



The role of recollection in source memory: An examination of schizophrenia patients and their first-degree relatives

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ABSTRACT

Source recognition memory deficits have repeatedly been observed in people with schizophrenia (SZ), and have also recently been observed in their first-degree relatives. These deficits have been hypothesized to result, at least in part, from impairments in the conscious recollection process. Although other processes are clearly also affected in SZ, it has been proposed that impairments in the conscious recollection process could be a parsimonious explanation for the source memory deficits observed in their relatives.

Here, we tested 25 patients with SZ and 34 of their non-affected parents, as well as two groups of matched healthy controls, on a short-term associative memory task that shares the characteristics of standard source recognition tasks but minimizes the need for recollection of stored information from memory. This task was administered in order to determine if deficits can still be observed in these people when involvement of the conscious recollection process is minimized.

We observed deficits on our short-term source memory task in people with SZ, but their first-degree relatives did not share this deficit. These results support the idea that multiple memory processes supporting associative/source memory are affected in SZ, whereas the source memory deficits previously observed in relatives of SZ seem specific to tasks that rely on the conscious recollection process.

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1. Introduction

Several aspects of cognition are impaired in patients suffering from schizophrenia (SZ) and among them, episodic memory is one of the most consistent and significant deficits (Heinrichs & Zakzanis, 1998; Saykin et al., 1991). This key cognitive function is considered as a potential endophenotype of SZ in part because of its impairment in unaffected relatives (Gur et al., 2007; Snitz, Macdonald, 3rd, & Carter, 2006). The magnitude of the impairment observed in patients with SZ or in their relatives however varies between studies and paradigms (Pelletier, Achim, Montoya, Lal, & Lepage, 2005; Snitz et al., 2006). For instance, people with SZ show relatively greater impairments when episodic memory is assessed by means of associative recognition tests, that require the encoding and later retrieval of a series of associations (between an item and another item or between an item and an associated characteristic), relative to item recognition tests in which individual items are rather presented (Achim & Lepage, 2003; Lefebvre et al., 2009; Le-

page et al., 2006; Pelletier et al., 2005). Such associative or source memory deficits have been observed across different types of associations (Achim & Weiss, 2008) and are often interpreted as reflecting impairments in the conscious recollection process (Lefebvre et al., 2009; Yonelinas, 2002), though other processes such as binding during memory encoding and post-retrieval monitoring during retrieval also seem particularly relevant to associative memory performance (Achim & Lepage, 2005a).

In a previous study (Lefebvre et al., 2009), we observed associative memory deficits in SZ as well as in their first-degree relatives for two different types of associations, namely item–item and item–location associations. Item recognition memory was also assessed in this study, revealing that the deficits observed in first-degree relatives was very selective to the associative memory conditions (i.e. was not accompanied by an item recognition deficit). This pattern of results suggested that impairments in conscious recollection of information from memory could represent an endophenotype of SZ.

Processes other than conscious recollection are however also particularly relevant for associative memory performance, including the binding of distinct pieces of information into a coherent whole during encoding, as well as monitoring and evaluation

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processes during retrieval. This is not to say that recollection is not affected in patients with SZ and/or their relatives, but it is nonetheless unclear whether this conscious recollection deficit represents a parsimonious explanation for the pattern of results.

In order to test whether conscious recollection of stored information can fully explain the associative memory deficits previously observed in patients with SZ or their relatives, one approach would be to devise a task that shares the characteristics of the associative memory tasks that have previously revealed deficits in these people, except for the need to recruit the conscious recollection process. One way to minimize the need for recollection of stored information is to rely on set sizes that favor maintenance of information online (Mitchell & Johnson, 2009; Mitchell, Johnson, Raye, & Greene, 2004) i.e., rely on a short-term associative memory task. In other words, when information is maintained online (as should be the case in short-term memory tasks), then there is no need to retrieve it from the pool of stored information because it is already in an active state.

Deficits have previously been reported in a few studies relying on short-term associative memory paradigms (also known as source memory paradigms) in patients with SZ (Burglen et al., 2004; Salame, Burglen, & Danion, 2006), supporting the idea that processes other than those involved in recollection of information from memory are also impaired in SZ. These previous studies were however limited to item–location associations, i.e., they did not assess other types of associations such as item–item or item–temporal context associations, due to which the generalization of deficit to other types of associations cannot be assessed. Furthermore, these studies included samples of relatively chronic patients, which can be biased towards the inclusion of more affected and more cognitively impaired patients, a bias that can be avoided by recruiting recent-onset patients. Hence, a first aim of the study was to assess the presence of impairments in short-term associative memory performance in people with recent-onset SZ spectrum psychotic disorders (SZSPD) for item–location as well as item–temporal context associations.

