A cognitive neuropsychological approach to false memory: Korsakoff patients and the DRM paradigm

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Although false memories have been studied extensively within the field of cognitive psychology, the role of cognitive neuropsychology in false memory research has been rather limited. Therefore, the present article aims at giving an integrated overview of both the neuropsychological study of amnesia and research on false memories. After considering some of the most important predecessors in the study of memory distortions/illusions, a list-learning paradigm designed to elicit and investigate false memories is described (i.e., the DRM paradigm), and theoretical accounts for the phenomenon are provided. Subsequently, the neuropsychological framework is presented, with an emphasis on Korsakoff patients' performance in the earlier described paradigm. Several experimental manipulations, as well as the results, are discussed and interpreted in the light of both encoding and retrieval factors. (*Netherlands Journal of Psychology*, 64: 96-111.)

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Although memory is a crucial part of virtually every aspect of day-to-day life, the use of the simple truth 'I remember it; therefore it happened' (Tulving, 2002, p. 272) is not as justified as it may seem. Not only do people forget things that *did* happen, they also remember things that did *not* happen or remember them differently from the way in which they happened (Roediger & McDermott, 1995). When the latter occurs, they are said to produce 'false' memories.

False memories have been the object of a long-standing research tradition in (cognitive) psychology (see Schacter, 1995, for a historical overview). Nevertheless, a recent rise in interest has occurred, mainly because of the increasing number of cases in which people report recovering repressed memories of childhood abuse. Memory scientists (e.g., Lindsay & Read, 1994; Loftus & Ketcham, 1994; Poole, Lindsay, Memon, & Bull, 1995; Schooler, 1994) have expressed the concern that some of these memories recovered during the course of therapy may in fact be false memories, induced by therapeutic techniques, suggestive questions, and the demands of an interview (Hyman, Husband, & Billings, 1995). Consequently, false memories are directly relevant to clinical psychology and psychiatrics, but also to cognitive psychology, neuropsychology, eyewitness testimony, sociology, etc. The present

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paper offers a cognitive neuropsychological approach to the phenomenon.

The reconstructive nature of memory

Already in 1932, Bartlett proposed a distinction between reproductive and reconstructive memory processes, based on the memory distortions obtained in his 'War of the ghosts' paradigm. He had subjects read an old Indian legend (entitled 'The War of the Ghosts'), followed by either 'repeated reproduction' or 'serial reproduction'. The former meant that participants were asked to recall the story repeatedly, whereas the latter implied one person recalling the story, the next person reading and then recalling this report, and so on. The severe distortions of the subjects' memories were explained by the reconstructive nature of memory: When confronted with meaningful materials like stories and real-life events, memory was thought to reflect an 'active process of filling in missing elements while remembering, with errors frequently occurring' (Roediger & McDermott, 1995, p. 803). Reproductive memory, on the other hand, referred to the accurate and exact reproduction of simple, straight-forward materials, such as word lists.

Starting from the early 1970s, in the context of eyewitness memory research, Loftus et al. (e.g., Loftus, Miller, & Burns, 1978; Loftus & Palmer, 1974; Schooler, Gerhard, & Loftus, 1986) have provided evidence for what is known as the 'misinformation effect': By exposing people to misleading information or suggestive questions after they have witnessed an event (e.g., simulated crime, traffic accident), the new information gets incorporated into memory, and recollection of the original event can become seriously distorted. In addition, it has been shown that entirely new (and thus false) memories can be implanted in people's minds. For instance, Ceci, Huffman, Smith, and Loftus (1994) convinced children that, when they were younger, they had gotten their hand caught in a mousetrap or had gone on a hot air balloon ride with their classmates; Hyman et al. (1995) convinced college students that they had, as children, been hospitalised because of a possible ear infection, had a specific type of birthday party, or spilled a punch bowl at a family wedding; and Loftus and Pickrell (1995) convinced adults that they had been lost in a shopping mall at the age of 5 (see also Loftus, 1997; Loftus, Coan, & Pickrell, 1996). Taken together, these studies have provided compelling evidence for both the induction of memory distortion and the creation of entirely new, false memories.

Whereas it was previously believed that only memory for meaningful materials was subject to distortion, nowadays it is known that virtually all memory is reconstructive in nature, and thus prone to errors. As stated by Tulving (2002, p. 273), 'scientific research has clearly established that it is not always possible to determine what happened in the past on the basis of a person's recollection, regardless of whom the person is, and regardless of how strongly – and genuinely – the person believes that he or she is telling the truth'. Nevertheless, within the tradition of Bartlett (1932), the majority of false memory scientists have kept on using complex materials, such as sentences (e.g., Bransford & Franks, 1971; Brewer, 1977), prose passages (e.g., Dooling & Christiaansen, 1977; Sulin & Dooling, 1974), series of slides depicting an event (e.g., Loftus, Feldman, & Dashiell, 1995; Roediger, Jacoby, & McDermott, 1996), and videotapes (e.g., Loftus & Palmer, 1974; Mitchell & Zaragoza, 1996).

However, an early study by Underwood (1965) demonstrated that it is possible to obtain false memories when using a simple list-learning paradigm as well. In this study, a continuous recognition task was used, in which subjects were to decide for each presented word whether or not it had been shown earlier in the list. As the relationships between previous and following words were experimentally manipulated, Underwood showed that words with associative or physical relations to previously presented words were falsely recognised. He argued that these false recognitions arose from 'implicit associative responses' that occurred during the initial study of the list items. Cermak, Butters, and Gerrein (1973, Experiment 4) replicated and extended Underwood's findings by showing that Korsakoff patients produced significantly higher false alarm rates than controls.

Since the late 1990s, as interest in false memory research increased and new techniques were being developed, another list-learning paradigm has started to dominate the false memory literature.

