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Authors

Minzenberg, Michael J

Ober, Beth A

Vinogradov, Sophia

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CRITICAL REVIEW

Semantic priming in schizophrenia: A review and synthesis

MICHAEL J. MINZENBERG,¹ BETH A. OBER,² AND SOPHIA VINOGRADOV¹

¹Department of Psychiatry, University of California, San Francisco and Department of Veterans Affairs Medical Center, San Francisco, California

²Department of Human and Community Development, University of California, Davis and Department of Veterans Affairs Northern California Health Care System, Martinez, California

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Abstract

In this paper, we present a review of semantic priming experiments in schizophrenia. Semantic priming paradigms show utility in assessing the role of deficits in semantic memory network access in the pathology of schizophrenia. The studies are placed in the context of current models of information processing. In this review we include all English-language reports (from peer-reviewed journals) of single-word semantic priming studies involving participants with schizophrenia. The studies to date show schizophrenic patients to exhibit variable semantic priming effects under automatic processing conditions, and consistent impairments under controlled/attentional conditions. We also describe associations with other neurocognitive dysfunction, neurochemical and electrophysiological disturbances, and clinical manifestations (such as thought disorder). (*JINS*, 2002, *8*, 699–720.)

Keywords: Semantic priming, Schizophrenia, Semantic memory, Language, Information processing

INTRODUCTION

Schizophrenia is primarily a disorder of thinking and language. Indeed, investigators have suggested that a defect in language information processing may be pathognomonic of the disorder (Clare et al., 1993; Crow, 1997; Saykin et al., 1994; Thomas et al., 1993). And yet, despite the current widespread interest in neurocognitive aspects of the illness, schizophrenia researchers have only recently begun to explore one of the most widely studied phenomena in cognitive psychology: priming effects in the semantic memory system.

We will review the literature on semantic priming experiments in schizophrenia, discuss the discrepant findings, address the important methodological issues in this research, and place this line of investigation in the broader context of the rapidly evolving field of neurocognition of schizophrenia.

Semantic Memory and Spreading Activation Network Models

All information processing models posit the existence of a long-term memory system. Within long-term memory, most theorists distinguish between episodic memory, which is the personal and contextually-bound memory for events/episodes, and semantic or generic memory, which is seen as all accumulated general (i.e., world) knowledge (Tulving, 1972, 1986). The semantic memory system is hypothesized to be an associative network of permanently stored general knowledge which is not tied to specific events and which builds up over an individual's lifetime. The semantic memory network is often viewed as a network of conceptual "nodes" connected by relational "links," with spread of activation occurring between connected conceptual nodes (Figure 1). Whenever a specific node is activated in memory, the surrounding nodes will be activated to a degree related to their proximity to the initially activated node (Anderson, 1983; Collins & Loftus, 1975; Collins & Quillian, 1969).

In addition to spread of activation, which is assumed to be an automatic process, controlled processes, such as those

Reprint requests to: Michael J. Minzenberg, M.D., Department Psychiatry, 116C, DVA Medical Center, 4150 Clement Street, San Francisco, CA 94121. E-mail: mjminz@itsa.ucsf.edu

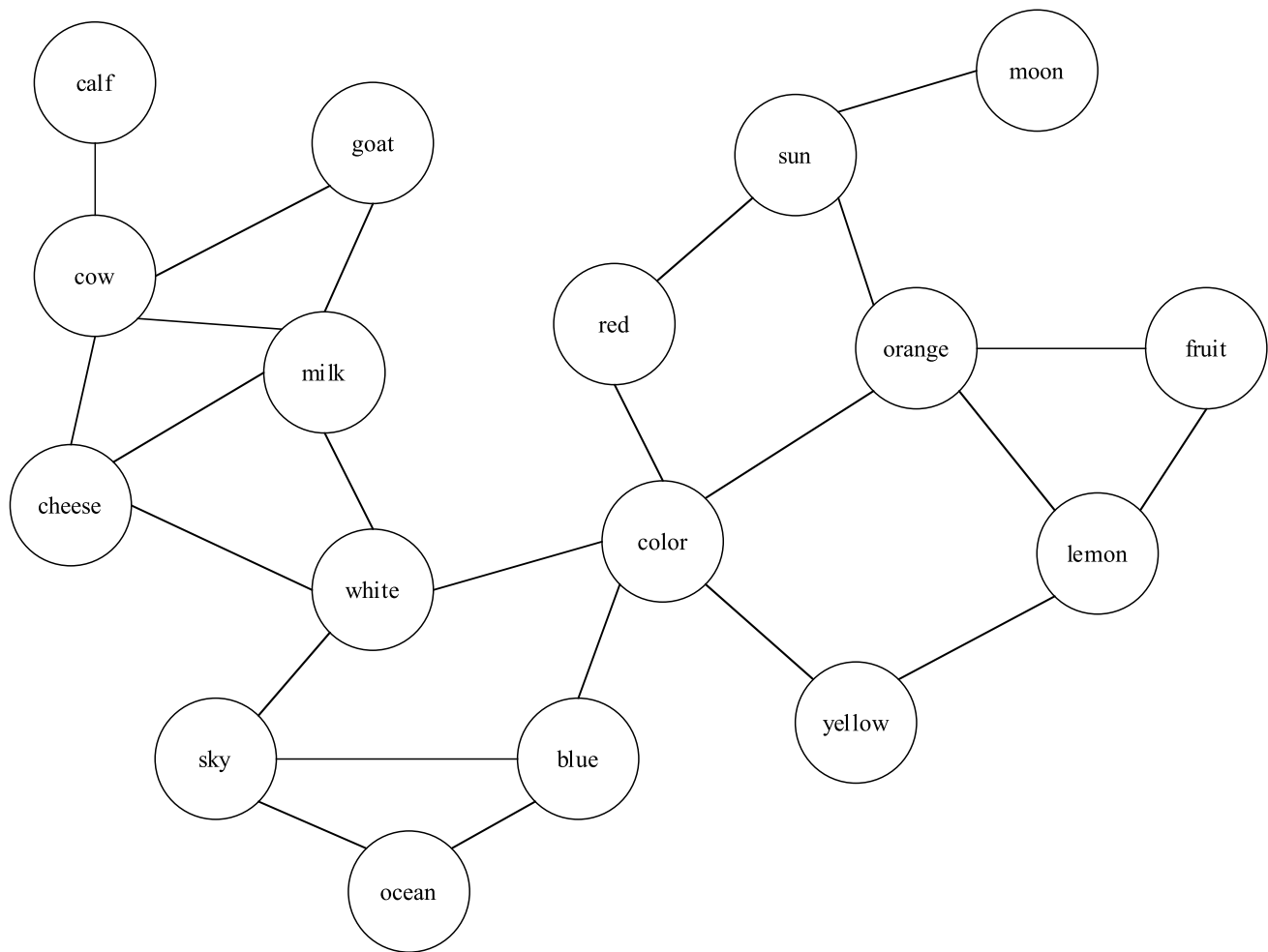


Fig. 1. Schematic semantic memory network.

related to attention and strategy, can influence the processing of information in semantic memory. Useful models for understanding some aspects of cognitive dysfunction have been generated by postulating changes in the structural and/or functional characteristics of such semantic memory networks (e.g., Cohen & Servan-Schreiber, 1992, 1993; Ober & Shenaut, 1988; Shenaut & Ober, 1996). Cognitive scientists occasionally emphasize the analogy between the structure and function of spreading activation networks and the function of neuronal arrays in the central nervous system (e.g., Gluck & Thompson, 1987; Kriekhaus et al., 1992; Ritter & Kohonen, 1989; Servan-Schreiber et al., 1990; Spitzer, 1995).

Semantic Priming Experiments: An Overview

Semantic priming describes the finding that the reaction time (RT) with which a target word is pronounced, or with which a string of letters is recognized as a word (referred to as a lexical decision task or LDT), can be decreased by presenting to the participant a semantically related word or

prime prior to the appearance of the target word (Meyer & Schvaneveldt, 1971; for reviews, see Neely 1991; Tulving & Schacter, 1990). For example, in a semantic priming experiment, the target word *orange* is pronounced faster when it is preceded by the related prime *lemon* than when it is preceded by an unrelated prime such as *chair*. Semantic priming effects are the RT differences between targets preceded by related *versus* unrelated primes (see Figure 2). The stimulus onset asynchrony (SOA) is the time between the appearance of the prime and the appearance of the target.

Semantic priming effects are currently believed to be the result of three independent processes (see Figure 3; Neely, 1991). The first of these is the automatic process of spreading activation as described above. Spreading activation occurs in all semantic priming paradigms, regardless of the relatedness proportion (the proportion of the total real word prime–target pairs that are semantically-related), the instructional set given to the participant, etc. This process is generally considered to generate solely facilitatory effects in semantic priming paradigms; inhibitory effects (observed as a slowed RT to targets preceded by unrelated primes in comparison to those preceded by neutral primes) are not

