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Lack of false recognition in schizophrenia: a consequence of poor memory?

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Abstract

The tendency to falsely recognize items as ones previously presented is increased in patients with frontal lesions and in older participants, whereas patients with medial temporal lobe damage may display such poor memory that they are not especially susceptible to false recognition. Since patients with schizophrenia are often compared to these groups neurocognitively, we explored the extent to which they are more susceptible to false memory. Participants were presented with word lists along a semantic theme, such as "bread". After list presentation, recognition tasks were administered which contained both the studied words as well as unstudied words. Some of the unstudied words were related to the theme of the previously studied words, but never actually presented (e.g. semantic "lures"). In a separate test, free recall of these lists of words was assessed. Interestingly, it was control participants who made more errors at recall, and were especially susceptible to intrusions of the semantic lures. Patients with schizophrenia did not make more false recognition errors in general, and surprisingly they made disproportionately fewer false recognition errors to semantic lures specifically. We conclude that despite poor memory, patients with schizophrenia are not especially susceptible to interference from previous tasks and are not particularly prone to false recollections.
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Keywords: False memory; Recall; Recognition; Schizophrenia

1. Introduction

Memory generally functions with a high degree of accuracy across a variety of situations. However, it is far from perfect, and at times can be extremely inaccurate. These inaccuracies occur because memories are not exact replicas of the stimuli that impinge upon the senses; rather they are the result of transformations and interpretations of these stimuli. Memory distortions may be defined as memory of events that never happened, or remembering events in a markedly altered way. Examining this process of distortion may enhance our understanding of memory encoding, storage and retrieval (Bartlett, 1932).

Korsakoff (1899a,b) observed memory distortions in patients who had severe amnesia for recent and remote events as a result of alcohol abuse (Mayes, 1988). Bonhoeffer (1904) additionally observed that these so-called Korsakoff syndrome patients recollected events that never occurred (i.e. confabulations). Confabulation has been observed in a variety of pathological conditions. Indeed, patients with frontal lesions (Janowsky, Shimamura. & Squire, 1989), as well as older individuals (Norman & Schacter, 1997) (especially those with dementia; Balota et al., 1999), have been shown to be especially susceptible to false recognition or recall. A neuropsychological examination of false recall and recognition in amnesics (in patients with damage to the medial temporal lobe or diencephalic regions (MTL/D), and patients with frontal lobe and medial temporal lobe damage) and non-amnesic patients with frontal lobe damage, found that both frontal lobe and MTL/D structures are important in accurate memory performance (Melo, Winocur, & Moscovitch, 1999, see also Schacter, Verfaellie, & Pradere, 1996). Specifically, if output from the medial temporal lobe is inaccurate and the frontal lobe is not able to monitor it, memory distortions are likely. When these structures are damaged, recall and recognition is hindered since patients are not able to readily access information related to target items. The consequence of this impairment is a reduction of the number of both true and false memories. Damage to

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MTL/D structures leads patients to rely on the gist, rather than a specific memory of the items, whereas damage to the frontal lobes (in certain regions) may disrupt the recollection process by interfering with the monitoring process that extracts the gist (indeed, damage to the right frontal lobe is often associated with confabulations; Parkin, 1997).

Episodic memory is central to explicit recollection of past experiences. Impairment in this domain is characteristic of schizophrenia, although not of all patients (for a review, see Elvevåg & Goldberg, 2000). Some of the memory deficits observed in schizophrenia are similar to those seen in patients with damage to the medial temporal lobe. However, an alternative possible cause for the deficits in schizophrenia is frontal lobe dysfunction, which in turn would give rise to problems either in encoding of information or effortful retrieval, and thus lead to memory impairments. Indeed, although frontal lobe dysfunction is often considered to be a key feature of schizophrenia (for a review, see Weinberger et al., 2001), medial temporal lobe abnormalities are also associated with schizophrenia (for a review, see Weinberger, 1999), which is consistent with schizophrenic patients' widespread episodic memory problems that are probably dependent on these latter structures (Squire, 1992).