Originally developed by Deese in 1959, but revived and adapted by Roediger and McDermott in 1995, the 'DRM' paradigm is a straightforward laboratory paradigm, leading to particularly high levels of false memory in normal, healthy subjects. It allows the investigation of experimental variables that elicit, inhibit, or influence the formation of false memories, and has also been applied to false memory in amnesic patients. As a standard list-learning paradigm, it contains no misleading information. In addition, testing occurs immediately after the study phase, and subjects are warned to be accurate and to avoid guessing. Even so, the false memories obtained are among the strongest ever reported in memory literature (Roediger & McDermott, 2000).

DRM 'false' memories

The paradigm

In the Deese/Roediger-McDermott (DRM) paradigm, people study lists of words (e.g., bed, rest, awake, tired, dream, night, ...), which are all semantically related to one critical word which is not presented in the list (e.g., sleep). On subsequent memory tests, they often 'recall' and/or 'recognise' this critical word, without actually having seen or heard it before.

In their pioneering study, Roediger and McDermott (1995) constructed word lists by obtaining the first 15 associations listed in norm tables for those words that would serve as the critical 'lures'. College students were presented with 16 of such lists, after half of which they received a free recall test, and after half of which they were asked to solve math problems. At the end of the experiment, an overall recognition test was administered, in which studied words were intermixed with new unrelated words and new related (critical) lure words. Each item had to be classified as either 'old' (studied) or 'new' (unstudied). For items judged to be old, an additional response was required: Students had to indicate whether they 'remembered' (i.e., could recollect specific details about the moment of presentation) or just 'knew' the word (i.e., were sure of having encountered it before, but could not remember its actual presentation; cf. Rajaram, 1993, and Tulving, 1985).

The students falsely recalled critical lure words with about the same probability as they recalled words that had actually been presented in the middle of the lists (i.e., excluding primacy and recency effects). On the final recognition test, false recognition of the critical lure was as high as the hit rate for studied items (about 80%). Furthermore, without having seen or heard the critical lure word before, participants claimed to be able to mentally relive the experience of studying it: They mistakenly 'remembered' the critical lures to the same level as they correctly 'remembered' studied items. The effect of initial testing on subsequent testing was the same for critical lures as for studied words: Prior recall enhanced later false as well as true memory (see also McDermott, 1996).

Since then, these results have often been replicated and extended (e.g., Dehon & Brédart, 2004; Geraerts, Smeets, Jelicic, van Heerden, & Merckelbach, 2005; Israel & Schacter, 1997; McKone & Murphy, 2000; Payne, Elie, Blackwell, & Neuschatz, 1996; Schacter, Verfaellie, & Pradere, 1996). The illusion has been shown to be very difficult to avoid, even when explicitly warned against it (McDermott & Roediger, 1998). The strength of the effect appears to be a direct function of the number of associations presented during study (e.g., Robinson & Roediger, 1997). However, the more study words correctly remembered at test, the less false memory is obtained (Roediger, Watson, McDermott, & Gallo, 2001).

'False' memories?

It should be noted that the idea that the DRM paradigm elicits 'false' memories has recently been questioned. Pezdek and Lam (2007) have argued that the term 'false memory' was originally intended to refer to planting memories for an entirely new event, and should only be used as such (see DePrince, Allard, Oh, & Freyd, 2004, for similar claims). According to the authors, the false alarm rate to critical non-presented DRM words (as well as other kinds of memory illusions and distortions) should be referred to as 'flawed memory', instead of 'false memory', in order to avoid confusion and enhance communication between scientists. Moreover, Pezdek and Lam suggest that the DRM paradigm is only used so much because it guarantees the occurrence of memory intrusions, with researchers failing to take into account that DRM studies cannot be generalised to situations involving memory for entirely new events, such as memories of sexual abuse. Nonetheless, Geraerts et al. (2005) recently showed that individuals reporting recovered memories of child sexual abuse are more susceptible to the DRM memory illusion than other participants. The association found between fantasy proneness and false memories, moreover, suggested that the DRM paradigm does tap susceptibility to pseudo-memories. Additionally, Wade et al. (2007) have reviewed each of the arguments made by Pezdek and Lam, and were able to refute virtually all of them. Concluding, the term 'false memory' can be used to describe a wide range of phenomena, including the DRM phenomenon.

Theoretical explanations

According to Roediger and McDermott (2000), false memories found in studies using the DRM paradigm represent experimental analogues of false memories in everyday life. As people make inferences about associatively related word lists, they also make inferences about conversations, observations, and experiences. Two main causes are thought to give rise to the illusion: associative activation followed by source monitoring problems, and the encoding and retrieval of 'gist' information.

Activation-monitoring theory

Source memory, or source monitoring, refers to the processes by which people remember when, where, and how a memory was acquired (Schacter, 1995). According to the source monitoring framework of Johnson, Hashtroudi, and Lindsay (1993), memory for source is an attribution, resulting from a judgement process.

Memory representations are assumed to possess certain characteristics reflecting the conditions under which they were formed (e.g., contextual and sensory information; see Johnson, 1985), and judgements about source are made by evaluating the type and amount of these characteristics in combination with more extended reasoning. The accuracy of the judgements will vary depending on the circumstances at the time of retrieval, as well as the characteristics of the underlying memory representation (Zaragoza & Lane, 1994).

In the memory distortion/creation experiments of Loftus et al., false memories can be accounted for by source misattribution: As the suggestive and/or misleading information becomes integrated with the actual memories of an event, people may forget the original source of the information and attribute the misleading information to the event (Loftus, 1997; see also Zaragoza & Lane, 1994). In a similar way, Roediger and McDermott (1995) have argued that the high levels of false memory obtained in their experiments could be accounted for by activation of the lure words, followed by source monitoring problems.