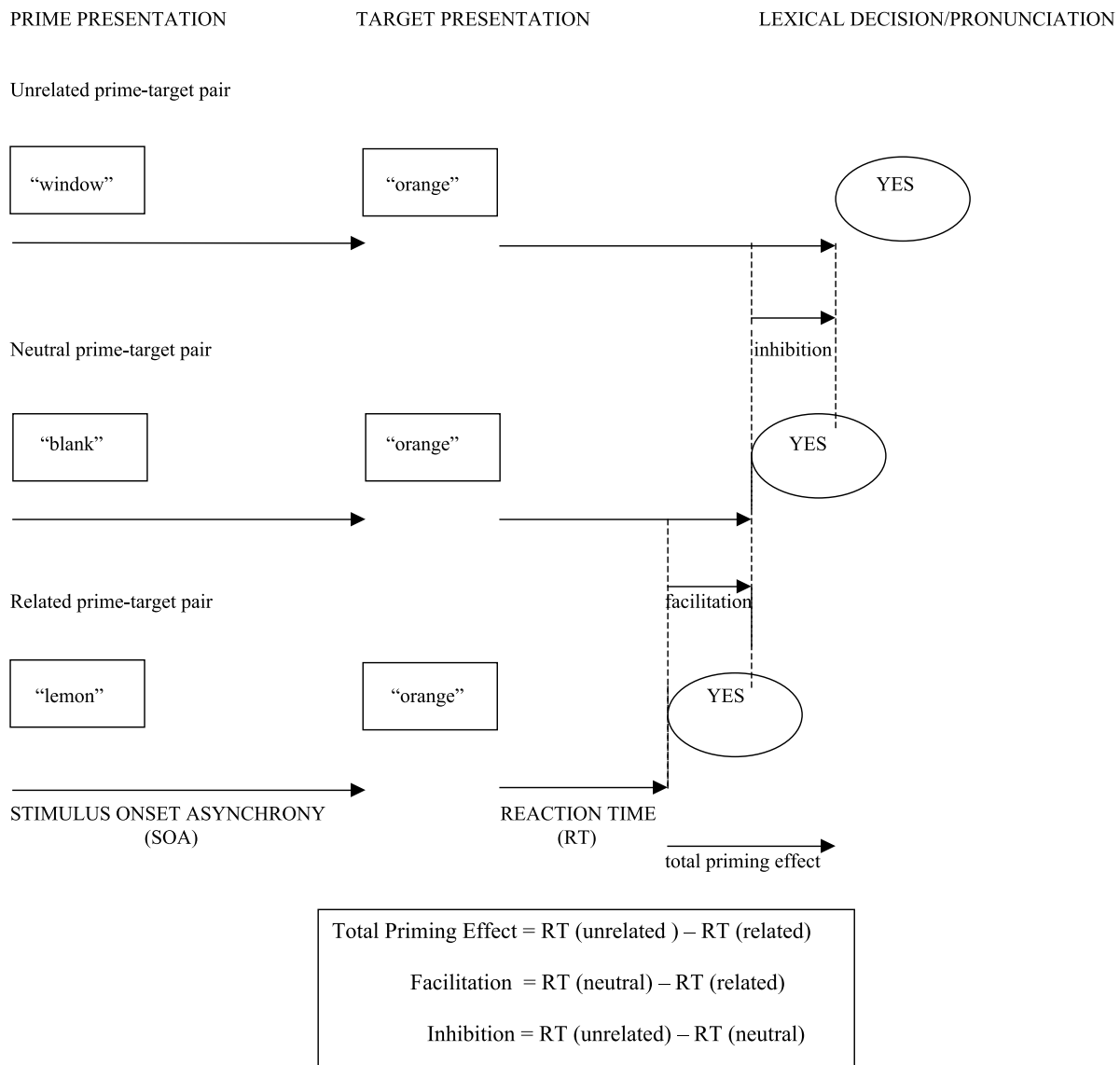


Fig. 2. Semantic priming experimental paradigm.

demonstrable under automatic processing conditions in general (Lorch et al., 1986; Neely, 1977, 1991). Some investigators suggest that "indirect" semantic priming (such as that from the word pair *lemon-sour*, which is mediated by the word *sweet*) is a particularly sensitive measure of spreading activation among schizophrenic participants (Spitzer, 1997). The other two processes are considered to be controlled or strategic processes, although it is important to note that they may occur without complete awareness. They may each generate either facilitatory or inhibitory effects, depending on how the experimental parameters are manipulated (Neely, 1977, 1991; Neely et al., 1989) (see Figure 3 for a schematic overview of automatic *versus* controlled semantic priming processes).

In the controlled process of expectancy, the participant uses the prime to generate an expectancy set of potential targets related to the prime; targets which are in this expect-

tancy set may then be processed more quickly than targets which are not (Becker, 1980). Expectancy is a pre-lexical mechanism, and it can facilitate or inhibit access of the target depending on what the participant expects. Expectancy effects are observed in both pronunciation and lexical decision paradigms, occur at SOAs of 400–500 ms or longer (deGroot et al., 1986; den Heyer et al., 1985; Neely et al., 1989), are influenced by the relatedness proportion (deGroot, 1984), and can be influenced by instructions given to the participant.

The second controlled priming process, semantic matching, is, unlike expectancy, post-lexical and occurs only in lexical decision (not pronunciation) tasks (Neely et al., 1989). In semantic matching, between the time that lexical access of the target has occurred and a word/nonword decision is made for the target, participants can use information about the relatedness of the prime-target pair to decrease the RT

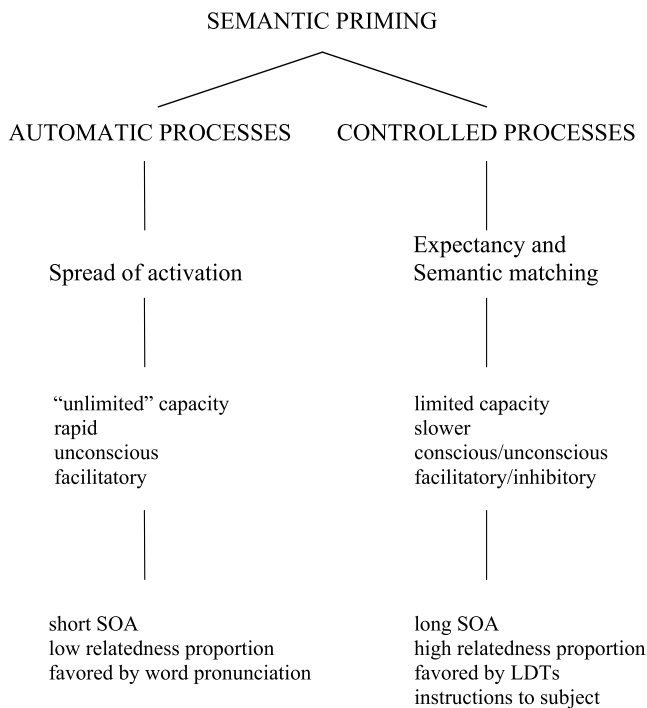


Fig. 3. Summary of information processing in semantic priming.

for a correct “word” decision as well as to decrease the RT for a correct “nonword” decision. If the participant mentally looks back to a previously presented prime or checks a prime which is being simultaneously presented with a target and notices that there is a semantic relationship between the prime and target, then the participant will be biased to a “word” response. However, if the participant looks back to the prime and notices the lack of a semantic relationship between the prime and target, then the participant will be biased towards a nonword response. Any conceptual nodes which are activated by seeing a nonword target will rarely be semantically related to the prime word with which it is paired. Thus, the bias toward a nonword response can occur for unrelated, word–target trials or nonword–target trials. If the nonword ratio, which is the proportion of all unrelated prime–target pairs in which the target is a nonword, is high, then participants will be biased toward a nonword response (deGroot, 1984; Neely et al., 1989). Increases in relatedness proportion, with a concomitant increase in the nonword ratio, can magnify priming effects obtained through postlexical semantic matching.

In sum, the study of semantic priming effects allows researchers to examine structural and processing characteristics of the semantic memory system; moreover, both automatic and controlled processes can be studied. Automatic processes are generally rapid in the time course of action, without capacity limitations, and always unconscious. In contrast, controlled processes are slower, capacity-limited, and can be either unconscious or conscious (Posner, 1986; Schiffrin & Schneider, 1984; Schneider & Shiffrin, 1977). Kiefer and Spitzer (2000) have reported on prelim-

inary attempts to experimentally dissociate “conscious” from “unconscious” components of semantic priming effects, using masked primes. Controlled processing includes such phenomena as attention and strategy implementation. Barch et al. (1996) have noted that it is useful to delineate intralexical from extralexical influences on semantic priming. Intralexical processes (which are referred to as prelexical processes in the psycholinguistics literature) can be automatic or attentional, and include both automatic spread of activation and expectancy. Extralexical processes (which include both prelexical and postlexical processes per the psycholinguistics literature) are always attentional, and include semantic matching, sentence context effects, and the use of instructions or distractors. The parameters of a given semantic priming experiment, such as the type of task (word pronunciation vs. LDTs), the relatedness proportion, the nonword ratio, the type and degree of associative links between primes and targets, the SOAs, and the instructional set given to the participant, will all influence specific aspects of cognitive processing in the semantic memory system, including both automatic and attentional mechanisms, and will emphasize different aspects of intralexical and extralexical processes.

Spreading Activation Network Models of Cognition in the Study of Clinical Disorders

The semantic memory system is believed to be a permanent associative network which is accessed continually during all types of cognitive operations, including perception, learning, language production and comprehension, and problem solving (Tulving & Schacter, 1990). Semantic priming effects simply provide one means of empirically testing hypotheses based on a spreading activation network model of processing by this memory system. These types of models have been successfully used to (1) simulate the operation of simple neural systems in the brain in a manner relevant to normal and abnormal cognition (e.g., Gluck & Thompson, 1987; Posner, 1986; Servan-Schreiber et al., 1990); (2) provide a theoretical approach to understanding the role of dopamine in the cognitive deficits of schizophrenia (Cohen & Servan-Schreiber, 1993; Spitzer, 1995); (3) demonstrate the effects of catecholamine release on gain and signal-to-noise ratio in a network of neural-like elements (Servan-Schreiber et al., 1990); (4) explain differences in contextual disambiguation between elderly participants and participants with Alzheimer’s dementia (Balota & Duchek, 1991); and (5) explain the patterns of normal *versus* “hyperpriming” of semantic associations in Alzheimer’s disease (Shehaut & Ober, 1996).

Cognitive psychologists and psycholinguists have made great strides during the past quarter-century in understanding many characteristics of the semantic memory network in the normal population, but studies of semantic priming in clinical populations, particularly in patients with Alzheimer’s dementia, the normal elderly, and certain neurologically impaired groups are much less abundant, having begun

in the mid-1980s (see, for example, Balota et al., 1992; Nebes, 1989; Posner, 1986; Shenaut & Ober, 1996). Despite some intriguing findings (Aloia et al., 1998; Barch et al., 1996; Chapin et al., 1989, 1992; Goldberg et al., 1998; Henik et al., 1992; Kwapil et al., 1990; Manschreck et al., 1988; Ober et al., 1995, 1997; Spitzer et al., 1993a, 1993b, 1994; Vinogradov et al., 1992; Volz et al., 1994), the role of semantic memory abnormalities in the numerous cognitive abnormalities observed in schizophrenia, particularly in language processing, is still unclear. And yet, the consequences of semantic memory processing dysfunction are far reaching indeed: perception, episodic memory, language comprehension and production, reality monitoring, and problem-solving skills can all be seriously affected by abnormal processes in the semantic memory system. In addition, recent experimental and neuropsychological work suggests that there are specific and perhaps pathognomonic deficits in language-related information processing in schizophrenia, separate from attentional impairment (Clare et al., 1993; Saykin et al., 1994; Thomas et al., 1993; Tracy, 1998). Any viable model of cognition in schizophrenia must therefore take into account changes in semantic memory structure and/or function.

SEMANTIC PRIMING EFFECTS IN SCHIZOPHRENIA IN THE CONTEXT OF THE TWO-COMPONENT MODEL OF INFORMATION PROCESSING

Most of the neurocognitive differences demonstrated between groups of schizophrenia participants and other participant groups have been interpreted in the context of a general type of information processing model, in which a strong distinction is made between automatic and controlled processes. This model has played a major role in the study of semantic priming, as per the preceding section (see Callaway & Naghdi, 1982; Carr et al., 1979; Dawson & Nuechterlein, 1984; Schiffrin & Schneider, 1984; Schneider & Schiffrin, 1977 for general discussions of automatic *versus* controlled cognitive processes). We will employ this model in examining the results of semantic priming studies published to date.

