Verbal memory processes in schizophrenia patients and biological relatives of schizophrenia patients: intact implicit memory, impaired explicit recollection

Scott R. Sponheim, Vaughn R. Steele, Kathryn A. McGuire

Abstract

Verbal memory deficits are arguably the most common cognitive abnormalities in biological relatives of schizophrenia patients. Because verbal memory is a complex cognitive function, it is necessary to differentiate its intact and compromised aspects in order to reveal aberrant neural systems that reflect genetic risk in relatives of schizophrenia patients. Using an experimental verbal memory task, we examined encoding, free-recall, repetition priming, and recognition of verbal material in 22 schizophrenia patients, 22 first-degree biological relatives of schizophrenia patients, and 23 nonpsychiatric control participants. Schizophrenia patients exhibited intact repetition priming, but worse size judgment task performance (encoding), recall, and recognition than the control participants. Biological relatives of schizophrenia patients exhibited intact size judgment task performance, repetition priming, and recognition, but a free-recall deficit. Although size judgment task performance at encoding was associated with recall of verbal material in schizophrenia and control groups, in the relative group encoding performance was associated with the degree of repetition priming. Findings are consistent with impaired explicit recollection of verbal material, but intact implicit verbal memory in schizophrenia patients and biological relatives of schizophrenia patients.

Keywords: Schizophrenia; Verbal memory; Genetic risk; Priming; Explicit memory

Verbal memory deficits are among the most severe cognitive impairments in schizophrenia (Aleman et al., 1999; Gold et al., 1992; Paulsen et al., 1995; Stone et al., 1998) and are frequently identified in first-degree biological relatives of schizophrenic patients (Cannon et al., 1994; Conklin et al., 2002; Egan et al., 2001; Faraone et al., 1995, 1999, 2000; Goldberg et al., 1990; Keri et al., 2001; Kremen et al., 1994; Laurent et al., 1999; Lyons et al., 1995; O'Driscoll et al., 2001;
Toomey et al., 1998; Toulopoulo et al., 2003a). Results of two recent magnetic resonance imaging studies suggest that these deficits may reflect brain abnormalities that are associated with genetic risk for schizophrenia. Seidman et al. (2002) found small left hippocampal size co-occurs with impaired verbal memory performance in first-degree relatives of schizophrenia patients. The association was stronger in relatives with more than one family member affected by the disorder. O’Driscol et al. (2001) found first-degree relatives of schizophrenia patients to have bilaterally small amygdala-anterior hippocampal volumes in contrast to normal posterior hippocampus size. The anterior reduction was associated with verbal memory performance. In both studies, investigators assessed verbal memory using an overall index of how accurately subjects reiterated a story that had been read to them (i.e., Logical Memory Test). Although useful in documenting verbal memory impairment, clinical neuropsychological tests generally do not allow delineation of cognitive processes that reflect specific neural mechanisms. To extend use of cognitive indices as measures of brain abnormalities in schizophrenia, researchers must break gross cognitive functions into components relevant to specific neural systems. Within verbal memory, implicit processes such as priming appear determined by cortical regions while explicit processes such as recollection more strongly involve hippocampal and medial temporal brain regions. In an attempt to parse verbal memory into components associated with neural systems, we employed an experimental verbal memory paradigm to characterize encoding, recall, repetition priming, and recognition of words by schizophrenia patients and first-degree biological relatives of schizophrenia patients.

Verbal memory deficits observed in relatives of schizophrenia patients are qualitatively similar to those of schizophrenia patients, but less severe (Egan et al., 2001; Faraone et al., 1995; Kremer et al., 1994; Lyons et al., 1995; Toomey et al., 1998). Overall, the deficits are greater in relatives having more than one individual with schizophrenia in the family (Faraone et al., 2000), have been found to persist over a 4-year period (Faraone et al., 1999), are present in unaffected monozygotic twins of schizophrenia patients (Goldberg et al., 1990), and are evident in relatives free of psychosis (Conklin et al., 2002; Faraone et al., 1995, 2000; Lyons et al., 1995; Roxborough et al., 1993), schizophrenia spectrum disorders (Goldberg et al., 1990; Seidman et al., 2002; Toulopoulo et al., 2003a), and virtually all forms of psychopathology (Keri et al., 2001; Laurent et al., 1999; O’Driscol et al., 2001; Toulopoulo et al., 2003b). Although these findings are suggestive of verbal memory dysfunction marking genetic risk for schizophrenia, verbal memory deficits have been found in first-degree relatives of bipolar patients (Keri et al., 2001). Some analyses have revealed evidence of little genetic influence on overall recall of verbal material in first-degree relatives of schizophrenia patients (Cannon et al., 2000; Tuulio-Henriksson et al., 2002), but significant genetic effects on semantic clustering, learning strategy, and recall errors (Tuulio-Henriksson et al., 2002). Varying genetic influences across memory indices may arise from measures tapping different aspects of verbal memory that depend on distinct neural mechanisms. For instance, studies have shown relatives of schizophrenia patients to exhibit little or no deficit in recognizing verbal material as compared to free recall of the same material (Conklin et al., 2002; Keri et al., 2001). Relatively preserved recognition performance may reflect the general ease of recognition tests compared to recall, or the effects of priming. Although recent studies indicate perceptual priming is unlikely to support recognition memory conceptual priming may increase the familiarity of material and thus enhance recognition (see Wagner and Gabrieli, 1998 for a review). Impaired explicit recollection (i.e., declarative memory or recall) likely reflects abnormalities in medial-temporal and hippocampal regions while intact priming suggests that aspects of neocortical function are preserved (Donaldson et al., 2001; see Squire and Knowlton, 2000 for a review). Several studies have shown schizophrenia patients to exhibit intact priming (Clare et al., 1993; Gras-Vincendon et al., 1994; Perry et al., 2000; Randolph et al., 1993). Although some have raised the possibility that schizophrenia patients and their relatives experience diminished priming (Kremen et al., 1994), to our knowledge there are no published studies of priming during verbal memory in relatives of schizophrenia patients. Priming is typically measured through effects on reaction time during a task that does not require explicit recollection of previously presented material (see Marsolek, 2003; Wagner and Koutstaal, 2002). By measuring the reaction time difference between
دریافت فوری متن کامل مقاله

امکان دانلود نسخه تمام متن مقالات انگلیسی
امکان دانلود نسخه ترجمه شده مقالات
پذیرش سفارش ترجمه تخصصی
امکان جستجو در آرشیو جامعی از صدها موضوع و هزاران مقاله
امکان دانلود رایگان ۲ صفحه اول هر مقاله
امکان پرداخت اینترنتی با کلیه کارت های عضو شتاب
دانلود فوری مقاله پس از پرداخت آنلاین
پشتیبانی کامل خرید با بهره مندی از سیستم هوشمند رهگیری سفارشات