According to such an 'activation-monitoring' account, critical lures may be mentally activated during study (i.e., an implicit associative response or 'IAR'; Underwood, 1965), either through the unconscious spreading of activation, or coming to mind consciously. On subsequent memory tests, participants are confronted with a source monitoring problem, in which they need to distinguish between items that were actually studied and items that were merely mentally activated during study. Confusion regarding the origin of the items (i.e., source confusion) may lead them to mistakenly recollect the critical lure as being part of the study list. In line with this explanation, Roediger et al. (2001) found backward associative strength (i.e., the probability with which the critical lure is elicited by the various list items; Deese, 1959; Roediger & McDermott, 1995) to be the strongest predictor of false recall in a multiple regression analysis. Moreover, Schacter and colleagues (e.g., Israel & Schacter, 1997) demonstrated that providing people with a strategy for evaluating the retrieved information improves their source monitoring abilities, and enables them to inhibit the occurrence of false memories.

Fuzzy-trace theory

A second possible explanation is that illusory memories arise because of their consistency with the 'gist' of the study lists. According to the 'fuzzy-trace theory' (Brainerd & Reyna, 1998, 2002; Reyna & Brainerd, 1995), memory can be based on either 'verbatim' or 'gist' traces that are encoded in parallel during the study phase. Verbatim traces preserve specific information about the identity of each item, whereas gist traces capture the general meaning and interpretations invoked by the studied items.

In the DRM paradigm, strong gist traces will be formed, because of the high levels of semantic overlap between the list words. At test, the mix of verbatim and gist retrieval will determine memory performance. Whereas both support true memory, they have opposite effects on false memory: Gist retrieval supports it; verbatim retrieval suppresses it. Findings of reduced false memory as more list words are being correctly remembered (Roediger et al., 2001) are in accordance with this claim. As a consequence, a negative correlation between true and false memory is to be expected when subjects are instructed to rely on verbatim traces as the basis for a true response, whereas a positive correlation is to be expected when subjects are instructed to rely on gist traces as the basis for a true response (Brainerd & Reyna, 2002).

Both kinds of information can lead to vivid forms of remembering. However, verbatim memory declines more rapidly in time than gist memory does. Hence, false memory may be more persistent over time than veridical memory is. McDermott (1996) tested participants two days after study, and found that false recall of critical lures exceeded veridical recall of studied words. Payne et al. (1996) reported similar findings concerning false recognition. More recently, Seamon et al. (2002) used retention intervals ranging from no delay to a two-month delay, and also obtained greater persistence for false memory than for veridical memory, in both free recall and recognition tasks (see also Thapar & McDermott, 2001). In this respect, fuzzy-trace theory shows considerable overlap with Bartlett's theory of remembering. According to Bartlett (1932), past experiences and reactions are actively organised in memory, and the central meaning of an experience is stored in a schematic form. Remembering is then considered a process of reconstruction, in which individuals make use of a particular 'schematic' organisation. With longer retention intervals, the tendency to make thematic errors increases: Memory for specific details of an event is expected to decay with time, whereas schema-consistent information remains relatively intact (Sulin & Dooling, 1974). It should be mentioned, however, that there are also studies that failed to find the greater stability of false memory compared with veridical memory. For instance, in Lampinen and Schwartz (2000), corrected recognition scores for critical lures decreased over time in a similar way as corrected recognition scores for list words did (see Gallo, 2006, for an overview of findings with the DRM paradigm). Also, in a study evaluating children's suggestibility and the preservation of children's false memories (Huffman, Crossman, & Ceci, 1997), implanted memories were shown to be less persistent over time than accurate memories were.

Constructive memory framework

The constructive memory framework of Schacter, Norman, and Koutstaal (1998) focuses on important encoding and retrieval processes that contribute to both accurate and inaccurate memories (Dodson & Schacter, 2001). Representations of new experiences are conceptualised as patterns of features, distributed across different parts of the brain, with different features representing different characteristics of the experience (e.g., sensory and conceptual information).

At encoding, feature binding and pattern separation are considered crucial. In order to form a coherent memory representation, all features comprising an episode must be linked together (i.e., feature binding), but different episodes need to be kept separate from each other (i.e., pattern separation). Remembering an episode then requires processes of focusing, pattern completion, and criterion setting. At a preliminary stage, a fine-tuned description of the characteristics of the to-be-remembered episode needs to be formed (i.e., focusing). Next, once a retrieval cue has activated a subset of the features of the target episode, the activation needs to be spread to obtain the complete pattern of features that constituted the original experience (i.e., pattern completion). Finally, a decision must be made about whether the activated information is a veridical memory, or rather resulted from imagination or thought (i.e., criterion setting).

Whereas problems with any of these processes can result in the occurrence of memory distortion, three of them are considered especially important in explaining false memories in the DRM paradigm (Dodson & Schacter, 2001). First of all, as people may internally generate critical lures during encoding of the lists (IAR; Underwood, 1965; cf. supra), poor focusing and criterion setting at test may cause source confusion, leading people to mistakenly believe that they studied the words they generated. Secondly, when studying strongly related items, pattern separation is very likely to fail because of the high levels of overlap among the corresponding memory representations. Hence, people will show good memory for what the items have in common (the gist of the list), but poor memory for the specific characteristics of each item separately. Whereas the first explanation extends and supports activation-monitoring theory, the second one supplements fuzzy-trace theory.

Neuropsychological perspective

Throughout history, neuropsychological studies of patients with amnesia, such as Korsakoff patients, have had a tremendous impact on research and theorising about memory. For instance, Korsakoff's syndrome has historically been considered the prime example of the dissociation between implicit and explicit memory performance (Phaf, Geurts, & Eling, 2000; Schacter, 1987): Whereas the patients perform poorly when explicitly asked to report about the past, they show a normal 'unconscious' effect of past experience in their performance on a variety of implicit memory tasks (e.g., Beauregard et al., 1997; d'Ydewalle & Van Damme, 2007; Graf, Shimamura, & Squire, 1985; Phaf et al., 2000; Warrington & Weiskrantz, 1974). In the following paragraphs, the syndrome will be discussed in detail, followed by an overview of studies examining Korsakoff (and other amnesic) patients' performance in the DRM paradigm.