Evidence for Enhanced Automatic Information Processing in the Semantic Priming Paradigm

Experimental designs that favor automatic, pre-attentional components of lexical access, which are typically employed to address the process of spreading activation, have yielded conflicting results (see Table 1).

Spitzer's laboratory reported enhanced semantic priming in automatic conditions in schizophrenia compared to normal control participants. In one study, 32 schizophrenia inpatients and 32 normal controls were each assessed for both "direct" semantic priming and "indirect" semantic priming

with an LDT. Indirect (or "mediated") priming is observed with word pairs such as "chalk" and "black," both of which have a direct association with the word "white." In this study of both direct and indirect semantic priming, each was assessed at an interstimulus interval (ISI) of zero and 500 ms (corresponding in this study to SOAs of zero or 700 ms; Spitzer et al., 1993b), and a 33% relatedness proportion. In comparison to the control group, the schizophrenia group showed non-significant increases in both direct and indirect semantic priming, for both SOAs (one favoring automatic and one favoring controlled processes). This included a trend toward a larger indirect semantic priming effect for the schizophrenia group at an SOA of zero; within the schizophrenia group the priming effect was significant, whereas it was not for the control group. These results were interpreted as representing either an increase in activation or a decrease in inhibition of spreading activation among schizophrenia participants. In a later study from the Spitzer laboratory (Weisbrod et al., 1998), primarily addressing semantic priming relationships with cerebral lateralization and thought disorder (TD), an increased semantic priming was obtained for the TD schizophrenia participants (those with a score over 3 on the Brief Psychiatric Rating Scale Item #4, Conceptual Disorganization; BPRS, Overall & Gorham, 1962). These participants showed an increase in both direct and indirect semantic priming in an automatic condition LDT, in comparison to both normal controls and non-TD schizophrenia participants (the latter group showed no difference from controls).

Another study of 70 schizophrenia inpatients showed a semantic priming effect which appeared greater than that for a group of 44 normal controls in an LDT at each of three SOAs: 200 ms, 400 ms, and 700 ms, and a 39% relatedness proportion; this semantic priming effect did not vary significantly within the schizophrenia group across the three SOAs (Spitzer et al., 1994). However, the schizophrenia and control groups were not directly compared statistically; indeed, the control group data had been previously collected and apparently was not published (see Spitzer et al., 1994, for reference). Spitzer et al. published a separate study of TD ($N = 29$) *versus* non-TD ($N = 21$) schizophrenia inpatients (with TD defined as BPRS item #4 score >2), again using an LDT with SOAs of 200 and 700 ms but with a 67% relatedness proportion, and also including a concurrent group of 50 normal controls (Spitzer et al., 1993a). In this study, the TD group showed significantly increased direct semantic priming compared to controls at both SOAs, as well as a significant increase in indirect semantic priming at the short SOA (where controls showed no significant within-group indirect semantic priming effect) and a trend toward greater-than-normal indirect priming at the long SOA. When the semantic priming effects were expressed as the percentage change in RT rather than absolute difference in RT, the group differences were abolished for direct semantic priming though preserved for indirect semantic priming. The difference between TD schizophrenia and control participants for the indirect semantic priming at the short SOA

Table 1. Summary of semantic priming studies comparing schizophrenic subjects with controls

	Scz sample	Paradigm: SOA; RP	Results for scz group(s)	Comment
Enhanced spread of activation				
Spitzer et al., 1993b	32 inpatients	LDT: 0/700; 67%	n.s. ↑ direct/indirect SP both SOAs	n.s. indirect SP for controls at 0 ms
Weisbrod et al., 1998	40 inpatients	LDT: 250; 67%	↑ direct (n.s.)/indirect (sig.) SP	TD (BPRS#4>3) >nonTD
Spitzer et al., 1994	70 inpatients	LDT: 200/400/700; 39%	↑ SP	historical controls; TD (BPRS#4>4) >nonTD
Spitzer et al., 1993a	50 inpatients	LDT: 200/700; 67%	sig. ↑ direct/indirect SP	also ↑ %indirect SP; TD>nonTD
Manschreck et al., 1988	12 outpatients	LDT: 250; 50%	TD>nonTD/controls	TD faster RTs overall vs. controls
Henik et al., 1995	16 “chronic”	LDT: 240/1840; 50%	↑ SP	data pooled across SOAs
Kwapil et al., 1990	21 outpatients	WP: 500; 33%	↑ accuracy	used degraded targets
Normal spread of activation				
Chapin et al., 1989	12 inpatients	LDT: 0; 50%	SP sig within each group	SP not compared between groups
Chapin et al., 1992	45 inpatients	LDT: 0; 50%	SP = control group	
Barch et al., 1996	100 inpatients	WP: 200 to 950; 50%	↓ SP only at 950 ms TD/nonTD	TD=BPRS#4>2; meds assoc. ↑ RT
Blum and Freides, 1995	20 outpatients	LDT: 350; 33%	TD/nonTD SP = control	TD=19.70 on SATLC
Passerieux et al., 1995	17 outpatients	LDT: 64/240; 33%	no inhib to unrelated prime 240 ms	scz/control both n.s. SP at 64 ms
Vinogradov et al., 1992	19 outpatients	WP/LDT: 250/17%	↓ SP in LDT only	6/19 scz med-free 7 d.
Ober et al., 1995	19 outpatients	WP/LDT: 250/17%	↓ SP for horiz. pair LDT only	vertical vs. horiz. category pairs.
Impaired spread of activation				
Henik et al., 1992	22 “chronic”	LDT: 240/1840; 50%	fewer scz with SP effect at 240 ms	scz/control sig SP when identical pairs
Ober et al., 1997	31 outpatients	LDT: 260/1000; 15%/46%	n.s. SP paranoid-category/noncat., nonparanoid-noncat.	all scz med-free 7 d.
Impaired controlled processing				
Aloia et al., 1998	20 inpatients	WP: >350; 63%	↓ SP for TD vs. nonTD/control for highly-related pairs	n.s. SP for TD; no fixed SOA
Henik et al., 1995	16 “chronic”	LDT: 100/1840; 50%	↓ SP on distraction both SOAs	
Passerieux et al., 1997	22 outpatients	LDT: 500; 50%	n.s. SP effect for TD	TD=SATLC>6
Besche et al., 1997	34 inpatients	LDT: 500; 50%	n.s. SP effect for TD	TD=SATLC>7; syntactic priming intact
Barch et al., 1996	100 inpatients	LDT: 950; 50%	↓ SP for TD/nonTD vs. control	TD=BPRS#4>2; meds assoc. ↑ RT
Vinogradov et al., 1992	19 outpatients	WP/LDT: 250/17%	↓ SP in LDT only	6/19 scz med-free 7 d.
Ober et al., 1995	19 outpatients	WP/LDT: 250/17%	↓ SP horiz. pair LDT only	vertical vs. horiz. category pairs

Note. LDT: lexical decision task; WP: word pronunciation task; SOA: stimulus onset asynchrony; RP: relatedness proportion; RT: reaction time; SP: semantic priming; TD: thought disorder; BPRS: Brief Psychiatric Rating Scale; scz: schizophrenia group; SATLC: Scale for the Assessment of Thought, Language and Communication; horiz.: horizontal; n.s.: not statistically significant.

(seen as a particularly sensitive measure of spreading activation) was offered in support of the hypothesis that TD participants differ from normals primarily in exhibiting enhanced “spread of associations.”

Other groups have observed increased semantic priming effects in schizophrenia groups. Manschreck et al. (1988) assessed 12 TD schizophrenia participants, 6 non-TD schizophrenia participants, 9 unipolar affective participants, and 11 non-psychiatric controls on an LDT with an SOA of 250 ms and 50% related prime–target pairs. All participants were outpatients, and TD was defined as a score of 3 or more on any one of five “features” of the Schedule for Affective Disorders and Schizophrenia (SADS; Spitzer & Endicott, 1977). The TD schizophrenia group showed not only greater semantic priming in comparison to the other three groups but also faster mean RTs for both related and unrelated prime–target pairs; this was not accounted for by an accuracy decrement (i.e., a speed–accuracy tradeoff). The enhanced semantic priming among the TD schizophrenia group was interpreted in the context of spreading activation. No explanation was offered for the finding of faster overall RTs, which has not been observed in any other semantic priming study involving group comparisons of schizophrenia and control participants.

Henik et al. (1995), in a complex LDT designed primarily to evaluate the effect of information processing load on semantic priming in schizophrenia (as described below in the section devoted to controlled information processing), also attempted to address spreading activation in schizophrenia participants. Sixteen schizophrenia participants (all said to be TD by undefined criteria from the SADS) and 16 normal controls performed an LDT with a 50% relatedness proportion. The SOAs were indicated as 100 ms and 1700 ms in the procedure section, but as 240 ms and 1840 ms elsewhere in the report. In this paradigm, where some conditions included distractors which were to be attended to or ignored, the non-distractor conditions revealed significantly larger semantic priming effects for the schizophrenia group in comparison to controls. Semantic priming effects for each participant appear to represent pooled data from both SOA conditions, confounding the authors’ suggestion that the hyperpriming observed may be related to uncontrolled or uninhibited automatic spreading activation.

Evidence for Normal Automatic Information Processing in the Semantic Priming Paradigm

Several groups of investigators have reported results suggesting that spreading activation in semantic memory (or more generally, automatic semantic priming processes) in schizophrenia is normal (Table 1). Chapin et al. (1989) assessed semantic priming in 12 schizophrenia inpatients, 12 inpatients with psychotic depression, and 12 normal controls using the LDT with an SOA of 0 ms and a 50% relatedness proportion. The semantic priming effect was

significant within each group; however, this effect was not compared directly between groups. A later study by this group (Chapin et al., 1992) assessed 45 schizophrenia inpatients (grouped by clinical subtype) and 15 normal controls in a paradigm apparently identical to the earlier study. The semantic priming effect was significant within each clinical group as well as the control group, but not different across groups. Barch et al. (1996) examined semantic priming in 75 medicated and 25 unmedicated schizophrenia patients as well as in 10 depressed and 28 non-psychiatric controls, using the word pronunciation task across SOAs from 200 to 950 ms, and a 50% relatedness proportion. The total schizophrenia participant pool of 100 was also divided into TD (those with BPRS Item #4 >2) and non-TD groups for separate analyses. To account for the relationship between longer RTs and spuriously increased semantic priming effects, regression equations were calculated from the semantic priming data of the normal control group. In this manner, semantic priming effects were expressed as the difference between observed and predicted semantic priming, for each participant (Chapman et al., 1994). Comparisons of group means showed that both TD and non-TD schizophrenia groups differed from either depressed or normal controls only by exhibiting a smaller semantic priming effect at the 950-ms SOA. The authors concluded that “automatic, intralexical semantic priming” is intact in schizophrenia, but that “deficits in higher level, extralexical processes that influence semantic priming” (i.e., controlled processes) may exist (findings regarding medication effects and clinical variables are discussed later).

