

Further evidence for “hyper-priming” in thought-disordered schizophrenic patients using repeated masked category priming

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Abstract

Background: Previous research has yielded evidence for enhanced semantic priming in formal thought-disordered schizophrenia patients, a result that fits well with the hypothesis of disinhibited processes of spreading activation in this population.

Methods: The present study tested this hypothesis by using masked repetition priming, which yields reversed semantic priming effects in healthy participants. Assuming that performance in this paradigm relies on a balance between activation and inhibition processes in healthy participants, we compared formal thought-disordered schizophrenia patients, non-thought disordered schizophrenia patients, and healthy controls.

Results: For thought-disordered schizophrenia patients, we found a large positive semantic priming effect for dominant category exemplars (primed by the category name), whereas healthy controls had a small negative effect. For non-thought disordered patients, we found a non-significant, numerically positive effect.

Conclusions: This result yields further evidence for the lack of inhibitory processes in thought-disordered patients.

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1. Introduction

It is a recurrent theme in schizophrenia research to explore access to semantic memory using the semantic priming paradigm (see [Minzenberg et al., 2002](#)). The backdrop of this research is given by hypotheses about the

potential pathological pathways of schizophrenia. One hypothesis suggests there is disinhibited spreading of activation in the semantic network of (formal thought-disordered) schizophrenia patients ([Manschreck et al., 1988](#); [Moritz et al., 2001a,b, 2002](#); [Quelen et al., 2005](#); [Spitzer et al., 1993, 1994](#)). The present study wants to add to this debate by using a new semantic priming technique that might shed some light on the specific characteristics of activation and inhibition processes in the semantic network.

1.1. Semantic priming and schizophrenia

The semantic priming paradigm is well-suited to explore semantic access (see [McNamara, 2005](#)). In its

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standard form, participants have to respond to a target stimulus either by naming it or by categorizing it as a word or non-word. The target is preceded by a prime word that is either semantically related to the target or not. Typically, response times are shorter in the former case (e.g., sister–brother) compared to the latter (e.g., wall–brother). A typical explanation of semantic priming effects found with short prime–target stimulus-onset asynchronies (SOA) is to assume processes of encoding facilitation for the target caused by the related prime. Then, the “classical” theory of encoding facilitation refers to spreading activation within a semantic network (e.g., Collins and Loftus, 1975). Of course, this process must be limited to support processes of, for example, focused thinking or language production in a functional way. Maher and Manschreck (Maher et al., 1987; Manschreck et al., 1988) were the first to test the hypothesis that unlimited processes of spreading activation serve as a major contribution to formal thought disorder in schizophrenia, its behavioural signature being enhanced semantic priming in schizophrenic patients.

Manschreck et al. (1988), Spitzer and colleagues (1993, 1994) and several subsequent studies found significantly greater semantic priming effects for formal thought-disordered schizophrenia patients compared to non-formal-thought-disordered patients or healthy controls (see also Moritz et al., 2001a,b, 2002; Quelen et al., 2005). There were, however, other attempts that failed to show this result (e.g., Barch et al., 1996), see Moritz et al. (2001b) for a discussion. Such failures remind of the fact that the interpretation of an increase in priming has to be made with caution. Individual differences in semantic priming effects are typically obtained only within the positive value range: non-thought disordered patients and healthy controls exhibited clear positive priming effects in the studies by Moritz and colleagues (2001a,b), as well. As long as we do not know the scaling qualities of the measured effects (that is, whether the magnitude of a latent process of spreading activation is linearly captured by a RT difference), an *advocatus diaboli* might remain skeptical about the meaning of a “more” or “less” of semantic priming. One is better off if *qualitative* differences between groups can be observed.

Wentura and Frings (2005) and Frings et al. (2008) recently introduced a priming technique with masked stimuli showing such *qualitative* differences in semantic priming. They used category exemplars as targets (e.g., *apple*) and corresponding category names (e.g., *FRUIT*) as primes, whereby primes were presented for only one refresh cycle of the screen (approximately 14 ms) before

they were replaced by a mask, again for one cycle. This sequence was repeated for 10 cycles so that the prime was presented for 143 ms overall. The main results was a *negative* semantic priming effect, that is, response times were *longer* for targets related to the prime. The authors found this effect to be restricted to low dominant exemplars. Frings et al. (2008) replicated the negatively signed effect, however, without the moderation by dominance. In addition, in most experiments this pattern was restricted to those participants whose performance was at chance level in a subsequent direct test of prime perception, that is, if masked primes were detected above chance level or not. Tentatively, the authors interpreted their results in terms of a center-surround inhibition mechanism (see Carr and Dagenbach, 1990). According to this account, a weakly activated node (e.g., by a masked prime) is surrounded by a ‘ring of inhibition’. Hence, presenting category exemplars from the prime category (which possibly lay in this surround) as the target should result in RT costs. Such an inhibitory mechanism might act to counterbalance mechanisms of encoding facilitation.

Here, we use this technique because it might be sufficiently sensitive to assess individual differences in the balance between two processes – spreading activation and inhibition – that possibly point into different directions. The hypothesis of a disinhibited spreading of activation in thought-disordered schizophrenia patients might be reframed as the hypothesis that there is a shortfall in the counterbalance of spreading of activation and center-surround inhibition. The investigation may also promise to shed light on the divergent results obtained in prior studies. Thus, we want to explore whether thought-disordered patients fail to show a negatively signed semantic priming effect and even show a positive effect as a result of an unrestricted spreading of activation that overruns center-surround inhibition.

The technique by Wentura and Frings (2005) offers additional advantages. It is generally acknowledged that mainly automatic processes are affected in thought-disordered patients as compared to healthy controls (Maher et al., 1987; Moritz et al., 2001a). Though it is a general claim that semantic priming effects found with short SOAs (interval between prime and target onset) reflect automatic processes, this assumption must always be challenged as long as participants can easily grasp the structure of the prime/target relationships. Therefore, it is worthwhile to mask primes to prevent participants from detecting prime/target relationships (see also, e.g., Kiefer and Spitzer, 2000). However, masked presentation is usually associated with brief

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