Korsakoff's syndrome

At the end of the 19th century, a Russian psychiatrist, Sergei Korsakoff, described a striking form of amnesia that resulted from alcohol abuse. Known today as Korsakoff's syndrome, the disorder is characterised by a severe impairment of memory for recent and remote events, but spared perceptual and linguistic abilities, along with IQ scores within the normal range (e.g., Butters & Cermak, 1980; Parkin & Leng, 1993).

Korsakoff's syndrome develops following years of chronic alcohol abuse and nutritional thiamine deficiency (e.g., Homewood & Bond, 1999). The most salient feature of the syndrome is without any doubt the disproportionate impairment of memory relative to other aspects of cognitive functioning (Butters & Cermak, 1980; Lezak, 1995; Parkin & Leng, 1993). Another striking symptom, however, is the patients' tendency to confabulate. As defined by Dalla Barba (1993, p. 2), confabulation is 'that particular symptom, frequently observed in amnesic patients unaware of their memory deficit, which consists of both actions and verbal statements that are unintentionally incongruous to the patients' history, background, and present situation'. More specifically, confabulations can be defined as erroneous memories, either false in themselves or resulting from the inappropriate retrieval or misinterpretation of 'true' memories (Kopelman, Guinan, & Lewis, 1995).

Neuropathologically, the critical role of damage to diencephalic and medial temporal brain regions has been well documented (e.g., Oscar-Berman & Evert, 1997; Parkin & Leng, 1993). More specifically, lesions to the mammillary bodies (e.g., Mair, Warrington, & Weiskrantz, 1979), the mediodorsal thalamic nucleus (e.g., Visser et al., 1999), the anterior thalamic nucleus (e.g., Harding, Halliday, Caine, & Kril, 2000), the mammillo-thalamic tract (Van der Werf, Witter, Uylings, & Jolles, 2000), and the hippocampus (Sullivan & Marsh, 2003) or at least some extended hippocampal system involving the hippocampal-anterior thalamic axis (Caulo et al., 2005; see also Aggleton & Saunders, 1997) have been linked to the severe (anterograde and retro-

grade) amnesia that characterises the syndrome. Recent brain imaging research (e.g., Kril, Halliday, Svoboda, & Cartwright, 1997; Reed et al., 2003) also suggests damage to the (pre)frontal lobes, in line with the executive function deficits often found in the patients (e.g., Brokate et al., 2003).

During the last decade, substantial evidence has been obtained that medial temporal / hippocampal brain regions are activated during both episodic memory retrieval and episodic memory encoding (e.g., Greicius et al., 2003). Several PET studies have linked increased activity in the medial temporal lobe to the conscious recollection of recently studied materials (e.g., Nyberg, McIntosh, Houle, Nilsson, & Tulving, 1996; Schacter, Alpert, Savage, Rauch, & Albert, 1996), which is in accordance with the well-established finding of Korsakoff patients' impaired performance on explicit memory tests. In addition, however, both brain imaging and behavioural studies have revealed the involvement of the hippocampal area in encoding new events into episodic memory (e.g., Kopelman, Stevens, Foli, & Grasby, 1998; Stern et al., 1996), and especially in (successfully) binding multiple elements into an integrated memory trace (e.g., Davachi & Wagner, 2002; Jackson & Schacter, 2004; Kroll, Knight, Metcalfe, Wolf, & Tulving, 1996). Indeed, several theories have proposed that the basic deficit in amnesia lies in the formation of new associations or relationships between previously distinct elements (e.g., Cohen, Poldrack, & Eichenbaum, 1997; Johnson & Chalfonte, 1994). Moreover, as Korsakoff patients suffer from frontal lobe damage, and prefrontal cortex has been shown to be associated with the strategic, 'effortful' encoding of information into memory (e.g., Gershberg & Shimamura, 1995) and the elaboration of meaning (e.g., Dolan & Fletcher, 1997; see also Fletcher & Henson, 2001), this particular group may be even more vulnerable to deficient encoding than other amnesic patients are.

Early studies demonstrating impaired implicit memory for newly formed associations in Korsakoff patients (e.g., Cermak, Bleich, & Blackford, 1988; Shimamura & Squire, 1989; for a review, see Bowers & Schacter, 1993) provided support for such a relational encoding deficit account. More recently, however, the associative priming effect has been shown to be closely intertwined with explicit memory or consciously controlled recollection (Badgaiyan, Schacter, & Alpert, 2003; Reingold & Goshen-Gottstein, 1996). Therefore, rather than an encoding deficit, the patients' explicit recollection deficit may have been responsible for the observed diminished performance. In line with this reasoning, Verfaellie, Cermak, Blackford, and Weiss (1990) provided evidence that Korsakoff patients show normal sensitivity to both associative and categorical relationships when tested implicitly. Moreover, Giovanello, Keane, and Verfaellie (2006) recently demonstrated that associative recognition in amnesic (Korsakoff) patients can be enhanced, to the extent that familiarity-based processes contribute to performance. The combination of these findings suggests that relational information is processed adequately during study, but can only be brought to mind involuntarily or automatically at test.

