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Increased Stroop facilitation effects in schizophrenia are not due to increased automatic spreading activation

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Abstract

Studies using the single trial Stroop task consistently reveal increased reaction time (RT) facilitation effects among schizophrenia patients. One possible mechanism underlying this effect is increased automatic spreading activation in semantic networks. The current study was designed to test this hypothesis. We administered the Stroop task and two semantic priming tasks to the same subjects. Patients showed greater Stroop RT facilitation than controls, no evidence of increased semantic priming at short stimulus onset asynchronies (SOAs), and reduced semantic priming at long SOAs. In addition, abnormal Stroop performance was related to the severity of Disorganization symptoms. These results are inconsistent with the spreading activation hypothesis. Alternative hypotheses regarding the source of Stroop task performance deficits in schizophrenia are discussed.

Keywords: Automatic spreading activation; Schizophrenia; Semantic priming; Stroop facilitation effects

1. Introduction

Disturbances of attention have been considered a prominent aspect of cognitive dysfunction in schizophrenia since the earliest descriptions of the illness (Kraeplin, 1950; Bleuler, 1950). The ability to select, from the range of available information sources, that which is most relevant for goal-directed behavior is an essential element of many higher cognitive functions, including short- and long-term memory and language production. Thus, establishing the precise nature and neural substrates of selective attention pathology in schizophrenia will provide an important link between pathophysiology and functional disability in this illness.

In the cognitive science literature, the Stroop (Stroop, 1935) color naming task has been used as a paradigmatic measure of selective attention [see MacLeod (1991) for a review]. This task has tremendous face validity as a measure of selective attention. Participants are presented with words printed in colors. They are instructed to ignore the word and to name the color in which it is printed as quickly and accurately as possible. When the word and its color conflict (such as RED printed in blue ink), participants are slower than when there is no such conflict. This very robust and reliable effect is called interference, and is thought

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to result from the obligatory nature of word reading disrupting color naming performance (MacLeod, 1991). In its original form, the Stroop task was presented as lists of colored words on cards. In subsequent years, Stroop studies of attention have presented one stimulus at a time on a computer screen (MacLeod, 1991). As a result of using this single trial design, a second Stroop effect has been documented. When presented with a word printed in a congruent color (e.g., RED printed in red ink), participants are faster than when they are presented with a neutral, color-unrelated stimulus (such as DOG printed in red ink). This effect, referred to as facilitation, was first reported by Dalrymple-Alford and Budayr (Dalrymple-Alford, 1972).

Not surprisingly, a number of studies have used the Stroop task to examine selective attention in schizophrenia. Many early studies using Stroop cards (Wapner and Krus, 1960; Golden, 1976; Abramczyk et al., 1983; Wysocki and Sweet, 1985; Everett et al., 1989) found that schizophrenia patients were slower than controls when color naming the color conflict card (color and word incongruent). These findings were interpreted as evidence of an increased influence of the irrelevant stimulus dimension (the word). However, in these early studies, patients were invariably slower on all conditions. The two studies which used a difference score did not find evidence for selectively increased interference effects among schizophrenia patients (Abramczyk et al., 1983; Everett et al., 1989). Thus, it is difficult to infer from these Stroop card studies that schizophrenia patients experience a differential deficit in selective attention.

More recently, investigators have begun to use single trial Stroop procedures to investigate selective attention in schizophrenia. A number of studies have shown a particular pattern of reaction time (RT) performance among schizophrenia patients tested with this procedure: increased facilitation, but not increased interference in RTs (Carter et al., 1992, 1993). In addition, those studies examining accuracy have found that this pattern of RT performance is combined with increased error interference (Taylor et al., 1996; Henik et al., 1998; Cohen et al., in press). These findings have been replicated in both medicated and unmedicated patients, and were evident even when subgroups of patients and controls with similar overall reaction times were compared (Carter et al., 1992; Taylor et al., 1996). This latter finding suggests that increased RT facilitation is not merely an artifact of overall slowing in schizophrenia patients.

