouleau, N., & Van der Linden, M. (2006). Use of the Hayling task to measure inhibition of prepotent responses in normal aging and Alzheimer's disease. *Brain and Cognition, 62*, 113–119.

Brainerd, C. J., & Reyna, V. F. (1998). Fuzzy-trace theory and children's false memories. *Journal of Experimental Child Psychology, 71*(2), 81–129.

Budson, A. E., Sullivan, A. L., Mayer, E., Daffner, K. R., Black, P. M., & Schacter, D. L. (2002). Suppression of false recognition in Alzheimer's disease and in patients with frontal lobe lesions. *Brain, 125*(Pt 12), 2750–2765.

Burgess, P. W., & Shallice, T. (1997). *The Hayling and Brixton tests*. Bury St. Edmunds: Thames Valley Test Company Limited.

Butler, K. M., McDaniel, M. A., Dornburg, C. C., Price, A. L., & Roediger, H. L. III, (2004). Age differences in veridical and false recall are not inevitable: The role of frontal lobe function. *Psychonomic Bulletin and Review, 11*(5), 921–925.

Calderon, J., Perry, R. J., Erzinclioglu, S. W., Berrios, G. E., Dening, T. R., & Hodges, J. R. (2001). Perception, attention, and working memory are disproportionately impaired in dementia with Lewy bodies compared with Alzheimer's disease. *Journal of Neurological Neurosurgery and Psychiatry, 70*, 157–164.

Collerton, D., Burn, D., McKeith, I., & O'Brien, J. (2003). Systematic review and meta-analysis show that dementia with Lewy bodies is a visual–perceptual and attentional-executive dementia. *Dementia and Geriatric Cognitive Disorders, 16*, 229–237.

Collette, F., Delrue, G., Van der linden, M., & Salmon, E. (2001). The relationships between executive dysfunction and frontal hypometabolism in Alzheimer's disease. *Brain and Cognition, 47*, 272–275.

Collins, A. M., & Loftus, E. F. (1975). A spreading activation theory of semantic memory. *Psychological Review, 82*, 407–428.

Dieudonné, B., Marquis, C., Ergis, A. M., & Verny, M. (2006). La démence à corps de Lewy: diagnostique clinique et neuropsychologique. In C. Belin, A. M. Ergis, & O. Moreaud (Eds.), *Actualité sur les démences: aspects cliniques et neuropsychologiques*. Solal, Marseille: Collection neuropsychologie.

Downes, J. J., Priestley, N. M., Doran, M., Ferran, J., Ghadiali, E., & Cooper, P. (1998–1999). Intellectual, mnemonic, and frontal functions in dementia with Lewy bodies: A comparison with early and advanced Parkinson's disease. *Behavioural Neurology, 11*, 173–183.

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). Mini-mental state: A practical method for grading the cognitive state of outpatients for the clinician. *Journal of Psychiatric Research, 12*, 189–198.

Goodglass, H., Kaplan, E., & Barresi, B. (2001). *The assessment of aphasia and related disorders* (3rd ed.). Philadelphia: Lippincott, Williams & Wilkins.

Graff-Radford, N. R., & Woodruff, B. K. (2007). Frontotemporal dementia. *Seminars in Neurology, 27*(1), 48–57.

Grober, E., & Buschke, H. (1987). Genuine memory deficits in dementia. *Developmental Neuropsychology, 3*, 13–36.

Hudon, C., Belleville, S., Souchay, C., Gély-Nargeot, M. C., Chertkow, H., & Gauthier, S. (2006). Memory for gist and detail information in Alzheimer's disease and mild cognitive impairment. *Neuropsychology, 20*(5), 566–577.

Johns, E. K., Phillips, N. A., Belleville, S., Goupil, D., Babins, L., Kelner, N., et al. (2009). Executive function in frontotemporal dementia and Lewy body dementia. *Neuropsychology, 23*(6), 765–777.

Josephs, K. A., Whitwell, J. L., Knopman, D. S., Boeve, B. F., Vemuri, P., Senjem, M. L., et al. (2009). Two distinct subtypes of right temporalvariant frontotemporal dementia. *Neurology, 73*, 1443–1450.