Blum and Freides (1995) administered a lateralized version of the LDT with an SOA of 350 ms and 33% relatedness proportion, to 10 TD schizophrenia participants, 10 non-TD schizophrenia participants, and 11 normal controls. All schizophrenia participants were right-handed male outpatients on medication; TD was assessed with the Scale for the Assessment of Thought, Language and Communication (TLC; Andreasen, 1979). The TD group had a mean score of 19.70, whereas the non-TD group had a mean score of 1.30 on the TLC; it’s not clear if *a priori* criteria were established for inclusion in these groups. In this study, neither RTs nor semantic priming effects were significantly different between TD schizophrenia, non-TD schizophrenia and control groups. The authors concluded that automatic semantic memory processes are normal in schizophrenia. Passerieux et al. (1995) employed an LDT with SOAs of 64 or 240 ms, and a 33% relatedness proportion, in a study of 17 schizophrenia participants (which were also grouped by clinical subtype) and 11 normal controls. While the 240-ms SOA is identified as a “controlled” condition by the authors, it should be noted that this SOA is generally considered too short to permit the emergence of true controlled information processing (e.g., deGroot, 1984; den Heyer et al., 1985; Neely, 1977, 1991). Nonsignificant semantic priming was observed for both schizophrenia and control groups at an SOA of 64 ms. At the 240 ms SOA, the control group showed the expected inhibition for unrelated

Table 2. Summary of reaction times and accuracy rates by subject group, for each study

	Schizophrenic group			Control group		
	<i>M RT (ms) ± SD</i>		<i>M accuracy</i>	<i>M RT (ms) ± SD</i>		<i>M accuracy</i>
	Unrelated	Related	Related (%)	Unrelated	Related	Related (%)
Spitzer et al., 1993b						
200 ms SOA, direct SP	1108.4 ± 408.9	993.3 ± 402.5	97.6 ± 4.9	697.4 ± 163.1	642.2 ± 147.5	98.4 ± 3.2
200 ms SOA, indirect SP	1108.4 ± 408.9	1050.5 ± 391.3	98.4 ± 4.1	697.4 ± 163.1	684.6 ± 155.7	97.0 ± 4.7
Weisbrod et al., 1998						
TD: Left hemisphere, direct SP	1188 ± 980	876 ± 441	n.a.	621 ± 156	573 ± 159	n.a.
TD: Left hemisphere, indirect SP	1188 ± 980	1076 ± 838	n.a.	621 ± 156	629 ± 169	n.a.
TD: Right hemisphere, direct SP	1203 ± 823	958 ± 514	n.a.	662 ± 193	591 ± 215	n.a.
TD: Right hemisphere, indirect SP	1203 ± 823	996 ± 499	n.a.	662 ± 193	633 ± 193	n.a.
Spitzer et al., 1994						
200 ms SOA	1095 ± 417	993 ± 318	(95.8 ± n.a.)	704 ± 133	641 ± 123	(96.2 ± n.a.)
Spitzer et al., 1993a						
TD: 200 ms SOA, indirect SP	1099 ± 340	1007 ± 312	(96.4 ± n.a.)	732 ± 196	716 ± 195	(97.0 ± n.a.)
Manschreck et al., 1988						
TD: 250 ms SOA	561 ± 178	478 ± 176	91 ± n.a.	642 ± 85	605 ± 92	99 ± n.a.
Henik et al., 1995	(pooled data 240/1840 ms SOA; raw RT data not given)					
Chapin et al., 1989	(RT data only in figure; no accuracy data)					
Chapin et al., 1992	(RT data only in figure, log-transformed; accuracy data only in figure)					
Barch et al., 1996						
Medicated: 200 ms SOA	750.9 ± 164.4	730.7 ± 150.6	>99 ± n.a.	546.0 ± 72.9	532.2 ± 62.8	>99 ± n.a.
Medicated: 300 ms SOA	713.9 ± 151.8	693.2 ± 146.0	>99 ± n.a.	508.1 ± 63.8	503.1 ± 65.7	>99 ± n.a.
Blum and Freides, 1995	(RT given for each of 7 visual fields; no accuracy data)					
Passerieux et al., 1995						
“Hebephrenic:” 240 ms SOA	978.8 ± n.a.	953.7 ± n.a.	91 ± n.a.	809.8 ± n.a.	788.4 ± n.a.	94 ± n.a.
Vinogradov et al., 1992						
LDT	657 ± 63	653 ± n.a.	(>99 ± n.a.)	595 ± 94	580 ± n.a.	(>99 ± n.a.)
Pronunciation	598 ± 47	580 ± n.a.	(>99 ± n.a.)	528 ± 62	515 ± n.a.	(>99 ± n.a.)

Ober et al., 1995							
LDT, "down"	711 ± 105	680 ± 94	n.a.	644 ± 113	617 ± 105	n.a.	
LDT, "up"	686 ± 81	678 ± 83	n.a.	623 ± 100	600 ± 100	n.a.	
Pronunciation, "down"	633 ± 68	608 ± 80	n.a.	561 ± 81	547 ± 79	n.a.	
Pronunciation, "up"	616 ± 79	598 ± 70	n.a.	553 ± 80	533 ± 68	n.a.	
LDT, typ-typ	691 ± 95	687 ± 103	n.a.	624 ± 131	593 ± 117	n.a.	
LDT, atyp-typ	680 ± 103	661 ± 82	n.a.	622 ± 135	607 ± 132	n.a.	
LDT, typ-atyp	698 ± 103	696 ± 99	n.a.	621 ± 112	613 ± 117	n.a.	
LDT, atyp-atyp	702 ± 114	699 ± 126	n.a.	636 ± 145	622 ± 136	n.a.	
Pronunciation, typ-typ	627 ± 74	617 ± 76	n.a.	553 ± 76	537 ± 77	n.a.	
Pronunciation, atyp-typ	614 ± 89	598 ± 76	n.a.	557 ± 81	537 ± 69	n.a.	
Pronunciation, typ-atyp	615 ± 81	608 ± 71	n.a.	564 ± 81	541 ± 73	n.a.	
Pronunciation, atyp-atyp	632 ± 67	620 ± 80	n.a.	567 ± 76	557 ± 79	n.a.	
Henik et al., 1992							
240 ms SOA	1020 ± 337	995 ± 378	93.2 ± n.a.	808 ± 319	766 ± 301	96.4 ± n.a.	
1840 ms SOA	1057 ± 487	926 ± 381	95.0 ± n.a.	829 ± 359	718 ± 239	95.6 ± n.a.	
Ober et al., 1997							
Paranoid: 250 ms SOA, category	618 ± 85	603 ± 96	(98.2 ± n.a.)	479 ± 51	456 ± 44	(99.8 ± n.a.)	
Paranoid: 250 ms SOA, noncat.	597 ± 81	598 ± 97	484 ± 45	469 ± 50			
Paranoid: 1000 ms SOA, cat.	641 ± 128	605 ± 131	497 ± 73	452 ± 58			
Paranoid: 1000 ms SOA, noncat.	628 ± 119	591 ± 103	495 ± 68	461 ± 64			
Aloia et al., 1998							
Mod/sev TD: high-assoc.	(no RTs; % SP only; no accuracy data)						
Mod/sev TD: med-assoc.	(no RTs; % SP only; no accuracy data)						
Passerieux et al., 1997							
TD	1390 ± 360	1398 ± 396	n.a.	1160 ± 128	1087 ± 134	n.a.	
Besche et al., 1997							
TD	777.8 ± 258.2	757.9 ± 243.2	97.9 ± 2.9	604.7 ± 82.2	559.6 ± 80.7	98.8 ± 2.2	
Barch et al., 1996							
Medicated: 950 ms SOA	681.8 ± 143.5	683.3 ± 165.4	n.a.	512.5 ± 66.6	493.2 ± 57.7	n.a.	
Kwapil et al., 1990							
Change in accuracy vs. neutral	(no RT data, only accuracy data)		18.4 ± 5.6 (acc change)	n.a.	n.a.	7.3 ± 12.2	

Note. Subject groups and experimental conditions (identified at left under authors) chosen for inclusion in table on the basis of greatest group differences observed in each study. See Table 1 or text for details of subject group identifiers, and experimental conditions.

Accuracy data given for related word pairs only. Parentheses indicate accuracy data available only for subject group across all conditions.

n.a. data not available.

primes *versus* neutral primes; however, the pooled schizophrenia group did not show this effect.

Our research group has also observed schizophrenia groups to perform similarly to normal control groups in semantic priming paradigms which favor automatic processes such as spreading activation. In Vinogradov et al. (1992), 19 schizophrenia outpatients (including 6 medication-free for 1 week prior to testing) and 20 normal controls were assessed in both LDT and word pronunciation tasks, each with SOAs of 250 ms and a 17% relatedness proportion, with highly associated prime–target pairs. Semantic priming effects were significant within each group for the word pronunciation task with no significant difference between groups, suggesting that spreading activation is intact in the schizophrenia group. In contrast, in the LDT, the control group showed a significant semantic priming effect while the schizophrenia group did not. The difference in performance of the schizophrenia group *versus* controls for the LDT in comparison to the word pronunciation task implies a deficit at a more controlled, postlexical stage of processing (see below). A later study with the same schizophrenia participants (Ober et al., 1995) utilized the same SOA and relatedness proportion, again in both the LDT and the word pronunciation task, but with “vertical” category relationships between primes and targets (e.g., where superordinate–subordinate word relationships such as *bird–robin* were utilized) as well as “horizontal” category relationships between primes and targets (e.g. *nickel–dime*). In addition, both typical and atypical category members were utilized as primes or targets in the “horizontal” priming conditions. The schizophrenia group showed semantic priming effects equal to those of controls, in all conditions except for the “horizontal” prime–target LDT. The horizontal category relationship of prime and target more closely replicates the condition of high-associate word pairings (from the earlier experiment) than does the vertical category relationship. This condition enhances the role of semantic matching in facilitatory effects (Becker, 1980) and would therefore be expected to be more sensitive to impairments in this postlexical process. Therefore the two studies taken together suggest normal automatic priming processes, but deficits in controlled, postlexical processes in schizophrenia.