Nevertheless, Korsakoff patients have been shown to perform worse than control participants even in some implicit memory tasks (e.g., Brunfaut & d'Ydewalle, 1996). Furthermore, the degree of (relational) encoding required to perform normally under explicit retrieval instructions might be higher than what is needed to perform normally under implicit retrieval instructions (see Graf, Squire, & Mandler, 1984; cf. transfer-appropriate processing approach, e.g., Morris, Bransford, & Franks, 1977). Support for such an argument is provided by the fact that Korsakoff patients' explicit memory performance for associative information is substantially improved when given additional time, explanation, and support (Van Damme & d'Ydewalle, 2008a). Moreover, it is in agreement with the suggestion by Hirst and Volpe (1988) that it is the failure to spontaneously organise and elaborately process the study materials that exacerbates Korsakoff patients' memory impairment compared with other amnesic patients (see also Jacoby, 1982). In line with this reasoning, studies by both Cermak et al. (1973) and Phaf et al. (2000) have indicated that Korsakoff patients do not use sophisticated (semantic) encoding strategies, unless they are explicitly instructed or requested to do so (cf. Van Damme & d'Ydewalle, 2008a; see also Butters & Cermak, 1980).

In sum, next to Korsakoff patients' explicit recollection deficit, two encoding aspects may be important to consider: First, deficient relational encoding might result from damage to medial temporal / hippocampal brain regions. Second, due to damage to the frontal lobes (see Hirst & Volpe, 1988), Korsakoff patients may not spontaneously use the same effortful, elaborative memory strategies as memory-intact controls do. However, as recent studies have revealed increased levels of DRM false memory in frontal lobe patients compared with controls (e.g., Melo, Winocur, & Moscovitch, 1999), the latter aspect might not be crucial in light of the present paradigm.

Korsakoff patients and the DRM paradigm

As pointed out by Dodson and Schacter (2001), the role of cognitive neuropsychology in research concerning the accuracy and distortion of memory has been rather limited. Despite the general interest in the clinical phenomenon of confabulation, few attempts have been made to combine a neuropsychological approach with the experimental study of false memory. Recently, however, this situation has begun to change. During the last decade, cognitive

neuropsychologists have started to look into the accuracy and distortion of the memory of amnesic patients, as well as other patient groups, including Alzheimer patients, frontal lobe patients, and schizophrenics (see Gallo, 2006, for an overview).

As confabulation is a well-known symptom of Korsakoff's syndrome, and damage to the frontal lobes has been linked to source monitoring errors and abnormally high levels of false memory (e.g., Budson, Sullivan, Mayer, Daffner, Black, & Schacter, 2002; Curran, Schacter, Norman, & Galluccio, 1997; Melo et al., 1999; Rapcsak, Reminger, Glisky, Kaszniak, & Comer, 1999; however, see also Bastin, Van der Linden, Lekeu, Andrés, & Salmon, 2006; Verfaellie, Rapcsak, Keane, & Alexander, 2004), increased false alarm rates to DRM critical lure words might also be expected in amnesic Korsakoff patients. On the other hand, in case of deficient relational encoding, one might rather predict decreased false memory compared with controls.

In 1973, using the continuous recognition paradigm of Underwood (1965), Cermak et al. found higher false alarm rates to associatively related lure words for Korsakoff patients than for alcoholic controls. In contrast with this early finding of increased false recognition, however, more recent studies using the DRM paradigm have revealed reduced levels of false memory in amnesic patients. Table 1 summarises the outcome of seven studies using (variations on) the DRM paradigm, ordered by type of memory test. Most of these studies used a mixed group of amnesic patients, rather than focusing on Korsakoff patients in particular, but found no differences in performance dependent on aetiology. For those studies in which differences between patient groups were obtained (e.g., Schacter et al., 1998b; Verfaellie, Schacter, & Cook, 2002), only Korsakoff patients' results are presented in Table 1.

Table 1 Overview of amnesic patients' performance in DRM memory tasks using word lists

Type of task	*Study*	*Type of list*	*Veridical memory*		*False memory*	
			Amnesics	Controls	Amnesics	Controls
Explicit retrieval						
Delayed recognition	Schacter et al. (1996c)	Associative	0.16*	0.66	0.16*	0.57
	Schacter et al. (1997b, Exp. 1)	Associative	0.21*	0.62	0.02*	0.42
		Perceptual	0.19*	0.57	0.06	0.18
	Schacter et al. (1997b, Exp. 2)	Associative	0.27*	0.64	0.16	0.24
		Perceptual	0.37*	0.75	0.12*	0.40
	Schacter et al. (1998b)	Associative	0.38*	0.79	0.22*	0.83
	Melo et al. (1999)	Associative	0.17*	0.56	-0.05*	0.66
	Verfaellie et al. (2002)					
	- Standard retrieval	Associative	0.28*	0.69	0.48*	0.68
	- Meaning retrieval	Associative	0.16*	0.60	0.33*	0.74
	Van Damme & d'Ydewalle (2008c)	Associative	0.34*	0.64	0.33*	0.75
		Categorised	0.48*	0.84	0.38*	0.57
Delayed free recall	Van Damme & d'Ydewalle (2008c)	Associative	0.03*	0.18	0.15*	0.43
		Categorised	0.04*	0.27	0.08*	0.32
	Van Damme & d'Ydewalle (2008b, Exp. 1)	Associative	0.01*	0.14	0.04*	0.34

