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False recognition in Lewy-body disease and frontotemporal dementia

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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

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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