One hypothesis regarding the nature of the mechanism underlying increased RT Stroop facilitation and error interference in schizophrenia is that these phenomena reflect an abnormality in an automatic component of processing (e.g., Carter et al., 1992; Taylor et al., 1996), such as enhanced spreading activation in the associative network which stores information about the relations between concepts and/or lexical items (Carter et al., 1992; Henik et al., 1998). This hypothesis implies that schizophrenia patients are able to adequately attend to the relevant dimension, but that the information contained in the irrelevant dimension is accessed more quickly or strongly, and thus has a greater influence over performance. The hypothesis that an increased influence of the word in schizophrenia is due to enhanced spreading activation is suggested by the findings of several recent studies on semantic priming in schizophrenia (e.g., Spitzer et al., 1994). In semantic priming paradigms such as word pronunciation (WP) and lexical decision (LD), participants are presented with two words, a prime and a target, usually in close succession. In WP participants pronounce the target word, whereas in LD they decide whether the target is a valid English word. RTs are consistently faster if the prime and target are semantically or associatively related than if they are not (e.g., Meyer and Schvaneveldt, 1971), an effect termed semantic priming. This priming effect is thought to result from at least two types of sources: (1) automatic spreading activation; and (2) the influence of extralexical processes, such as strategic expectations and higher level language processing. In the literature on normal language processing, manipulations of stimulus onset asyncrony (SOA; the difference in time onset between two consecutively presented stimuli) are one means by which researchers tease apart automatic and strategic components of priming. SOAs

shorter than 500 ms are thought to preclude the use and influence of trial-specific strategic processes. In contrast, SOAs longer than 500 ms are thought to allow time for participants to apply strategic processes (Neely, 1991).

A number of studies have now demonstrated increased semantic priming among schizophrenia patients, primarily at short stimulus onset asynchronies (SOAs) (Maher et al., 1987; Manschreck et al., 1988; Kwapil et al., 1990; Spitzer et al., 1993, 1994; Henik et al., 1995). These findings have been interpreted as providing evidence for enhanced spreading activation among schizophrenic patients, based on the assumption that spreading activation is the only process producing priming at short SOAs (e.g., 500 ms or less; Neely, 1991). This assumption stems from the research described above, suggesting that short SOAs preclude the use of trial-specific strategic processes that can also produce priming (Neely, 1991). Thus, findings of increased priming at short SOAs in schizophrenia patients are consistent with the hypothesis that increased RT Stroop facilitation and error interference in schizophrenia reflect enhanced spreading activation. None the less, one might wonder how enhanced spreading activation could influence Stroop performance deficits in schizophrenia, given that in the Stroop task, there is no ''prime'' and ''target'' between which activation can spread, but instead only a single stimulus with two dimensions (color and word). Spreading activation is thought to influence the processing of single words as well as the relationships between words. For example, many models of word reading posit that during the processing of a visually presented word, activation spreads among orthographic, phonological, and semantic representations (e.g., Plaut et al., 1996). If schizophrenic patients suffer from enhanced spreading activation, information about the word dimension of the Stroop stimulus (either phonological or semantic) should be accessed either more quickly or more strongly, and thus may have a greater impact on color naming.

Not all of the literature on semantic priming in schizophrenia, however, consistently reports enhanced priming at short SOAs. Several studies have either not found abnormalities at short SOAs (Chapin et al., 1989; Vinogradov et al., 1992; Chapin et al., 1992; Ober et al., 1995; Barch et al., 1996), or have found evidence of decreased priming at either short (Vinogradov et al., 1992; Henik et al., 1992;) or long (Barch et al., 1996) SOAs. It should also be noted that most of the studies reporting enhanced priming in schizophrenia have used LD and not WP. This may be due, in part, to the fact that priming effects in WP are typically smaller than those found in LD, although still consistent and reliable (Neely, 1991). However, LD appears to be more influenced than WP by strategic mechanisms, which may operate even at short SOAs (e.g., Neely, 1991). For example, semantic matching (a strategic process) is thought to play a role in LD, even at short SOAs, but not in WP (e.g., Seidenberg et al., 1984). Thus, using the LD paradigm, it is difficult to be sure that priming abnormalities, even at short SOAs, are attributable to disturbances in automatic spreading activation. The use of WP with short SOAs may provide a more selective measure of automatic spreading activation. None the less, even though the majority of studies have used LD, there is still considerable evidence for the spreading activation hypothesis, and further investigation of the relationship between semantic priming and Stroop abnormalities in schizophrenia is warranted.