Evidence for Impaired Automatic Information Processing in the Semantic Priming Paradigm

Several groups have observed smaller semantic priming effects among schizophrenia groups compared to controls, in conditions that favor automatic processing (Table 1). Henik et al. (1992) employed a two-part experimental design in an attempt to dissociate the processes of activation of a word’s representation from the spreading activation among conceptual nodes. In Part 1, a standard LDT was used with an SOA of either 240 or 1840 ms, and a 50% relatedness proportion at each SOA. At the 240 ms SOA, 14/17 normal controls showed a positive semantic priming effect, *versus* 14/22

“chronic” schizophrenia participants (significant by the sign test for the control group only). The schizophrenia group showed decreased semantic priming compared to the controls; however, this comparison was not analyzed statistically. In contrast, at an SOA of 1840 ms the sign test was significant for both groups’ semantic priming effect. In Part 2, the same paradigm was used except for identical prime–target pairs being presented in place of related (but different) prime–target pairs (e.g., *NURSE–nurse* instead of *DOCTOR–nurse*). This condition was employed to isolate “node activation” from spreading activation *per se*, which should theoretically not contribute to a semantic priming effect where prime and target words are identical. Here the semantic priming effect was reported as significant for both groups at each SOA; sign test results were not reported, though it should be noted that mean semantic priming effects at each SOA were slightly higher for the schizophrenia compared to control group (also not compared statistically). The authors concluded that, in schizophrenia, “while lexical access is not different from normal, the spread of activation seems to be slowed down but may be compensated when time allows for it.”

Investigators at NIMH have also observed semantic priming deficits among schizophrenia participant groups under conditions that were hypothesized as favoring automatic processing (Aloia et al., 1998). Their participants performed a word pronunciation task with a 350-ms ISI. The SOA was not fixed in this paradigm because the prime presentation was terminated upon the participant’s pronunciation of the prime, before later pronunciation of the target. The effective SOA would likely be at least 500 ms given typical RTs of either control participants or schizophrenics. In addition, the relatedness proportion was 63%. As a result, this study appears to favor controlled processing; the results are discussed below in the section devoted to controlled processing deficits.

We have also observed differences in semantic priming between schizophrenia and control participants that appear to reflect impairments in automatic processing. In Ober et al. (1997) a group of 31 schizophrenia outpatients (all medication-free for 7 days, and subgrouped into paranoid *vs.* nonparanoid) performed an LDT with either an SOA of 260 ms and 15% relatedness proportion (the automatic priming condition), or an SOA of 1000 ms and 46% relatedness proportion (the controlled priming condition). In this study semantic priming effects were nonsignificant for schizophrenia groups only, in some of the automatic conditions only. These included paranoid schizophrenia participants responding to category-related pairs; paranoid schizophrenia participants responding to noncategory-related pairs; and nonparanoid schizophrenia participants responding to noncategory-related pairs. Semantic priming effects were significant for all groups under controlled conditions. In this set of experiments, we concluded that some schizophrenia participants may experience either a delayed onset or subnormal peak level of spreading activation. In considering overall the data that we have obtained over several stud-

ies with various experimental designs (Ober et al., 1995, 1997; Vinogradov et al., 1992) we have found unreliable semantic priming effects among schizophrenia groups only with LDTs and only when a short SOA is used. This suggests that deficits may exist in both automatic and controlled processes in contributing to the unreliable semantic priming found in our participant populations.

Summary of Studies Addressing Automatic Processing Among Schizophrenia Participants in the Semantic Priming Paradigm

Generalizations regarding the performance of schizophrenia participants on semantic priming tasks under automatic conditions are difficult, given the diversity in participant samples and experimental conditions employed. Evidence for abnormal automatic semantic priming in schizophrenia must be interpreted with caution and with careful consideration of the relationship between RTs and semantic priming effects, as well as clinical factors such as acute symptom recurrence and medication status, all of which may serve to establish the spurious appearance of normal or supranormal performance of schizophrenia participants on a semantic priming task. In addition, the various findings may reflect a lower reliability in estimates of automatic processing (compared to controlled processing) in semantic priming experiments (see section on methodological concerns below). Those concerns aside, the diversity of findings may reflect an uncharacterized heterogeneity in schizophrenia. This may be related to illness severity, presence or absence of TD, underlying impairments in semantic memory access and/or organization (as differentially assessed by varying experimental factors such as word category membership, typicality, etc.), or any number of other factors identifiable at physiological, neuropsychological, or clinical levels of analysis.

Evidence for Impaired Controlled/Attentional Information Processing in the Semantic Priming Paradigm

Consistent with a large body of research evidence demonstrating deficits in attentional mechanisms in schizophrenia, several research groups have addressed the role of impaired controlled/attentional information processing in the semantic priming paradigm as a possible factor in the pathology of the disorder. Henik et al. (1995), in a report described in part above, focused primarily on the impact of an increased cognitive processing load on semantic priming effects. Sixteen schizophrenia participants and 16 normal controls performed an LDT with SOAs favoring either automatic or controlled processes (inconsistently indicated in the report as either 100 and 1700 ms, or as 240 and 1840 ms), and 50% relatedness proportion. In this LDT, the schizophrenia group showed a dramatic attenuation of semantic

priming when a secondary task (detecting a concurrent distracter stimulus, either visual or auditory), was included. In a second experiment, with a secondary task (presentation of a concurrent visual distractor stimulus), the schizophrenia group again showed reduced semantic priming even when participants were instructed to ignore the distractor stimuli. This effect was not replicated in a task requiring the schizophrenia participants to ignore an auditory distractor. These results suggest the role of a limited-capacity, single-channel attentional resource in semantic priming effects, as emphasized by Henik et al. The larger impact of intramodal (*versus* cross-modal) distractors is noted as inconsistent with Callaway and Naghdi's (1982) hypothesis of a general modality alternation effect in information processing. Callaway and Naghdi (1982) have hypothesized that schizophrenic participants may be more easily distracted when the signal to be ignored is presented in a sensory mode differing from the signal to be attended to. In addition, the observation of this secondary task/distractor effect, even at a short SOA (100 ms) suggests to Henik et al. that perhaps even spreading activation may require mental resources. The authors refer to Kahneman (1973; Kahneman & Treisman, 1984) who hypothesizes an automatic allocation of processing resources according to demand (see also Cohen et al., 1990, for a somewhat different discussion of the interaction of controlled and automatic processes). Furthermore, the authors attempted to resolve their apparently conflicting data showing that schizophrenia groups demonstrate hyperpriming in the absence of distractors, yet subnormal priming upon interference. They postulated reduced "control" as a general phenomenon possibly underlying both uninhibited activation as well as increased vulnerability to distraction; this notion of control is not elaborated in their discussion. In any event, it is not entirely clear how to resolve these results with those of an earlier study by this group (Henik et al., 1992), where the long SOA (1840 ms) appeared to eliminate the schizophrenia group's semantic priming impairment seen at the short SOA (100 ms). The authors employ the somewhat vague notion of "compensation," which may refer to controlled processes other than those that are assessed by the paradigm employed in the 1995 study.

A French research group, in two LDT studies with similar conditions, has found TD participants unable as a group to demonstrate a significant semantic priming effect, in contrast to non-TD schizophrenia participants, psychiatric controls or normal controls (Besche et al., 1997; Passerieux et al., 1997). In these studies the SOA was 500 ms and relatedness proportion was 50% (though in Passerieux et al. they indicate that the "proportion of related prime-target pairs was 16.7%," this figure appears to be the proportion of total pairs of letter strings, i.e., including pairs with non-words, that are related word pairs). Thought disorder was defined in Besche et al. as TLC score greater than 7, and in Passerieux et al. as TLC score greater than 6. Interestingly, Besche et al. report syntactic priming to be intact in the TD group that failed to demonstrate semantic priming. Barch

et al. (1996), as discussed earlier, found impaired semantic priming among 100 schizophrenia participants compared to normal or psychiatric controls, only at the longest SOA (950 ms). This was true for the schizophrenia participants whether identified as TD/non-TD, or medicated/unmedicated. In contrast, semantic priming was intact for the schizophrenia group at SOAs from 200 ms to 700 ms.

Aloia et al. (1998) have also found deficits in semantic priming effects in conditions that appear to reflect controlled processing. Twenty schizophrenia inpatients (divided into *high TD* and *mild TD* groups by a cut-off global score of 2 on the TLC; see companion article by Goldberg et al., 1998) and 21 normal controls performed a word pronunciation task with an ISI of 350 ms (and no fixed SOA, as indicated above). Of the related prime–target pairs (63% of all word pairs), equal numbers (or 21% each of the total) were classified as either *high relatedness*, *medium relatedness*, or *low relatedness*, as derived from word-association norms. The high TD group showed no significant semantic priming (expressed as percentage semantic priming) at any of the three word-relatedness conditions, whereas control and mild TD groups showed significant semantic priming at the high- and medium-relatedness conditions. Furthermore, group comparisons of effect sizes showed controls to have significantly greater semantic priming than the high TD group for high- and medium-relatedness pairs, and significantly greater semantic priming than the low TD group for high-relatedness pairs. The mild TD group in turn showed significantly greater semantic priming than the high TD group for high-relatedness pairs as well (this study also examined relationships between semantic priming and various other cognitive measures, as discussed later).

Spitzer's group, in the course of a primary focus on spreading activation, has obtained equivocal evidence for increased semantic priming effects in three studies with schizophrenia participants performing LDTs under controlled processing conditions (each of the three studies described below have been described in the prior sections on automatic priming processes). In Spitzer et al. (1994) the semantic priming effect at an SOA of 700 ms (and a 39% relatedness proportion) for a group of 70 schizophrenia participants is greater than that for the comparison group of 44 normals; unfortunately this control group is historical. Spitzer et al. (1993b) reported a greater absolute indirect semantic priming effect for 32 schizophrenia participants at an SOA of 700 ms (calculated from the indicated prime display time of 200 ms plus the ISI of 500 ms), and 67% relatedness proportion; this was not statistically significant in comparison to a concurrent group of 32 normal controls. Finally, Spitzer et al. (1993a) also report a significant increase in absolute direct semantic priming and a trend for an increase in absolute indirect semantic priming for a TD schizophrenia group compared to normal controls, again at an SOA of 700 ms and a 67% relatedness proportion; however, both of these differences are abolished upon conversion to percentage semantic priming scores. Given this pattern of findings, Spitzer et al. appropriately focus on aspects of automatic

processes (see section above), as their findings are inconclusive regarding differences in controlled/attentional processing.

In addition, as stated earlier, the results from our laboratory indicate that schizophrenic participants exhibit impaired semantic priming particularly in conditions where semantic matching or other postlexical processes are operative, such as in LDTs (in comparison to word pronunciation tasks), or with high-associate pairings or "horizontal" category relationships between prime and target. Therefore, our major conclusions overall from the results of our studies are that schizophrenics as a group most consistently exhibit deficits in postlexical, controlled processes (i.e., semantic matching) as they relate to semantic priming impairments. These may coexist with changes in automatic spread of activation that manifest variously as enhanced or impaired automatic semantic priming effects, reflecting heterogeneity among schizophrenic patients.