Kaplan, E. F., Goodglass, H., & Weintraub, S. (1983). *The Boston Naming test* (2nd ed.). Philadelphia: Lea and Febiger.

Kertesz, A., McMonagle, P., Blair, M., Davidson, W., & Munoz, D. G. (2005). The evolution and pathology of frontotemporal dementia. *Brain, 128*, 1996–2005.

Kirshner, H. S. (2010). Frontotemporal lobe dementia. *EMedecine* Available from <http://www.emedicine.com/NEURO/topic140.htm>.

Krantz, D. H., Luce, R. D., & Tversky, A. (1971). *Foundations of measurement*. New York: Academic.

Lambon, R. M. A., Patterson, K., Graham, N., Dawson, K., & Hodges, J. R. (2003). Homogeneity and heterogeneity in mild cognitive impairment and Alzheimer's disease: A cross-sectional and longitudinal study of 55 cases. *Brain, 126*, 2350–2362.

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). Oxford: Oxford University Press.

McDermott, K. B., & Watson, J. M. (2001). The rise and fall of false recall: The impact of presentation duration. *Journal of Memory and Language, 45*, 160–176.

McKeith, I. (2005). Behavioural changes and psychological symptoms in dementia disorders. *Lancet Neurology, 4*, 735–742.

McKeith, I., Mintzer, J., Aarsland, D., Burn, D., Chiu, H., Cohen-Mansfield, J., et al. (2004). Dementia with Lewy bodies. *Lancet Neurology, 3*, 19–28.

Metzler-Baddeley, C. (2007). A review of cognitive impairments in dementia with Lewy bodies relative to Alzheimer's disease and Parkinson's disease with dementia. *Cortex, 43*, 583–600.

Nasreddine, Z. S., Phillips, N. A., Bédirian, V., Charbonneau, S., Whitehead, V., Collin, I., et al. (2005). The Montreal cognitive assessment, MoCA: A brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society, 53*, 695–699.

Neary, D., & Snowden, J. (1996). Fronto-temporal dementia: Nosology, neuropsychology, and neuropathology. *Brain Cognition, 31*, 176–187.

Neary, D., Snowden, J., & Mann, D. (2005). Frontotemporal dementia. *Lancet Neurology, 4*, 771–780.

Reyna, V. F., & Brainerd, C. J. (1992). A fuzzy-trace theory of reasoning and remembering: Paradoxes, patterns, and parallelism. In A. Healy, S. Kosslyn, & R. Shiffrin (Eds.). *From learning processes to cognitive processes: Essays in honor of William K. Estes* (Vol. 2, pp. 235–259). Hillsdale, NJ: Erlbaum.

Reyna, V. F., & Brainerd, C. J. (1995). Fuzzy-trace theory: An interim synthesis. *Learning and Individual Differences, 7*, 1–75.

Roediger, H. L., III, & McDermott, K. B. (1995). Creating false memories: Remembering words not presented in lists. *Journal of Experimental Psychology: Learning, Memory, & Cognition, 21*, 803–814.

Roediger, H. L., III, & McDermott, K. B. (1999). *False alarms about false memory: Psychological review, 106*, 406–410.

Roediger, H. L., III, & McDermott, K. B. (2000). Tricks of memory. *Current Directions in Psychological Science, 9*, 123–127.

Spreen, O., & Strauss, E. (1998). *Compendium of neuropsychological tests* (2nd ed.). New York: Oxford University Press.

Thuillard Colombo, F., & Assal, G. (1992). Le test de dénomination de Boston: Adaptation française et versions abrégées. *Revue Européenne de Psychologie Appliquée, 1*, 67–73.

Verfaellie, M., Schacter, D. L., & Cook, S. P. (2002). The effect of retrieval instructions on false recognition: Exploring the nature of the gist memory impairment in amnesia. *Neuropsychologia, 40*, 2360–2368.

Walker, M. P., Ayre, G. A., & Cummings, J. L. (2000). Quantifying fluctuation in dementia with Lewy bodies, Alzheimer's disease and vascular dementia. *Neurology, 54*, 1616–1624.

Watson, J. M., Balota, D. A., & Sergent-Marshall, S. D. (2001). Semantic, phonological, and hybrid veridical and false memories in healthy adults and in dementia of the Alzheimer's type. *Neuropsychology, 15*, 254–267.