The primary goal of the present study was to test the hypothesis that increased RT Stroop facilitation and increased error interference among schizophrenia patients reflects enhanced spreading activation. To do so, we used multiple measures which were differentially sensitive to spreading activation versus more strategic aspects of information processing. Specifically, we examined performance on two semantic priming tasks—WP and LD—at both a short SOA (300 ms) and a longer SOA (950 ms). We used LD to provide continuity with previous semantic priming studies of schizophrenia, the majority of which have used LD. We also used WP because of difficulties which arise in isolating the source of priming at short SOAs in LD, as described above. Thus, the use of WP in addition to LD provides converging evidence regarding the source of any priming changes found in LD. In the context of the current study, using WP has the additional advantage of using

the same verbal response modality as the Stroop task. We used two SOAs because, as discussed above, different processes are thought to operate during task performance at short versus long SOAs.

If enhanced spreading activation is the source of increased Stroop effects in schizophrenia, we would predict that compared to controls, patients should display: (1) enhanced Stroop RT facilitation and error interference; and (2) greater semantic priming at the 300 ms SOA in both the WP and LD paradigms. In contrast, if patients show enhanced Stroop RT facilitation and error interference, but do not show greater semantic priming at the 300 ms SOA, such a result would be inconsistent with the hypothesis that enhanced spreading activation is the mechanism leading to Stroop performance deficits in schizophrenia patients.

A secondary goal of this study was to evaluate the clinical significance of increased RT Stroop facilitation in schizophrenia. Carter et al. (1993) found that increased facilitation was limited to patients who met DSM-III-R criteria for the undifferentiated subtype. Patients with the paranoid subtype displayed a different pattern of Stroop performance: normal RT facilitation and increased RT interference. However, Carter et al. (1993) did not examine specific symptoms. Thus, it is not clear whether impaired Stroop performance is related to particular symptoms, or to more global subtype distinction. Liddle and Barnes (1990) have suggested that attentional impairment in schizophrenia might be specifically related to Disorganization symptoms (i.e., thought disorder, bizarre behavior). Therefore, in the current study we conducted an analysis of the relationship between Stroop facilitation and symptoms to examine this hypothesis.

2. Materials and methods

2.1. Participants

Participants were: (1) 56 DSM-IV schizophrenic or schizoaffective patients; and (2) 25 normal controls. All schizophrenic/schizoaffective patients were medicated inpatients at Mayview State Hospital who had been receiving the same medications and dosages for at least 2 weeks. Diagnoses for patients were based on a semi-structured interview for the Positive and Negative Symptom Scale (PANSS, Kay, 1991), a review of the participant's medical records, and consultation with the patient's treatment team. Normal controls were recruited through local advertisements and were evaluated using the Structured Clinical Interview for DSM-III-R. Controls were excluded if they had any lifetime history of Axis I psychiatric disorder other than simple phobia, or any first-order family history of psychotic disorders. Both patients and controls were excluded for: (1) substance abuse within the previous 6 months; (2) neurological illness or history of head trauma with loss of consciousness; (3) mental retardation (based on chart diagnoses); (4) English as a second language; (5) color blindness; or (6) poor visual acuity. Color blindness was tested by having participants name the color of patches that were the same color as the stimuli used in the Stroop experiment. Visual acuity was tested by having participants read words presented at the same visual angle that was used in the experiments described below.

The demographic and clinical characteristics of both participant groups are shown in Table 1. The control participants were matched with patients for age, gender, and years of parent education (to match approximately for socio-economic status) and did not differ significantly on any of these variables. Of the patients, four received a diagnosis of schizoaffective disorder, all of whom were actively psychotic at the time of participation. Of the 52 patients with schizophrenia, 28 received a subtype of paranoid, 21 of undifferentiated, 1 disorganized and 2 residual. Thirteen patients were taking risperidone, 10 haloperidol, 9 fluphenazine, 8 clozapine, 3 chlorpromazine, 4 thiothixine, 2 sertindole, and 1 each of the following: thioridazine, mesoridazine, trifluoperazine, and perphenazine. Daily oral doses of anti-psychotics for patients were converted to chlorpromazine equivalents according to guidelines suggested by Davis et al. (1983). Depot doses were converted to average daily dosages using the guidelines sug-