One other study is reported that may involve postlexical processes in an experimental design unusual among the present literature. This is the report by Kwapil et al. (1990), who employed a word pronunciation task, with the target word systematically degraded (in a manner referred to as titration) in order to adjust the rate of accurate identification of both related and neutral words to approximately 50%. They compared a group of 21 schizophrenic outpatients to 18 bipolar patients and 21 normal controls; the SOA was 500 ms, with a 33% relatedness proportion. Each group's performance was expressed as percentage correct responses. Facilitation was calculated as the increase in percent correct responses to targets following related primes, compared to that for targets following a neutral prime. Inhibition was calculated as an analogous decrease in percentage correct responses to unrelated pairs compared to neutral prime–target pairs. This approach was employed to address the issue raised by Chapman and Chapman (1988, 1989) of spurious findings when calculating difference scores between two dichotomously scored free-response tasks. Using this paradigm, Kwapil et al. found the schizophrenia group to have both significantly enhanced facilitation for related pairs (compared to either bipolar or normal participants) and a small, nonsignificant enhancement of inhibition for unrelated pairs. The enhanced facilitation was discussed in terms of automatic spreading activation; the lack of significant difference in a measure of inhibition was noted as consistent with the influence of processes other than spreading activation.

There are a few issues to be addressed regarding this study. Word pronunciation tasks preclude postlexical semantic matching, one of the important controlled processes involved in the LDT (deGroot, 1984). However, the use of degraded targets increases priming effects due to an uncharacterized mechanism (Neely, 1977), which may involve a postlexical "guessing" strategy. Together with the "long" SOA utilized, it therefore seems likely that this experimental paradigm assesses both automatic and controlled processes. It should also be noted that resolving these results

with the other studies is difficult, given the emphasis on accuracy rather than speed. While the semantic priming phenomenon can be observed as an accuracy enhancement (Neely, 1991), the relationship of speed to accuracy in this paradigm is probably not characterized well enough to easily compare these results with those where semantic priming is seen as an enhanced RT.

Summary of Studies Addressing Controlled/Attentional Processes in Semantic Priming

The results of studies examining the performance of schizophrenic participants in experimental conditions favoring controlled processes are relatively consistent. The data demonstrate impairments when increasing cognitive loads are implemented, or, more generally, impairments in the ability to employ cognitive strategies. While schizophrenic participants are able to utilize these strategies to enhance semantic priming effects over those due to spread of activation alone, they are impaired relative to normals in both the degree of enhancement as well as the overall magnitude of priming under controlled conditions. This observation is consistent with a large body of literature addressing other measures of deficits in attentional or strategic functions in schizophrenia (e.g., Braff, 1993; Dawson & Nuechterlein, 1984; Nuechterlein, 1977). This includes electrophysiological measures of strategic operations of semantic memory in language comprehension (studies investigating the relationship of electrophysiology to semantic priming in schizophrenia are discussed below).

SEMANTIC PRIMING EFFECTS AND CLINICAL PHENOMENOLOGY

Much of the existing literature has been concerned with the relationship of semantic priming effects to clinical phenomenology. Because these studies were described in detail in preceding sections, we will now briefly summarize the findings from these studies, with a specific focus on the clinical variables under study.

The various clinical subtypes of schizophrenia seem to generally share similar semantic priming effects. Passerieux et al. (1995) did find a group of 10 paranoid schizophrenia participants (categorized by criteria from the *International Classification of Diseases, 9th edition*; also as undifferentiated schizophrenia by DSM-III-R) to be lacking in inhibition (i.e., longer RT for the unrelated compared to neutral-prime condition), in contrast to the performance of a group of 7 ICD-9 hebephrenic schizophrenia participants (disorganized schizophrenia by DSM-III-R), which in turn was comparable to that of a group of normal controls. However, facilitation (i.e., faster RT for related compared to neutral-prime condition) was similar across the three groups. These results were obtained in an LDT with an SOA of 240 ms. Our group has failed to find significant

differences between paranoid and nonparanoid schizophrenia subgroups under either automatic or controlled conditions (Ober et al., 1997); similar results were obtained by Chapin et al. (1992), where paranoid, undifferentiated and schizoaffective groups were compared.

A study examining the relationship of length of illness (LOI) to semantic priming effects has been reported (Maher et al., 1996). Thirty schizophrenia participants with LOI ranging from 5 to 31 years performed an LDT with an SOA of 250 ms and 50% relatedness proportion. Semantic priming effects in this group declined with increasing LOI in a manner not accounted for by age or chlorpromazine equivalents. While this study is cross-sectional in design, it suggests that the natural course of schizophrenia may involve either declining spreading activation or increasing inhibition (or both). In addition, these results may have significant methodological implications (as discussed below). In a pilot study addressing the state *versus* trait dependency of semantic priming effects, Spitzer's group reported retest data for 11 of a sample of 70 schizophrenia participants, tested initially as inpatients (Spitzer et al., 1994). These participants performed an LDT again "on remission," usually 4 to 6 weeks later. In this group, retest semantic priming was reduced (partially normalized) from before, but not significantly; the medication status of the participants at retest was not specified.

Most of the semantic priming studies involving clinical variables attempt to examine the relationship of semantic priming effects to TD. TD schizophrenia groups have been found to demonstrate enhanced semantic priming (Henik et al., 1995; Manschreck et al., 1988; Spitzer et al., 1993a; 1993b; Weisbrod et al., 1998), semantic priming comparable to those of non-TD schizophrenia groups or controls (Barch et al., 1996; Blum & Freides, 1995), or reduced semantic priming effects compared to those of non-TD schizophrenia groups or controls (Aloia et al., 1998; Besche et al., 1997; Henik et al., 1992; Passerieux et al., 1997). In addition, Spitzer's group has reported on a non-clinical participant sample among whom high scorers on a scale derived from the language-related complaints of schizophrenia patients showed significantly increased semantic priming compared to the low scorers on this scale (Moritz et al., 1999). In almost all of these studies, the TD criterion consists of an arbitrary threshold score (which is not standardized across studies) on a single item from a symptom rating scale (see Bhandari & Curtis, 1998, as well as Spitzer, 1998, for comment). Furthermore, direct statistical comparison between TD and non-TD schizophrenia groups has generally been lacking, with few exceptions (Aloia et al., 1998; Passerieux et al., 1997; Spitzer et al., 1993a, 1994). In these few studies, the sample sizes are small (Passerieux et al., 1997), significant effects are abolished upon conversion to percentage semantic priming scores (this applies to indirect semantic priming in Spitzer et al., 1993a), data is pooled across conditions for statistical analysis (Spitzer et al., 1994) or the condition (e.g., SOA) is unspecified (Aloia et al., 1998). These latter two methods confound any distinction

between automatic and controlled processes that might be specifically associated with TD. On a more theoretical level, the relationship of semantic priming phenomena (or lexical access in general) to other aspects of language and “thought” and its expression appears to be complex and poorly characterized at this time (Rieber & Vetter, 1994; Rochester, 1980; Spitzer, 1997). As a result, it is presently unclear how semantic priming disturbances (should they be reliably demonstrated) may be related to TD as manifested clinically.

RELATIONSHIP OF SEMANTIC PRIMING EFFECTS TO OTHER NEUROPSYCHOLOGICAL AND NEUROBIOLOGICAL FACTORS

Relationship of Semantic Priming to Phonological and Syntactic Priming

Several aspects of language function have been assessed in relation to semantic priming. Spitzer et al. (1994) had 70 schizophrenia inpatients perform an LDT where word pairs were related either semantically or phonologically (i.e., sharing a rhyming relationship), across SOAs of 200, 400 and 700 ms. This task included a 39% semantic relatedness proportion and a 22% phonological relatedness proportion. The control and non-TD schizophrenia groups both appeared to display slower RTs in response to phonologically related primes (significant at the 200 ms SOA and a trend at the 400 ms SOA). In contrast, the TD participants at SOAs of 200 and 400 ms were found to lack this slowed response. As this TD group also displayed increased semantic priming with data pooled across the three SOAs, the investigators concluded that a deficit in “automatic inhibitory processes” might underlie both phenomena. It is unclear how the slowing in this experimental paradigm, which they term “inhibition,” relates to inhibition as it is operationally defined in the semantic priming literature as a slowed response to a prime–target pair in comparison to a neutral prime–target pair. Besche et al. (1997) report on a sample of 24 TD schizophrenia inpatients performing a syntactic priming task, wherein a prime that is syntactically congruent (i.e., a prime that can pair with the target in a grammatically acceptable manner) facilitates recognition of the target. In this task, with an SOA of 500 ms and a 50% syntactic congruence proportion, the processing of syntactic congruence was intact in the schizophrenia group even in the absence of significant semantic priming effects. This is consistent with other reports of preservation of (and independence of) syntactic processing in the face of deficits in the operation of semantic memory (Carpenter, 1976; Miller & Phelan, 1980).

Relationship of Semantic Priming Effects to Other Neurocognitive Functions

Our group has recently reported a study with 26 schizophrenia outpatients who were medication-free for 1 week prior

to assessment with a neuropsychological battery, clinical rating scales, and a lexical decision semantic priming paradigm (Poole et al., 1999). The latter included automatic (an SOA of 260 ms and a 15% relatedness proportion) and controlled (an SOA of 1000 ms and a 50% relatedness proportion) conditions. The index of response inhibition (three alternation/inhibition tasks from Luria’s Motor Signs Inventory) positively correlated with semantic priming effects obtained in the automatic condition (which does not rule out post-lexical effects as the task was a LDT). This correlation was preserved after controlling for IQ. The index of executive dysfunction (perseverative and non-perseverative errors on the Wisconsin Card Sorting Test, WCST) was negatively correlated with controlled semantic priming; this correlation was reduced after controlling for IQ. These findings were irrespective of clinical subtype. These results suggest that findings of enhanced priming under automatic conditions may be related in particular to impaired inhibitory processes, and that the ability to employ controlled processes in a semantic priming task may require intact executive function.

The Aloia et al. (1998) study described earlier involved assessing 20 medicated schizophrenia inpatients on a neuropsychological battery. Employing a word pronunciation task with an ISI of 350 ms (and no fixed SOA), these authors found semantic priming from medium-related word pairs to be significantly correlated with a semantic fluency score (calculated as the difference between semantic fluency and phonological fluency). Measures of attention, working memory and executive function were not associated with semantic priming effects, as expected for a priming paradigm favoring automatic processes.

