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Increased automatic spreading of activation in thought-disordered schizophrenic patients

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Abstract

Previous research on semantic priming in schizophrenia has produced conflicting findings. While several studies provided evidence for an enhanced cognitive spreading of activation in schizophrenia patients with formal thought disorder, other research has failed to replicate. The aim of the present study was to resolve some of the ambiguities in the literature. Thirty-two schizophrenic patients (12 with and 20 without symptoms of formal thought disorder according to the PANADSS) and 65 healthy controls were compared in a semantic priming task using word pronunciation. Irrespective of baseline condition (neutral or unrelated condition) patients with formal thought disorder (TD) exhibited a significantly greater indirect semantic priming effect than both non-TD patients and healthy controls. Known confounding variables such as length of illness, neuroleptic dosage and psychomotor slowness did not moderate priming. Results further strengthen the spreading activation model of formal thought disorder put forward by Maher/Manschreck and Spitzer. Data indicate that hyper-priming is not confined to lexical decision tasks. Possible reasons why several studies have failed to replicate greater priming in TD schizophrenic patients are discussed.

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1. Introduction

Using the semantic priming paradigm, a technique adopted from cognitive psychology, Maher et al. (1987) and Spitzer et al. (1993a, 1994) have provided empirical evidence for the hypothesis that enhanced spreading of activation underlies positive

formal thought disorder (associative loosening/tangential speech) in schizophrenia. For semantic priming experiments, trials each consisting of two strings of characters/words (referred to as prime and probe) are successively presented. The subject is usually instructed to silently attend to the first string of characters/word and then either (a) has to read aloud the probe [word pronunciation (WP) procedure] or (b) has to decide whether the probe is a word or a string of meaningless characters [lexical decision (LD) procedure]. Instructional emphasis is put on response speed.

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A large literature from cognitive psychology shows that responses are reliably facilitated to a probe that is preceded by a semantically related prime word (e.g. sister–brother) relative to probes which are preceded by unrelated (e.g. hat–table) or neutral primes (e.g. xxx–table). This so-called semantic priming effect is attributed to a spread of activation initiated by the display of the prime word which is thought to pre-activate semantically related information (Neely, 1977, 1991). Whereas stimulus onset asynchronies (SOA; interval between prime and probe onset) below 400/500 ms solely reflect automatic spreading of activation, priming effects for larger SOAs may also be influenced by expectancy and other controlled processes.

Maher et al. (1987), Spitzer et al. (1993a) (for a review, see Spitzer, 1997) and, more recently, Weisbrod et al. (1998) were able to demonstrate that TD schizophrenic patients exhibit increased semantic priming at short SOAs. It is argued that these patients display a greater automatic spreading of activation in the semantic network. Spitzer measured even greater priming effects in TD patients when using indirectly related words (i.e. words which are indirectly mediated through a nonpresented word, tiger–lion–stripes, day–night–black). This suggests that TD schizophrenic patients do not only have faster but also farther-reaching associations than controls.

Despite this set of findings, the literature on semantic priming has remained contradictory, since several studies have not found larger priming in schizophrenia (Barch et al., 1996; Chapin et al., 1989, 1992; Ober et al., 1995; Vinogradov et al., 1992). This failure to find enhanced semantic priming in schizophrenia may be accounted for by several methodological shortcomings in these studies (see also Moritz et al., 1999 for more details).

(a) Several studies have used very short prime presentation times (lower or equal to 100 ms; Barch et al., 1996; Blum and Freides, 1995; Ober et al., 1995; Vinogradov et al., 1992) that may preclude deep semantic processing in schizophrenic patients since most patients are perceptually slowed and have problems perceiving quickly presented material (see Cadenhead et al., 1997).

(b) Some studies did not subdivide patients according to the presence of formal thought disorder (Chapin et al., 1989, 1992; Ober et al., 1995, 1997; Vinogra-

dov et al., 1992). Enhanced priming is not inferred for all syndromic patterns of schizophrenia but only for positive formal thought disorder. Therefore, these studies do not falsify the initial claims made by Maher et al. (1987).

(c) Some studies have used rather long SOAs (≥ 500 ms) which do not exclusively tap automatic processes (e.g. Aloia et al., 1998) and, thus, do not challenge the basic hypothesis.

(d) All studies that failed to replicate Maher's results have measured direct semantic priming effects. However, according to the theoretical account put forward by Spitzer (1997), the assessment of indirect semantic priming is a more valid and powerful correlate of enhanced spreading of activation, since it allows estimation of how fast and how far associations spread in semantic networks (Spitzer, 1997).

On the other hand, methodological concerns have also been raised against the original studies conducted by Maher et al. (1987) (also Manschreck et al., 1988) and Spitzer et al. (1993a,b). Barch et al. (1996) have pointed out that greater than normal priming has only been demonstrated in LD tasks but not in WP tasks. They argue that LD tasks may not solely measure automatic processes. Moreover, it has been suggested that the computation of priming ratios as carried out by Spitzer might not adequately address the impact of psychomotor slowing on priming. Finally, it has been claimed that the effects of antipsychotic medication were not sufficiently addressed.

In three previous studies, we have collected additional evidence in support of the hyper-priming hypothesis while addressing several of the aforementioned objections. In our first study (Moritz et al., 1999), we split a large sample of healthy subjects ($n=156$) according to a measure of language disturbance (Frankfurt Complaint Questionnaire subscale language). We found significantly greater priming in subjects with elevated language disturbance scores. Importantly, we were able to show that priming for short SOAs (200 ms) was not associated with psychomotor slowing. This suggests that motor retardation as evidenced by overall greater RT latencies in schizophrenic patients alone does not imply elevated priming. In a second study (Moritz et al., 2001a), we have replicated Spitzer et al.'s (1993a) results using an identical priming paradigm. TD schizophrenic patients showed greater priming than psychiatric and

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