Type of task	*Study*	*Type of list*	*Veridical memory*		*False memory*	
Delayed cued-recall	Verfaellie et al. (2005, Exp. 1)	Associative	0.13*	0.35	0.00*	0.27
	Van Damme & d'Ydewalle (2008b, Exp. 1)	Associative	0.30*	0.46	0.18*	0.39
Immediate recognition	Van Damme & d'Ydewalle (2008c)	Associative	0.78	0.91	0.86	0.77
		Categorised	0.90	0.95	0.79*	0.56
Immediate free recall	Schacter et al. (1996c)	Associative	0.27*	0.52	0.29	0.33
	Melo et al. (1999)	Associative	0.24*	0.52	0.19	0.41
	Van Damme & d'Ydewalle (2008c)	Associative	0.27*	0.47	0.30*	0.47
		Categorised	0.35*	0.59	0.26	0.32
Immediate cued-recall	Verfaellie et al (2005, Exp. 2)	Associative	0.36*	0.78	0.28*	0.44
	Van Damme & d'Ydewalle (2008b, Exp. 2)	Associative	0.37*	0.56	0.45*	0.65
Implicit retrieval						
Delayed stem completion	Verfaellie et al. (2005, Exp. 1)	Associative	0.14	0.20	-0.04*	0.13
	Van Damme & d'Ydewalle (2008b, Exp. 1)	Associative	0.19	0.22	0.07	0.12
Immediate stem completion	Verfaellie et al. (2005, Exp. 2)	Associative	0.32	0.33	0.01*	0.12
	Van Damme & d'Ydewalle (2008b, Exp. 2)	Associative	0.25	0.32	0.17	0.16

Recognition scores were calculated by subtracting the proportion of false alarms to study and critical distractors from the proportion of hits to the corresponding study words/critical lures. Priming scores were obtained by subtracting the proportion of stems completed to study words/critical lures from non-studied lists (i.e., the baseline completion rate) from the proportion of stems completed to study words/critical lures from studied lists. * = significantly different from controls ($\alpha = 0.05$). For Melo et al. (1999), the results presented reflect the performance of the amnesic group with frontal lobe damage and their control group. For Schacter et al. (1998b) and Verfaellie et al. (2002), the results presented only reflect the Korsakoff group and their controls.

Immediate recall – delayed recognition

Following the original procedure of Roediger and McDermott (1995), most experiments carried out so far have focused on immediate recall and delayed recognition: After studying and recalling several DRM lists one after the other, participants' recognition memory for list words and critical lures from all of the lists is tested.

In a pioneering experiment by Schacter et al. (1996c), this procedure was applied to amnesic patients (both Korsakoff and amnesics of mixed aetiology) and non-amnesic controls. As expected, the patients' veridical memory (recall as well as recognition) was strongly impaired compared with controls'. In addition and more importantly, the patients also showed a depressed false recognition rate, suggesting that they were less susceptible to the effect than control participants were. False recall, however, was approximately the same for patients and controls. Nevertheless, since amnesic patients produced more non-critical intrusions than controls did, both participant groups differed in their global pattern of free recall.

Subsequent studies tried to explain and extend Schacter et al.'s (1996c) findings. Melo et al. (1999) applied the same procedure to three distinct patient groups (non-amnesic patients with frontal lobe damage; amnesic patients with and without frontal lobe damage) and matched controls, and obtained similar results: Both groups of amnesic patients produced less false recognition than either frontal-lobe patients or controls. At free recall, amnesics with frontal damage in-

truded fewer critical lures (albeit non-significantly so) and amnesics without frontal damage intruded more critical lures than did controls.

Schacter, Verfaellie, and Anes (1997) included lists with perceptually related words in the DRM paradigm and, again, obtained similar results: Under conditions in which healthy participants showed high levels of false recognition, false recognition in amnesic (Korsakoff) patients was strongly reduced: After studying lists of perceptually as well as conceptually related words, patients were less likely than controls to correctly recognise list words and falsely recognise critical lures. Likewise, studies using abstract novel objects (Koutstaal, Schacter, Verfaellie, Brenner, & Jackson, 1999) and categorised common objects (Koutstaal, Verfaellie, & Schacter, 2001) revealed diminished false recognition of critical lures in amnesic (Korsakoff) patients, under conditions in which controls showed robust false recognition (although it should be mentioned that in the former study results concerning the subgroup of Korsakoff patients were less clear-cut due to large baseline differences with their respective controls).

Schacter, Verfaellie, Anes, and Racine (1998) repeated the study-test procedure of the DRM paradigm five times, and obtained quite surprising results. With repeated study and test trials, both amnesic patients and non-amnesic controls correctly recognised increasingly more list words. However, while controls' false recognition level decreased across the five study-test trials, Korsakoff patients falsely recognised increasingly more critical lures. A mixed amnesic group, on the other hand, showed a fluctuating false recognition pattern.

To account for all of these findings, Schacter et al. (e.g., 1996c, 1997b, 1998b) formulated the following explanation, based on fuzzy-trace theory: When presented with many words converging on a central theme, healthy participants 'bind together' studied items and generated associations, thereby creating a well-organised, focused representation of the theme of the list. At test, both list words and related distractors matching this thematic representation will invoke a strong sense of familiarity or recollection, and hence, are likely to be judged as old. Amnesic patients, on the other hand, form or retain a much poorer, degraded representation of the theme of the list, rendering false recognition of related lures less likely.

Although offering an important first step in explaining the data obtained, this explanation actually leaves room for two alternative interpretations (or a combination of both): Either amnesic patients do not encode/store a sufficiently organised thematic representation (due to deficient relational and/or elaborative processing), or the thematic information is not available to them at test due to impaired explicit retrieval (see also Verfaellie, Page, Orlando, & Schacter, 2005; Verfaellie et al., 2002).

So, on the one hand, it could be that amnesic patients are less likely than controls to (spontaneously) use elaborative, relational processing strategies at study. Such a deficit would imply that the patients are less likely than controls to engage in associative processes, and to extract the gist of the lists. Hence, the necessary semantic information is not (or at least less) available to them, because they do not *encode* a well-organised thematic representation. On the other hand, however, it could also be that the amnesics are just not able to *retrieve* the (well-encoded) thematic information at test, just as they have problems with consciously retrieving specifically studied list words. As all previously reported experiments used explicit instructions at the time of memory testing (free recall and/or recognition), direct reference was made to past learning and amnesic patients were required to intentionally and consciously recollect the studied lists. This implies that not only the list words, but also the general theme of the lists needed to be remembered explicitly, and that the amnesic false memory deficit might have been due to faulty intentional retrieval.