Relationship of Semantic Priming to Cerebral Lateralization

Two reports have attempted to address hemispheric asymmetry as a possible factor in semantic priming effects among schizophrenia participants. Weisbrod et al. (1998) studied 40 schizophrenia participants and 38 normal controls in an LDT with an SOA of 250 ms, and 67% relatedness proportion (of which half were intracategorically related or “direct” pairs, and half were intercategory related or “indirect” pairs). The word pairs were presented to either the right visual field (RVF, and therefore, left cerebral hemisphere) or left visual field (LVF, and therefore, right cerebral hemisphere), with participants instructed to fixate on a central point on the screen (Spitzer, personal communication). Direct semantic priming did not differ between visual field presentations for either group. In contrast, TD participants showed positive indirect semantic priming with RVF stimuli (as did all groups for LVF stimuli); controls and non-TD groups in contrast showed slight slowing in response to related word pairs. The authors suggest that both “less focused activation of semantic networks” as well as TD may be localized to the left hemisphere. Evidence supporting this conclusion is given by a combined semantic

priming/event-related potential study of normal participants conducted by this group (Kiefer et al., 1998). They found that normal participants exhibit the N400 priming effect (defined below) to indirectly-related word pairs only in the right hemisphere (over the inferior frontotemporal region), in contrast to the bilateral N400 priming in response to directly related word pairs. Blum and Freides (1995) also varied the visual field location of stimuli in an LDT with an SOA of 350 ms and a 33% relatedness proportion. Participants were instructed to fixate on a central point on the screen, though eye movements were not recorded. They found a significantly faster RT for RVF stimuli *versus* LVF stimuli; however, semantic priming effects were not significantly different between stimulus locations. It remains unclear, therefore, whether schizophrenic participants experience a cerebral hemispheric asymmetry in a manner that significantly impacts semantic priming effects.

Relationship of Semantic Priming Effects to Electrophysiological Findings

Electrophysiological recording during semantic priming tasks has yielded interesting observations pertaining to information processing deficits in schizophrenia. Much of the investigation of event-related potential (ERP) correlates of abnormalities in the operation of semantic memory has focused on the negative deflection in the electroencephalogram (EEG) that occurs with a 400 ms latency after a stimulus, known as the N400. This ERP is evoked by a stimulus which is "potentially meaningful within a complex associative cognitive system" (Halgren, 1990). The N400 is elicited to word targets which are either unrelated to single word primes (Bentin, 1987; Bentin et al., 1985; Kutas & Hillyard, 1984; Polich, 1985; Rugg, 1985) or incongruent with incomplete sentences (Kutas, 1997; Kutas & Hillyard, 1980; Van Petten, 1995) preceding the target. The N400 is often referred to as the "mismatch negativity." There is evidence that the generator of this potential may be located in the parahippocampal anterior fusiform gyrus, on the basis of intracranial recordings (McCarthy et al., 1995; Nobre et al., 1994); the underlying anatomy remains a subject of active investigation. While this response has usually been considered as an index of semantic congruency determination (probably because it is more commonly assessed in sentence completion tasks than in single word priming tasks), it may be more precisely related to semantic expectancy, since unexpected but semantically congruent sentence endings elicit N400 potentials in normal participants (Fischler et al., 1983; 1984; Kutas & Hillyard, 1980). In single word priming tasks, the N400 evoked in response to a target preceded by an unrelated prime is larger (in normal participants) than that evoked by a target preceded by a related prime; this difference in magnitude is referred to as the "N400 priming effect."

A diminished N400 priming effect has been seen among schizophrenic groups in single word semantic priming paradigms (Condray et al., 1999; Koyama et al., 1991). Con-

dray et al. demonstrated a diminished N400 priming effect (relative to controls) in both medicated and unmedicated subgroups of their schizophrenic participants. However, the N400 priming effect was enhanced in the controlled condition (relative to the automatic condition) to the same degree for the schizophrenia group compared to controls, though the N400 priming effect remained smaller for the schizophrenia group compared to controls. It is important to consider that the diminished N400 priming effect may in some instances reflect the enhanced N400 response to related primes rather than a diminished N400 response to unrelated primes. This may be the case in Condray et al., where the difference for unmedicated schizophrenic participants responding to related prime-target pairs in automatic conditions was particularly large, though not significantly different from control, or unmedicated schizophrenic participant groups. In many N400 studies, these comparisons are not made; instead, either the "priming effect" alone is calculated, or alternatively the N400 amplitudes are averaged across all conditions for participant group comparisons. This may obscure the possibility that schizophrenics are manifesting general deficits in using contextual information to generate expectancies, rather than a more specific impairment in recognizing incongruity/unrelatedness (see Nestor et al., 1997, for discussion).

The relationship of N400 priming effects to semantic priming effects is presently unclear. In the two LDTs reported, despite consistently diminished N400 priming effects, the semantic priming effects in the schizophrenic participant groups vary across the three studies. In the Koyama et al. (1991) study, 13 schizophrenics and 26 normal controls performed an LDT with an SOA of 1000 ms, and a relatedness proportion of 50%, with all related pairs consisting of antonyms in Kanji (Japanese) characters. The semantic priming effects appeared to be equivalent between the schizophrenia group and the control group when the effect was calculated as the difference between the RT to targets preceded by related primes, and the RT to targets preceded by unrelated primes. When semantic priming was calculated as the difference between the RT to targets preceded by related primes, and the RT to targets preceded by neutral primes, the effect was considerably larger for the schizophrenia group; however, absolute RTs for the schizophrenia group are much slower under each condition, and, in any case, no statistical comparisons are performed between groups for the semantic priming effects. In the Condray et al. (1999) study, 37 schizophrenia participants voluntarily admitted to an inpatient research unit were quasirandomly assigned to perform an LDT with either an automatic processing condition (with an SOA of 350 ms and 33% relatedness proportion) or a controlled condition (with an SOA of 950 ms and 67% relatedness proportion). A significant semantic priming effect was observed for both schizophrenia and control groups overall (across conditions); however, it was significantly smaller for the schizophrenia group compared to controls. The schizophrenia participant group who performed the controlled condition LDT showed

increased semantic priming effects relative to the schizophrenia participant group who performed the automatic condition LDT, though the schizophrenia group priming effect remained smaller than the control group effect even in the controlled condition. Thus, Condray et al. provide some evidence that diminished N400 priming and semantic priming effects can co-occur together in schizophrenic participant groups, and furthermore that they can be improved (but not normalized) in parallel by shifting the task demands to include increased controlled processing. Another methodological consideration is important to consider in these studies. The authors of these papers do not directly address the possible confounding effects of assessing ERP phenomena in a paradigm that involves a behavioral response. Each of the studies report criteria for excluding potentials due to artifact (most commonly from muscle contraction). However, other studies (in normal participants) have identified many scalp potentials that may precede, accompany, or even follow a motor response; the possible contribution of these to the altered potentials observed among schizophrenic participants has not been addressed (see Regan, 1989, for a thorough discussion of these and other methodological issues in ERP studies). Kutas and Hillyard (1989) have demonstrated that N400 priming effects can be elicited in response to word pairs on the basis of semantic relatedness, in a manner dissociated from other electrical potentials that occur adjacent in time. Nevertheless, it remains unclear to what degree the N400 effects observed in the studies detailed above, may result from performance-related components of processing unrelated to semantic memory access.

Other disturbances in ERPs have been reported in relation to semantic priming deficits. We (Vinogradov et al., 1996) have reported a study involving 13 of the schizophrenia participants examined in Ober et al. (1997) who also performed an auditory event-related potential (AERP) task. Another measure in the ERP paradigm which appears to be altered among schizophrenia participants is the positive potential with a 50 ms latency (the P50) in response to a "test" click, which in normals is reduced when preceded by a "conditioning" click. This P50 conditioning:test ratio, which is larger in many schizophrenia participants (believed to reflect an impaired sensory "gating" response; see Freedman et al., 1991), was highly correlated with intracategory semantic priming effects under automatic priming conditions. A "defect in inhibitory pathways" was suggested to underlie both phenomena. In the Condray et al. study the positive deflection with a 300 ms latency (the P300, considered to be a measure of the allocation of attentional resources) also showed an attenuated priming effect in the schizophrenia group compared to controls. The P300 peak magnitude was paradoxically smaller for related words compared to unrelated words among the drug-free schizophrenia group in the automatic condition (in contrast to the controls which showed the usual enhancement for related words); this effect was reversed in the controlled condition though remained smaller than that for the control group.

The Role of Monoamines in Semantic Priming Effects

Modulation of semantic priming effects by central nervous system monoaminergic activity is suggested by two studies of normal participants. Spitzer et al. (1996) demonstrated indirect semantic priming effects which appeared to parallel plasma levels of an active metabolite after administration of psilocybin (an indolealkylamine hallucinogen with multiple monoaminergic effects) to eight normal participants. Kischka et al. (1996) found exogenous L-DOPA to decrease the indirect semantic priming effect observed relative to placebo-administered control semantic priming effects, in 31 normal participants performing an LDT with either an SOA of 200 or 700 ms. In addition, the role of neuroleptics in modifying semantic priming effects is suggested by Barch et al. (1996), who obtained a significant positive correlation between participants' chlorpromazine equivalent dosages and semantic priming effects at SOAs ranging from 200 ms to 700 ms, and a negative correlation at an SOA of 950 ms. This relationship appears to be mediated by an increase in RTs, however, as it was abolished when semantic priming effects were corrected for overall RTs. The first two studies described above suggest a role for brain monoamine systems in modulating the processes subserving semantic memory operation.

MAJOR METHODOLOGICAL ISSUES

Participant Characteristics

Variation in the selection and characterization of the participant groups employed in the various studies has posed problems in interpretation of the data. In particular, many studies have employed hospitalized participants, and virtually all have involved participants on medication at the time of testing. The use of acutely ill inpatients makes state *versus* trait determinations difficult to discern; medication status clearly impacts the neurocognitive functions that appear to be related to semantic priming processes (Spohn & Strauss, 1989), and there is direct evidence for an association between neuroleptic dose and semantic priming effects, which appears to be mediated by an increase in RTs (Barch et al., 1996). Another clinical variable that should be considered in participant sampling is illness duration, which may be a factor in semantic priming effects even after age and neuroleptic dose are accounted for (Maher et al., 1996), though no study has either examined the effects of cumulative neuroleptic exposure or followed participants longitudinally. While it is presently unclear if DSM-related diagnostic subgroups show differential semantic priming effects (see above), we have recently obtained evidence that discrete symptom clusters co-vary with schizophrenia participants' performance on an LDT (Minzenberg et al., 2001). Unfortunately, many studies have not characterized their sample by clinical features such as illness subtype or duration.