Table 1
Demographic and clinical characteristics of the groups

	Normal controls ($N=25$)		Schizophrenia patients ($N=56$)	
	Mean	SD	Mean	SD
Age	35.4	(5.3)	38.3	(8.8)
Sex (% male)	52		57	
Parent's education	12.9	(2.2)	12.3	(3.1)
Education	14.9	(2.2)	11.9	(1.6)
Length of current hospitalization (days)			411	(624)
Age of first hospitalization			21.6	(6.6)
Length of illness (years)			16.7	(6.9)
Chlorpromazine equivalents			1336	(1564)
% Taking antiparkinsonians			46	
% Taking antidepressants			13	
% Taking mood stabilizers			50	
% Taking benzodiazepines			30	
PANSS—Reality Distortion			10.4	(3.8)
PANSS—Poverty Symptoms			13.4	(6.0)
PANSS—Disorganization			11.1	(4.4)

gested by Baldessarini (1985). All participants signed informed consent forms in accordance with the university and Mayview State Hospital institutional review boards. All participants were paid for their participation.

The PANSS (Kay, 1991) was used to evaluate clinical state. Ratings were completed by one of two Ph.D.-level clinical psychologists. A subset of 17 patients were rated by both psychologists. Because we did not have the power to examine each individual symptom, symptoms were grouped into three factors. We used the three factors suggested by Liddle (1987))—Reality Distortion, Poverty Symptoms, and Disorganization. Based on a review of studies examining the dimensional structure of the PANSS, the following items were chosen for each scale: (1) delusions, hallucinations, and unusual thought content for Reality Distortion (alpha = 0.76); (2) blunted affect, emotional withdrawal, passive social avoidance, motor retardation, and lack of spontaneity for Poverty (alpha = 0.86); and (3) conceptual disorganization, mannerisms and posturing, difficulty in abstract thinking, and poor attention for Disorganization (alpha = 0.77). Interrater reliability, measured using intraclass correlations (Shrout and Fleiss, 1979) with raters treated as random effects and the individual rater as the unit of reliability, was 0.95 for Reality Distortion, 0.95 for Poverty Symptoms, and 0.94 for Disorganization.

2.2. Materials

2.2.1. Stroop task

The stimuli were identical to those used by Carter et al. (1992) and consisted of 96 trials: 24 (25%) congruent trials; 24 (25%) incongruent trials; and 48 (50%) neutral trials. Each trial consisted of a word printed in one of four colors: red, blue, green, or purple. The congruent stimuli consisted of one of the four color names presented in its own color. The incongruent stimuli consisted of each of the four color names presented in one of the three remaining colors. Neutral stimuli were one of four color-unrelated words (dog, bear, tiger, or monkey) printed in one of the four colors. The neutral words matched the four color words in length and frequency and were from a single semantic category to eliminate semantic confounds.

2.2.2. Semantic priming tasks

Semantic priming was assessed using both a WP and a LD paradigm. For both tasks, prime type (related, unrelated) and SOA (300 and 950 ms) were within-subject factors. A subset of

160 target words were taken from the 200 words used in our previous study of semantic priming in schizophrenia (Barch et al., 1996). For each of these target words, a related and an unrelated prime had been constructed from lists of published norms. The list of 160 target words was randomly divided into two sets of 80 target words. Each participant received one set of 80 stimuli in the WP task and the other set of 80 stimuli in the LD task. The word set used for each task was counterbalanced across participants. For each participant, a target was presented in only one condition (related, unrelated prime) and one SOA, and each target was used only once for a given participant. Condition of presentation and SOA for each target was counterbalanced so that within every four participants, a target appeared once in each condition (related, unrelated prime) at both SOAs. For the WP task, every participant was presented with 80 prime–target pairs, which included 40 related pairs and 40 unrelated pairs, 20 of each at the two different SOAs. For each participant, SOAs were randomly intermixed across trials, with the constraint that 20 related and 20 unrelated pairs would be presented at each SOA and that all conditions were sampled once in every eight trials. For the LD task, every participant was presented with 80 prime–word target pairs (40 related, 40 unrelated) and 80 prime–nonword targets. The nonwords had been constructed by switching one letter in a real word, which maintained pronounceability. Each participant was presented with 20 related, 20 unrelated, and 40 nonword targets at each of the two SOAs. As with the WP task, SOAs were randomly intermixed across trials, with the constraint that all conditions would be sampled once in every 12 trials.