Schacter et al.'s (1998b) finding of increased veridical and false recognition in Korsakoff patients with repeated study-test trials can be explained by each one of these alternatives. According to the first option, depressed false recognition rates on the first trial were due to deficient relational encoding. Repeated exposure to the study and test lists will then provide additional opportunities to encode the associative relationships between the list words, and to store an increasingly better structured gist representation. Hence, increasingly higher levels of false memory will be obtained. According to the second option, however, depressed false recognition rates on the first trial were due to deficient explicit recollection. Repeated exposure to the study and test lists will then induce an increasingly stronger feeling of familiarity, leading to increasingly higher false recognition levels in the Korsakoff patients. Non-amnesic controls, on the other hand, will use recollection-based monitoring processes to counteract this feeling of familiarity, leading to decreasing levels of false memory.

In a recent study, Verfaellie et al. (2002) tried to disentangle the possibilities of degraded gist encoding and impaired gist retrieval by comparing amnesic and control performance in two distinct retrieval conditions: 'Standard' recognition instructions focused on the retrieval of specific study words, whereas 'meaning retrieval' instructions encouraged participants to rely on gist information and to accept any item that matched the general theme of the study list as old. As the patients' false recognition rate remained reduced in the meaning retrieval condition, Verfaellie et al. concluded that amnesics'

impairment in gist memory is not attributable to a failure to access well-formed gist representations at test, but rather to a deficit in the encoding (or maintenance) of gist information. Importantly, however, the task given to the participants still comprised explicit memory instructions, and hence required the intentional retrieval of gist information. Therefore, the conclusion drawn might not be justified based on the results obtained.

Immediate recognition – delayed recall

As previous studies did show strongly reduced false recognition, but not necessarily reduced false recall in amnesic patients (Melo et al., 1999; Schacter et al., 1996c), Van Damme and d'Ydewalle (2008c) hypothesised that the delay of testing might be a crucial element in explaining Korsakoff patients' false memory. Hence, an experiment was carried out in which the order of recall and recognition tests was manipulated between-subjects: Half of the participants received eight immediate free recall tests and one delayed recognition test, whereas the other half received eight immediate recognition tests and one delayed free recall test. It was expected that Korsakoff patients would be able to retrieve (some of) the gist of the lists on the basis of automatic and short-term memory processes at immediate testing, but much less when testing was delayed until the end of the experiment. In addition, both associative and categorised word lists were included in the experiment, in order to investigate the possibility of an encoding deficit. As Smith, Gerkens, Pierce, and Choi (2002) showed that associative false memories can be attributed to semantic processes during study (i.e., due to high backward association strength), whereas categorical false memories can be attributed to the use of semantic knowledge during test (see also Koutstaal & Schacter, 1997), it was expected that an encoding deficit would cause a larger difference in false memory between patients and controls with the former than with the latter kind of list.

Results revealed a smaller difference in false recall between patients and controls at immediate than at delayed testing, as was expected. Moreover, in the immediate recognition task, Korsakoff patients obtained even higher false memory scores than control participants did (cf. Cermak et al., 1973). This could be attributed to familiarity-based retrieval, unopposed by explicit recollection. No convincing evidence was obtained for an encoding deficit. Rather on the contrary, impaired retrieval could account for all of the data obtained, whereas the encoding deficit hypothesis was consistent with some, but inconsistent with other data. Nonetheless, the possibility that Korsakoff patients do suffer from some sort of encoding problem in addition to their explicit retrieval deficit could not be excluded on the basis of the results obtained.

Stem completion

As stated before, the DRM paradigm traditionally includes explicit retrieval instructions: Participants are asked to intentionally remember previously studied information. By now, however, several studies (e.g., McKone & Murphy, 2000) have shown that implicit retrieval instructions can also be used to elicit DRM false memories. Such instructions do not refer to the study phase, but rather focus on the (non-memory) task at hand, and hence reflect automatic rather than deliberate retrieval of information from memory.

In order to further investigate both of the earlier described alternatives, Verfaellie et al. (2005) evaluated implicit false memory in amnesic patients (both Korsakoff and other) and matched controls. They argued that, if earlier findings of impaired false memory in amnesics were due to the typically used explicit retrieval instructions, then the patients should show normal implicit memory for critical lures. Alternatively, if the impairment in amnesics' false memory was due to an inability to form well-structured gist representations at study, it should persist even under implicit retrieval instructions. Participants were presented with eight DRM lists, after which memory was tested by means of both implicit stem completion and explicit cued-recall (either following presentation of all study lists in Experiment 1, or immediately following each separate list in Experiment 2). Amnesic patients did show intact priming for studied list words, but failed to show priming for critical lures. So, whereas veridical memory for studied items was impaired only when tested explicitly, false memory for critical lures was impaired regardless of the retrieval instructions used. Hence, the authors concluded that previous findings of depressed false memory in amnesia must be due to an encoding deficit rather than to problems with explicitly retrieving the gist of the lists at test.

However, as certain methodological aspects in Verfaellie et al. (2005) made it difficult to reach a straightforward conclusion, Van Damme and d'Ydewalle (2008b) tried to replicate their results in two experiments using a slightly different design and procedure. In addition to some methodological changes, encoding instructions emphasising the relational nature of the materials were added in Experiment 1, and varying encoding durations were used in Experiment 2. Both manipulations were meant to support Korsakoff patients' (relational) encoding during the study phase. Unexpectedly, emphasising the relational nature of the lists (Experiment 1) did not influence the level of false memory obtained in patients nor controls. Providing participants with longer study durations (Experiment 2) significantly improved their explicit memory for the list words, and also slightly enhanced explicit memory for the gist of the lists. Most importantly, however, Korsakoff patients' *implicit* true and false memory scores did not differ from con-

trols' in both experiments. When tested under explicit retrieval instructions, on the other hand, true and false memory were significantly impaired. Hence, in contrast to Verfaellie et al.'s findings, retrieval instructions influenced amnesic memory for studied list words and for unstudied critical lures in similar ways. This does not point to an encoding deficit, but rather suggests that deficient recollection might have been the crucial factor determining the earlier found reduced false memory levels.