Deese (1959) examined healthy individuals who studied lists of words that were strong associates of a non-studied critical item. He found that participants often falsely recollected the non-studied but strongly associated items at recall. This paradigm offers a powerful method for inducing false recollections (Roediger & McDermott, 1995; Underwood, 1965), and hence there has been renewed interest in this task, recently modified by Roediger & McDermott (1995). In this new version of the task, participants initially study lists of words (e.g. butter, food, eat, sandwich, rye, flour, dough, crust, slice etc.) that are based on associates of a critical, but non-presented word (bread). Then, after the study-phase, participants are given recall and/or recognition tasks, which include lists of previously studied words as well as non-studied words (which include the critical non-studied theme words or semantic lures, such as bread). This design has been shown to produce a very high level of false positives (i.e. claiming recognition of words that were not on the list; e.g. bread) in the recognition task with healthy normal participants. By employing a similar paradigm, we sought to establish whether patients with schizophrenia would produce more false positives than controls during recall and recognition tasks in which lures share a semantic relation to studied items.[1]

[1] We note that a recent study looking at false recognition in schizophrenia found an increased level of false recognition in patients (Weiss, Dodson, Goff, Schacter, & Heckers, 2002). However, the paradigm that was employed was rather different to the current design (namely auditory presentation of study items with either text or pictures accompanying) and was specifically designed to examine the effects of repeated foils on subsequent recognition, and thus required participants to recollect the source information regarding previous exposure to the stimulus.

2. Methods

2.1. Participants

Twenty-two inpatients from the National Institute of Mental Health research wards participated (see Table 1). All patients fulfilled DSM-IV criteria for schizophrenia, as determined by the Structured Clinical Interview for DSM-IV (SCID) with three psychiatrists reaching a consensus diagnosis. Patients generally had multiple hospital admissions due to incomplete responses to conventional treatments. Twenty-one of the patients were receiving neuroleptic medication at the time of the study (15 on clozapine or olanzapine, 6 on risperidone and 3 on high potency neuroleptic drugs such as haloperidol, fluphenazine, or loxapine). One patient was receiving only a mood stabilizer at the time of testing. Six patients also received anticholinergic medication, and 13 were on adjunctive medication of various types. Twenty-five normal healthy control volunteers were recruited through the National Institutes of Health volunteer panel and were paid for their participation. Exclusion criteria for all participants included a history of traumatic brain injury, epilepsy, developmental disorder, diagnosable current substance dependence, or other known neurologic condition. All participants had normal or normal corrected vision. The internal review board at the National Institute of Mental Health approved this study and informed consent was obtained from all participants prior to testing.

2.2. Neuropsychological evaluation

2.2.1. Intelligence

Two tests were used to index intellectual function (see Table 1). The first was a test of reading proficiency—the Wide Range Achievement Test-Revised Reading (WRAT-R; Jastak & Wilkinson, 1984), which is standardly used as a putative measure of premorbid intellectual functioning

Table 1
Characteristics of patient and control samples

	Patients, $n = 22$ (17M, 5F)		Controls, $n = 25$ (9M, 16F)	
	M	S.D.	M	S.D.
Age (years)	33.4	7.8	34.6	12.6
WRAT-R IQ	105.6	10.5	110.0	8.7
WAIS-R IQ*	93.1	13.9	107.9	11.9
Neuroleptic medication	21	0		
Clozapine/olanzapine	15	–		
Risperidone	6	–		
High potency drugs[a]	3	–		
Anticholinergics	6	–		
Adjunctives[b]	13	–		

[a] Haloperidol, fluphenazine, loxapine.
[b] Lithium, depakote, sertraline, lorazepam, venlafaxine, clonazepam, buspirone.
* $P < 0.01$.

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