2.3. Procedure

Each participant was tested individually. Order of task presentation was counterbalanced across participants. Stimuli for all tasks were presented on an Apple Macintosh computer and color monitor (with a phosphor persistence of 300 μs), using PsyScope software (Cohen et al., 1993). For the priming tasks, each word was centered in a fixation box measuring approximately 2 cm × 1 cm and was presented in non-degraded, lowercase, Helvetica font, white against a black background. For the Stroop task, each word was centered in the screen, and was presented in non-degraded, uppercase, Helvetica font, against a black background. All stimuli subtended a visual angle of approximately 2–3°, which was maintained across participants through the use of chin rest fixed in place. RTs for onset of word articulation in the WP and Stroop tasks were automatically recorded by the computer using a microphone and a voice-activated relay. RTs for LD (word, nonword) were automatically recorded by the computer via a custom-made button box. For each of the tasks, a short practice period preceded the actual testing to ensure that participants understood the instructions, were comfortable with the apparatus, and were performing the task appropriately.

2.3.1. Stroop

Participants were told that they would be presented with a series of words, one at a time. Their job was to read the color in which the word was printed, as quickly and accurately as possible. Each word remained on the screen until the participant responded, or until 2000 ms elapsed, and then was replaced by a fixation cross that lasted until the onset of the next stimulus. Regardless of RT, a new trial started 4 s after onset of the previous stimulus, so that the pace of the task was fixed for all participants. Participants' verbal responses were tape-recorded for later coding of accuracy.

2.3.2. Semantic priming tasks

Participants were told that they would be presented with pairs of words. Their job was to read the first word silently. For WP, participants were told to say the second word aloud, as fast as they could. For LD, participants were told to decide whether the second word was a real word or nonword as quickly as possible by pushing one of two buttons on the button box. Participants used their dominant hand and responded with adjacent fingers (one for each button) on the same hand. The prime appeared for 100 ms and then the screen went blank (without masking) for either 200 or 850 ms, depending on SOA condition. The target word was then presented and participants had a

total of 2 s from its onset in which to respond. Following either the participant's response or 2 s, the screen went blank. Regardless of RT, a new trial started 4 s after onset of the previous target, so that the pace of the task was fixed for all participants.

2.4. Data analysis

Medians for correct responses were used in analyses examining RTs. For the Stroop and LD, faster RTs were associated with more accurate performance, indicating an absence of speed–accuracy tradeoffs. Error rates were not examined for WP because they were less than 1% for all groups. Data were subjected to repeated-measures analyses of variance (ANOVAs), as described below. Where appropriate, Greenhouse–Geisser corrections for degrees of freedom were applied. Planned comparisons were used to follow-up on main effects and interactions predicted by specific hypotheses. As noted above, normal controls and schizophrenia patients were matched on age, gender, and father's education. However, participant education differed across groups. Multiple regression analyses indicated that none of the demographic variables, including participant education, accounted for a significant amount of variance in any of the experimental measures. In addition, regression analyses also indicated that medication dosage (in chlorpromazine equivalents) was not significantly associated with any of the experimental measures.

3. Results

We began by examining group differences between the controls and the schizophrenia patients. We examined RTs on the Stroop task (Table 2) using a two-way ANOVA, with group as the between-subjects factor and condition (congruent, neutral, incongruent) as the within-subjects factor. This ANOVA revealed main effects of group [$F(1,79)=38.90$, $p<0.001$] and condition [$F(2,158)=117.98$, $p<0.001$]. Controls were faster than schizophrenia patients, and both groups responded faster to congruent stimuli than to neutral stimuli [$F(1,79)=60.84$, $p<0.001$], and more slowly to incongruent stimuli than to neutral stimuli [$F(1,79)=81.32$, $p<0.001$]. There was also a significant interaction between group and condition [$F(2,158)=3.08$, $p<0.05$]. Planned comparisons indicated that schizophrenia patients displayed significantly more facilitation (neutral RT–congruent RT) than controls [$F(1,79)=8.27$, $p<0.005$], but did not differ in interference (incongruent RT–neutral RT) [$F(1,79)=0.96$, $p>0.30$].[1] The magnitude of RT facilitation and interference for patients and controls is displayed in Fig. 1.