It should be noted that, similar to Verfaellie et al. (2005), the implicit false memory effect in Experiment 1 of Van Damme and d'Ydewalle (2008b) was only significant for control participants, whereas it was not for Korsakoff patients. Nevertheless, rather than reflecting some kind of encoding problem, this pattern seemed to result from the presentation duration used during the study phase: In Experiment 2, controls as well as patients did not show significant false priming with the standard presentation rate of 2000 ms, whereas they both did when presentation rates of 250 ms or 5000 ms were used.

In sum, considering both Verfaellie et al. (2005) and Van Damme and d'Ydewalle (2008b, 2008c), no clear-cut evidence for an encoding deficit in amnesic patients was obtained. Rather, impaired intentional retrieval of thematic (as well as item-specific) information appears to be the main cause of earlier findings of diminished false memory. Of course, the apparent difficulties to obtain significant priming for critical lures under implicit retrieval instructions (cf. McBride, Coane, & Raulerson, 2006) substantially complicate the interpretation of participants' performance. Further studies are needed to clarify the pattern of results. In light of the earlier made suggestion that the degree of (relational) encoding required to demonstrate normal explicit memory might be higher than what is needed to demonstrate normal implicit memory, it is important to mention that in Experiment 2 of Van Damme and d'Ydewalle (2008b), Korsakoff patients' explicit memory scores improved up to the level of controls' when they were given additional study time. Hence, the patients' explicit recollection deficit might – at least to some extent – be due to deficient encoding.

Summary and concluding remarks

Studying illusory memories from a neuropsychological viewpoint can contribute to the understanding of how 'normal' memories can be distorted. As pointed out by Schacter and colleagues (e.g., 1998b), the finding that amnesic patients show impairments in false as well as true memory suggests that the medial temporal/diencephalic regions that mediate true memory are also important for the storage and retrieval of the information that drives false memory. However, as long as it is not entirely clear to what extent false memory for gist information is impaired/preserved in these patients, it is impossible to make strong claims about specific neuroanatomical correlates.

Early findings of experiments using the 'original' DRM paradigm with free recall and recognition tests all revealed decreased false memory in amnesic (Korsakoff) patients. Based on the explanation provided by Schacter et al. (e.g., 1996c, 1997b, 1998b) in terms of degraded gist representations, subsequent studies were designed to evaluate the extent to which encoding deficits on the one hand, and explicit/strategic retrieval deficits on the other hand, were involved in causing these findings. Results were mixed. Whereas the evidence for the involvement of an explicit recollection deficit was quite strong and recurring, the evidence for the involvement of an encoding deficit was inconclusive and ambivalent. Nevertheless, none of the experiments succeeded in excluding the possibility of impaired encoding. Whereas the evidence obtained by Verfaellie at al. (2005) pointed unequivocally to an encoding deficit as the main cause of deficient explicit *and* implicit false memory, the results of Van Damme and d'Ydewalle (2008b) rather suggested that relational encoding in Korsakoff patients is sufficiently good to obtain normal *implicit* memory, but might be too weak to induce high levels of *explicit* false memory. Most likely, of course, both aspects do play a role. Future experiments will need to determine to what extent and in which situations one of them might be more important than the other.

Recent neuroimaging data have revealed quite similar patterns of brain activity during both veridical and false recognition in healthy young individuals (see Gallo, 2006, for a comprehensive review): First, evidence of medial temporal lobe (including hippocampal) activation has been demonstrated during both types of recognition (Cabeza, Rao, Wagner, Mayer, & Schacter, 2001; Schacter, Buckner, Koutstaal, Dale, & Rosen, 1997; Schacter et al., 1996b; see also Schacter & Slotnick, 2004). As explained before, the medial temporal brain region is involved in both conscious recollection and relational encoding, and damage to this region is part of the neuropathology of Korsakoff's syndrome. Hence, its activation during false recognition in healthy subjects is in agreement with the false memory deficit found in Korsakoff patients in situations where explicit recollection of studied information is required. Secondly, neuroimaging data have also indicated prominent frontal lobe involvement in both veridical and false recognition (Cabeza et al., 2001; Schacter et al., 1996b, 1997a). As Schacter et al. (1997a) provided evidence for a late onset of this anterior prefrontal activity relative to other brain areas, it most likely reflects processes operating on the output of the memory system, such as monitoring or decision making. This is in accordance with findings of height-

ened false memory in frontal lobe patients (e.g., Melo et al., 1999), as well as with Korsakoff patients showing increased false memory compared with controls in situations where familiarity can be used to fulfil the task at hand, such as the immediate recognition task in Van Damme and d'Ydewalle (2008c).

Finally, it should be noted that, whereas most of the explanations and interpretations throughout this article were inspired by fuzzy-trace theory, the activation-monitoring account is equally reconcilable with all of the arguments made. According to such an account, critical lures are mentally activated during study (either coming to mind consciously or through the unconscious spreading of activation), after which participants have difficulties distinguishing them from items they actually studied (i.e., source confusion). Both encoding problems and explicit retrieval difficulties fit within this framework and could easily be applied to explain diminished false memory. An additional aspect to consider would then be whether diminished encoding implies defective (unconscious) spreading of activation, or rather a failure to consciously produce the critical lure during the study phase. Hopefully, future studies will be able to disentangle these two alternatives and to enlarge our knowledge about amnesic patients' false memory in particular and memory processes in general.

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