Most importantly, the second aim was to determine whether relatives of people with SZSPD share the short-term associative memory deficit previously reported in SZ (Burglen et al., 2004; Salame et al., 2006). If such deficits were observed in relatives of people with SZSPD, it would indicate that processes other than conscious recollection are also at play that could represent alternative potential endophenotypes of SZ. On the other hand, the absence of short-term associative memory deficits in relatives of SZSPD patients would support our initial proposition (Lefebvre et al., 2009) that the associative memory deficit previously observed in these people (i) is specifically linked to the memory retrieval phase (because it is more prominent for long-term than short-term memory) and (ii) involves the conscious recollection process (because the long-term memory deficit is specific to associative recognition as shown in Lefebvre et al., 2009).

2. Method

2.1. Participants

We recruited 25 recent-onset (i.e. less than 5 years since the first contact with psychiatric services) and clinically stabilized patients meeting DSM-IV (American Psychiatric Association, 1994) criteria for SZ ($n = 21$) or SZ spectrum psychotic disorders¹ (schizophreniform disorder ($n = 3$), or delusional disorder ($n = 1$)). Patients were recruited from Clinique Notre-Dame-des- Victoires, an outpatient clinic that provides services to patients experiencing a first-episode of psychosis. At the time of testing, all patients were

taking a second-generation antipsychotic as their primary medication (see Table 1 for socio-demographic and clinical characteristics of the samples). Since the patients were stabilized and relatively well-functioning recent-onset outpatients, any deficits observed in the present study would be unlikely to result from sampling biases potentially affecting samples of prevalent chronic cases, for instance bias toward recruitment of patients with poorer outcome, or from a perturbation related to acute psychotic symptoms.

Thirty-four of their unaffected parents were also included in the study (for 9 patients, both parents accepted to participate). Patients and their unaffected parents were recruited from the Clinique Notre-Dame-des-Victoires (affiliated to the Institut Universitaire en Santé Mentale de Québec, Québec city), a specialized clinic that treats patients with recent-onset psychotic disorders.

Two comparison groups were also recruited through advertisements in local newspapers, dental, and medical offices. The first control group comprised 25 persons matched for gender and age to the patients. The second control group comprised 34 persons matched for gender and age to the unaffected parents. In addition to being matched in terms of gender and age, the controls of unaffected parents were also matched to the parents for their number of years of educations. Because SZ often occurs in young adults and can interfere with patient's education achievements, the average education level for the controls of patients was also matched to that of the unaffected parents instead of that of the patients.

Exclusion criteria for all participants were (i) a history of alcohol or substance abuse/dependence in the 6 months preceding the study, (ii) a history of a neurological illness, (iii) or of a head injury with a loss of consciousness greater than 5 min, (iv) current use of benzodiazepine medication on a daily basis, (v) an estimated IQ lower than 70, as measured with a dyad (Vocabulary and Block Design; Jeyakumar, Warriner, Raval, & Ahmad, 2004) from the Wechsler Adult Intelligence Scale (WAIS-III; Wechsler, 1997), (vi) age less than 18 or greater than 60 years. In addition, we excluded controls or parents with a history of psychotic disorders, as assessed with the SCID-IV (Spitzer, Gibbon, & Williams, 1995), or with a cluster A Axis II disorder (paranoid, schizoid or schizotypal), as assessed with the SCID-II-Q (Spitzer, Williams, Gibbon, & First, 1990). We also excluded controls if such a diagnosis was suspected in any of their first-degree relatives.

In patients, symptom severity was rated by their psychiatrist using the Positive and Negative Syndrome Scale (PANSS; Kay, Fiszbein, & Opler, 1987) and the Social and Occupational Functioning Assessment Scale (SOFAS; Goldman, Skodol, & Lave, 1992) was used to assess global level of social functioning.

The socio-demographic characteristics of all participants as well as the clinical characteristics of the patient group are presented in Table 1. All participants had taken part in our previous associative memory study (Lefebvre et al., 2009).

The local ethics committee approved this study, attesting of its accordance with the Helsinki Declaration. All participants gave their written consent after receiving a detailed description of the research protocol and were offered a compensation of 20\$ CAN for their participation.

2.2. Experimental task

The experimental task included two conditions (see Fig. 1 for a schematic representation). The first condition (*where*) was aimed to assess memory for item–spatial context associations whereas the second condition (*when*) was designed to assess memory for item–temporal context associations. The sequence of presentation was counterbalanced between participants. Each condition included two practice trials and 24 experimental trials. For each trial, six letters were sequentially presented, each in a different location on a computer screen. The strings of six letters were formed from a

¹ Kendler, Neale, and Walsh (1995), among others, demonstrated that these disorders are manifestations of the same underlying genetic vulnerability.

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