A similar ANOVA examining accuracy revealed main effects of group [$F(1,79)=11.33$, $p<0.001$] and condition [$F(2,158)=43.40$, $p<0.001$], and a group × condition interaction [$F(2,158)=12.32$, $p<0.001$]. Schizophrenia patients were less accurate than controls, and both groups responded less accurately to neutral stimuli than to congruent stimuli [$F(1,79)=8.26$, $p<0.01$], and to incongruent stimuli than to neutral stimuli [$F(1,79)=63.42$, $p<0.001$]. Planned comparisons indicated that, compared to controls, schizophrenia patients displayed larger decreases in accuracy from neutral to incongruent stimuli [$F(1,79)=22.53$, $p<0.001$]. In other words, compared to controls, schizophrenia patients displayed more interference as measured by errors.

We next examined whether schizophrenia patients displayed more priming than controls at the short SOA in the priming tasks, as predicted by the spreading activation hypothesis. To do so, we analyzed the short SOA RT data (Table 3) from both of the priming tasks using a three-way ANOVA, with diagnostic group as the between-subjects factor, and task (WP, LD) and prime type (related, unrelated) as within-subjects factors. This ANOVA revealed main effects of group [$F(1,63)=46.69$, $p<0.001$], and task [$F(1,63)=33.78$, $p<0.01$], and a group × task interaction [$F(1,63)=7.51$, $p<0.01$]. RTs were slower in the

[1] We also examined the source of this interaction by examining the conditional (residual) effects as suggested by Rosenthal and Rosnow (1985). This produced similar results, suggesting that schizophrenia patients displayed more RT facilitation (+31) than would be predicted by the group and condition main effects, but no significant differences in RT interference (−11.5).

Table 2
Means and standard deviations for Stroop task for each of the groups

	Normal controls ($N=25$)		Schizophrenia patients ($N=56$)	
	Mean	SD	Mean	SD
RT (ms)				
Congruent	638	(97)	834	(178)
Neutral	692	(88)	950	(178)
Incongruent	812	(117)	1047	(199)
Errors				
Congruent	0.005	(0.02)	0.013	(0.03)
Neutral	0.013	(0.02)	0.023	(0.04)
Incongruent	0.051	(0.08)	0.155	(0.13)

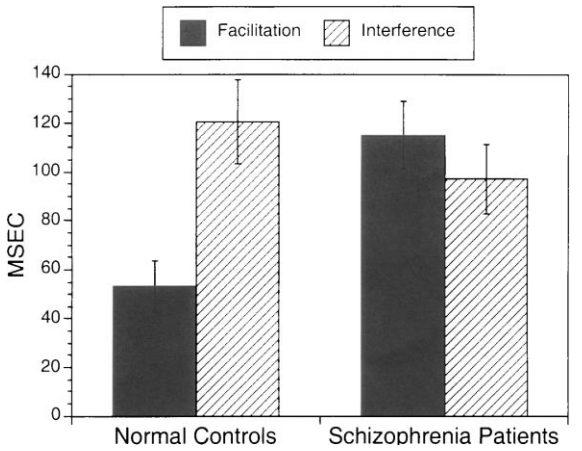

Fig. 1. Stroop reaction time facilitation and interference as a function of group.