It is even more important to carefully and reliably assess clinical variables when they are implicated in the primary hypothesis under study. In particular, the definition and assessment of TD has been inconsistent across studies (as noted above). Thought disorder is surely one of the most complex and variegated of all phenomena in psychopathology, and poorly characterized in terms of its neurocognitive and pathophysiological basis (Andreasen, 1982; Berenbaum & Barch, 1995; Rieber & Vetter, 1994; Spitzer, 1997; Willis-Shore et al., 2000). Therefore attempts to associate TD to semantic priming effects should involve a more detailed and reliable assessment of the schizophrenia participant sample. Several researchers have begun to address this issue (Aloia et al., 1998; Besche et al., 1997; Blum & Freides, 1995; Passerieux et al., 1997).

Experimental Conditions

As discussed above, the semantic priming studies published to date have varied widely in their experimental designs, often making it difficult to interpret results or resolve conflicting data across studies. For instance, the experimental parameters in many studies have been set in a manner that precludes any clear distinction between automatic and controlled processes. This has confounded the assessment of hypotheses in many studies. Prime–target relationships also have an important effect on task performance and any attribution of impairment to schizophrenia participants as a result. Semantic priming effects can vary significantly, for example, when RTs to related prime–target pairs are compared with RTs to unrelated prime–target pairs, as opposed to when they are compared with RTs to neutral prime–target pairs (deGroot et al., 1982). A second example comes from Spitzer, who suggests that indirect semantic priming may be a more sensitive measure of spreading activation than direct semantic priming (Spitzer, 1997). The categorical relationships, and typicality, of prime–target pairs also modify semantic priming effects (Neely, 1991; Rosch, 1975). In addition, the influence of these variables theoretically reflects not only the efficiency of lexical and semantic/conceptual access, but is dependent on the underlying semantic/conceptual memory organization as well (a subject beyond the scope of this review).

Psychometric Issues

No studies have yet been reported addressing psychometric aspects of semantic priming task performance among schizophrenia participants. Performance characteristics such as construct validity, internal reliability, and test–retest reliability have not been determined. This significantly constrains an investigator’s ability to either conclusively interpret the data generated or to resolve the results across different studies. The psychometric performance of this task in non-clinical populations remains undetermined as well. There is recent evidence that measures of “implicit” memory are less reliable than those for “explicit” memory, on a range of

verbal tasks including a degraded word identification task (Buchner & Wippich, 2000). By analogy, perhaps hypothetical differences in reliability between the measures of automatic *versus* controlled semantic priming effects account for the relative inconsistency in automatic priming effects across the various studies, in contrast to controlled priming effects. Stolz et al. (2000), in a preliminary report addressing this issue, suggest that the test–retest reliability of automatic semantic priming effects is much lower than that for controlled priming. In addition, the ecological validity of semantic priming tasks, that is, the relevance to psychological function in everyday human experience, has not been evaluated.

Expression of the Data and Statistical Analyses

One problematic aspect of performance on semantic priming tasks is the relationship of RTs to semantic priming effects. The mean RTs of schizophrenia participant groups have been reported as longer than that of a control group in every study but one; this is not surprising given the phenomenon of longer and more variable RTs among schizophrenia participants on virtually every cognitive task reported in the literature (Nuechterlein, 1977; Schatz, 1998; Vinogradov et al., 1998). Medication effects may also give rise to slower RTs (Barch et al., 1996). The pathophysiological significance of general RT slowing in schizophrenia remains unclear, as does the relationship of general RT slowing to semantic priming phenomena (cf. the literature empirically addressing this unresolved issue in normal aging: Laver & Burke, 1993; Lima et al., 1991; Myerson et al., 1992). Nevertheless, a few data-analytic issues merit discussion. First, the interpretation of semantic priming effects is problematic in the study of either subject groups or single individuals, such as those with schizophrenia, where reaction time slowing (compared to normal subject groups) is the rule. This is related in part to the fact that semantic priming effects are calculated as difference scores. As mentioned above in the context of the Kwapil et al. study, the difference score of two reaction time (or accuracy) measures is artifactually related to the sum of the two scores (see also Chapman & Chapman, 1988, 1989, for fuller discussion). This is because, as Kwapil et al. state, “Sources of difficulty [manifest in slower processing times] more often interact instead of acting additively” (p. 216). This is likely a common source of type I errors in the present literature, specifically, interpreting subject group differences (such as enhanced automatic semantic priming in schizophrenic groups) on the basis of artifactually inflated priming effects. Despite this observation, only a few investigators have attempted to account for potentially spurious semantic priming effect size determinations (Aloia et al., 1998; Barch et al., 1996; Kwapil et al., 1990; Poole et al., 1999; Spitzer et al., 1993a, 1994, 1996; Weisbrod et al., 1998). Most of these studies have done so by utilizing a proportion transformation, usually with the priming effect expressed as “per-

centage priming.” This too may be inappropriate, in light of another issue present in reaction time studies. The (typically linear) formulas that describe slowing of many other clinical populations (relative to normal subject groups) include a constant term which is not accounted for in a proportion transformation. In other words, when linear Brinley functions are generated from the data, they typically fail to have an intercept through the origin, and therefore, the data violate an assumption inherent in this method of transformation (see Faust et al., 1999, for a detailed discussion of this issue). Faust and colleagues recommend linear regression and *z*-score transformations in order to more appropriately meet a goal which we believe is also fundamental to the present body of research:

to separate global information-processing factors—which influence the large-scale structure of response latencies (on the order of 100s of milliseconds) across a wide range of individuals and conditions—from task-specific factors, which influence the small-scale structure of response latencies (on the order of 10s of milliseconds; e.g., semantic priming). (Faust et al., 1999, p. 787)

Inadequate sample size appears to constrain the ability of many investigators to detect differences among schizophrenia in comparison to control groups. As relatively small effect sizes are combined with routinely increased variability among schizophrenia participants’ performance, semantic priming tasks need to include larger sample sizes. This is necessary to avoid type II errors in hypothesis testing, or, in other words, spurious conclusions that semantic priming effects are not demonstrable.

Finally, a crucial issue in statistical analyses of schizophrenia participants’ performance, which has profound implications for any theory of schizophrenia etiology and pathophysiology, involves the issue of heterogeneity. There is evidence from the foregoing literature that schizophrenia patients manifest heterogeneity in semantic priming tasks; this appears true for cognition in schizophrenia in general. Heterogeneity in semantic priming task performance may potentially be related to either multiple clinical factors (such as acuity or duration of illness, symptom profile, medication exposure, etc.), or fundamental differences in underlying etiological processes. Future investigations will need to properly assess and control for the first set of factors so that the second set of factors can be explored. Unfortunately, the assessment of group means when comparing the performance of schizophrenia participants to that of controls minimizes the recognition of both sources of heterogeneity. A common misinterpretation of results in the schizophrenia literature involves comparing group means on a task, which inevitably show schizophrenia as a group to perform more poorly (or at least differently) than a group of controls, and then concluding that schizophrenia is a unitary phenomenon, while the presumption of homogeneity among the profile of deficits (or degree of impairment) within that group of schizophrenic patients is never addressed. There-

fore, use of other statistical methods (such as correlational analysis; Humphreys, 1978), as well as establishing clearly defined subgroups of schizophrenia participants with adequate sample sizes for these subgroups, is advised in order to more powerfully establish associations among continuous variables and to characterize more fully the differential task performance among schizophrenia participants.

CONCLUSIONS

The investigation of semantic priming phenomena in schizophrenia is at an early stage, and yet several interesting conclusions are suggested by the literature.

1. Schizophrenia appears to be characterized by both automatic and controlled processing-related changes as manifested in semantic priming paradigms.
2. With regard to automatic processes, groups of schizophrenia participants may be composed of individuals with enhanced spreading activation in semantic memory networks as well as individuals who demonstrate normal spreading activation (and possibly individuals with slowed spreading activation). This heterogeneity in the speed of automatic spreading activation may reflect clinical factors such as illness acuity and duration and medication exposure, as well as heterogeneity in the underlying etiopathophysiology of the disorder.
3. Changes in controlled processing in semantic memory, on the other hand, appear more homogeneous in nature. Schizophrenic participants tend to show reduced semantic priming effects compared to controls under a number of conditions, and this appears to reflect underlying deficits in attentional or strategic functions.
4. In addition, the abnormal semantic priming effects that are obtained in the two domains may well be partially independent of each other and reflect partially independent processes. These different impairments may be associated with different (though likely overlapping) sets of neural circuitry and related neurocognitive functions.

For example, in experimental conditions that favor automatic processes such as spreading activation, efficient information processing may rely primarily on “bottom-up” processes. This term refers to fundamental elements of information processing in the brain, which may be represented by the rate of signal generation or efficiency of signal transmission/transduction of individual neurons, as well as the temporal correlation of groups of neurons (Koch, 1993). This may be manifest at the behavioral level, for instance, in the speed of processing in the performance of experimental tasks, as well as in sensory gating phenomena (Freedman et al., 1991). Slowing on timed task performance is a feature of the information processing impairments routinely demonstrated in schizophrenia (Nuechterlein, 1977; Schatz, 1998; Vinogradov et al., 1998). Interestingly, this slowing may be largely related to impairments in central

slowing may be largely related to impairments in central processing rather than simply in input or output (i.e., sensorimotor) components of information processing.

In contrast, dysfunction of “top-down” processes may underlie the impaired strategies contributing to the lexical and semantic/conceptual access and retrieval deficits observed in the semantic priming paradigm. These and other higher order processes may be distributed across multiple associational cortical networks (McClelland & Plaut, 1993), with a particular role for prefrontal circuitry in establishing and coordinating top-down phenomena. Possible defects in the structure of semantic memory organization among schizophrenic participants are also likely to reflect underlying prefrontal circuit dysfunction. It is possible, furthermore, that the organization of semantic memory (as well as the operation of access to this structure) may be an active, dynamic process in a manner analogous to the maintenance of primary sensory cortical representations (Merzenich & Sameshima, 1993).

Heterogeneity in the presence or degree of dysfunction within these two neurocognitive domains in schizophrenia may, in turn, be related to the diversity in symptom profiles. In particular, the possible contribution of disturbances in lexical and semantic/conceptual access to the clinical phenomenon of TD is an intriguing one, and likely a highly mediated relationship that awaits further characterization. Other clinical factors, such as illness acuity/duration and medication status, introduce ambiguity in interpreting the results, but also suggest interesting aspects of the pathophysiology underlying semantic priming-related deficits. Some hypotheses that arise from this work include the possible decline in spreading activation in the natural course of schizophrenia (Maher et al., 1996), and the role of monoamines in modulating semantic network activation (Kischka et al., 1996; Spitzer et al., 1996).

Methodological issues are, and will continue to be, of paramount importance in evaluating this literature. Psychometric aspects of the semantic priming task have yet to be evaluated among schizophrenia participant populations. In addition, experimental conditions (as well as statistical analyses of data generated) need to be brought in line with what is known about semantic priming task parameters, including the implications of these variables for interpreting results within the models of information processing in general, and the models of lexical access in particular.

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