LD than WP task for both groups, and schizophrenia patients were slower than normal controls in both the WP [$F(1,63)=24.48$, $p<0.001$] and the LD task [$F(1,63)=39.09$, $p<0.001$]. The group × task interaction reflected the fact that the increase in RTs from the WP to LD task was greater in patients than controls [$F(1,63)=7.51$, $p<0.01$]. The ANOVA also revealed a main effect of prime type [$F(1,63)=6.87$, $p<0.01$], suggesting that RTs to related primes were faster than RTs to unrelated primes at the short SOA in both tasks, among both groups. To confirm this, we conducted one-tailed paired t-tests which indicated that controls displayed significant priming at the short SOA in both WP [$t(24)=2.10$, $p<0.05$] and LD [$t(24)=3.55$, $p<0.01$]. Among patients, the priming effect was not significant for WP [$t(39)=0.28$, $p>0.30$] but was for LD [$t(39)=1.9$, $p<0.05$]. However, the group × prime type interaction [$F(1,63)=0.00$, $p>0.90$] and the group × task × prime type interactions [$F(1,63)=0.05$, $p>0.80$] were not significant.[2]

Next, we examined performance at the long SOA in the priming tasks. To do so, we used another three-way ANOVA to analyze the RT data from the long SOAs. This ANOVA revealed significant main effects of group [$F(1,63)=44.58$, $p<0.001$], and task [$F(1,63)=44.20$, $p<0.01$] and a marginal main effect of prime type [$F(1,63)=2.90$, $p=0.09$]. Schizophrenia patients were again slower than controls, and RTs were slower in LD compared to WP. Further, there was a marginal group × prime type [$F(1,63)=2.99$, $p=0.088$] and group × task [$F(1,63)=3.47$, $p=0.067$] interaction. As can be seen in Table 3, the marginal group × task interaction indicated that although RTs were slower in LD than WP among both controls [$F(1,63)=9.30$, $p<0.01$] and schizophrenia patients [$F(1,63)=47.09$, $p<0.001$], there was a trend for schizophrenia patients to display a greater slowing of RTs between the WP and LD

[2] Because of previous studies suggesting enhanced priming at short SOAs only among thought disordered schizophrenia participants (e.g., Spitzer et al., 1993), we also conducted this analysis with schizophrenia patients divided into nonthought disordered and thought disordered using the same criteria as Spitzer. The results of this analysis were identical, with neither nonthought disordered nor thought disordered patients displaying increased priming compared to controls at the short SOA in either WP or LD.

Table 3
Means and standard deviations for priming tasks

	Normal controls ($N=25$)		Schizophrenia patients ($N=56$)	
	Mean	SD	Mean	SD
Word pronunciation				
Short SOA				
Related RT	574	(79)	737	(153)
Unrelated RT	583	(83)	743	(148)
RT priming	8.8	(21.2)	5.6	(73.4)
Long SOA				
Related RT	549	(75)	727	(160)
Unrelated RT	558	(82)	719	(152)
RT priming	9.2	(29.7)	−7.6	(56.6)
Lexical decision				
Short SOA				
Related RT	630	(111)	912	(200)
Unrelated RT	658	(112)	943	(221)
RT priming	28.5	(39.8)	30.8	(102.6)
Related errors	0.017	(0.03)	0.032	(0.07)
Unrelated errors	0.008	(0.02)	0.046	(0.06)
Long SOA				
Related RT	630	(105)	896	(201)
Unrelated RT	668	(116)	904	(190)
RT priming	37.3	(54.1)	7.3	(111.6)
Related errors	0.005	(0.02)	0.034	(0.06)
Unrelated errors	0.024	(0.03)	0.032	(0.04)

tasks than controls. Planned contrasts to explore the group × prime type interaction indicated that the main effect of prime type was significant among controls [$F(1,63)=4.79$, $p<0.05$], but not among patients [$F(1,63)=0.00$, $p>0.9$]. Further, there was a trend for controls to display more priming overall than the schizophrenia patients [$F(1,63)=2.99$, $p=0.09$]. To further clarify these results, we conducted one-tailed paired t-tests which indicated that controls displayed significant priming at the long SOA in both LD [$t(24)=3.30$, $p<0.001$] and marginally significant priming at the long SOA in WP [$t(24)=1.50$, $p=0.08$]. Among patients, the priming effect was not significant for LD [$t(39)=0.41$, $p>0.60$] or for WP [$t(39)=0.85$, $p>0.30$].

We also examined accuracy in the LD task, using a three-way ANOVA with group as the between-subjects factor, and prime type and SOA as within-subjects factors. As noted above, error rates were not examined for WP because they were less than 1% for all groups. This ANOVA revealed a main effect of group [$F(1,63)=7.10$, $p<0.01$], and a group × prime type × SOA interaction [$F(1,63)=5.35$, $p<0.05$]. Schizophrenia patients were less accurate than controls, but only with unrelated targets at the short SOA [$F(1,63)=9.50$, $p<0.01$], and with related targets at the long SOA [$F(1,63)=6.05$, $p<0.05$].

As discussed in the Introduction, a secondary goal of this study was to examine the clinical significance of increased Stroop facilitation in schizophrenia. In particular, our goal was to determine whether increased Stroop facilitation was related to particular symptoms that differed between undifferentiated patients and paranoid patients. Consistent with previous research (Carter et al., 1993), independent sample t-tests indicated that undifferentiated patients had significantly more Poverty [$t(47)=2.57$, $p<0.05$] and Disorganization symptoms [$t(47)=1.99$, $p<0.05$] than paranoid patients. The two subtypes did not differ significantly in Reality Distortion symptoms [$t(47)=0.87$, $p>0.10$]. Thus, we examined the association between Stroop performance and both

Poverty Symptoms and Disorganization. Poverty Symptoms were not associated with facilitation or interference in either RT or errors (average $r = -0.05$, *range* $= -0.18$ to 0.02). Disorganization was not significantly associated with facilitation in either RT ($r = 0.14$, $p > 0.10$) or errors ($r = 0.21$, $p > 0.10$), but displayed a significant negative correlation with RT interference ($r = -0.27$, $p < 0.05$) and a significant positive correlation with error interference ($r = 0.36$, $p < 0.01$). To determine whether Disorganization was significantly more strongly correlated with either RT or error interference as compared to either Poverty Symptoms or Reality Distortion, we utilized methods for comparing correlated correlation coefficients suggested by Meng et al. (1992)). These analyses suggested that among schizophrenia patients, Disorganization was significantly more strongly correlated with error interference compared to either Poverty Symptoms ($Z = -2.03$, $p < 0.05$) or Reality Distortion ($Z = -1.71$, $p < 0.05$). However, the correlation between Disorganization and RT interference did not differ significantly from the correlations between RT interference and either Poverty Symptoms or Reality Distortion.

Studies with schizophrenia patients are often confounded by the effects of longer RTs, because difference scores can be spuriously inflated in participants who exhibit overall worse or more variable performance (Chapman et al., 1994). This issue is relevant for our study given that schizophrenia patients displayed larger facilitation scores than controls, but also had longer RTs. Thus, it is possible that the larger facilitation scores simply reflect an artifact of longer RTs among schizophrenia patients. Chapman et al. (1994) have suggested an approach to examining this issue, through computing the regression equation that predicts difference scores (e.g., neutral RT –congruent RT) from a measure of overall RT, using only the data from the control participants, and then determining whether the schizophrenia patients fall on this same regression line. However, the distribution of overall RT scores among our controls does not fully overlap with the RT distribution patients. Thus, such an analysis could be criticized on the basis that one cannot make predictions about values that fall outside the range of values used to generate the original regression equation (Chapman et al., 1994). Therefore, we used two alternative approaches to address this issue. First, we used as the measure of RT in each condition (for each participant), the normal mean-deviate (i.e., *Z*-score) of the mean RT across all conditions for that participant. The logic behind this analysis is that the SD across the conditions for patients should be larger than the SD for controls. *Z*-Scores are calculated as a function of the magnitude of the SD. Thus, if the magnitude of facilitation scores among schizophrenia patients is simply proportional to their overall longer RTs, an analysis using normal mean-deviates should show no group differences in the magnitude of facilitation. We conducted this analysis with our data, and the results of the ANOVA using *Z*-scores again indicated a significant two-way interaction between group and condition [$F(2,158) = 4.08$, $p < 0.05$]. Second, we also compared subgroups of controls and schizophrenia patients ($N = 16$ each) who did not differ significantly on average RT (766 ms for controls, 744 ms for patients). The ANOVA using this subset of participants for Stroop RTs continued to show a significant group × condition interaction [$F(2,60) = 3.80$, $p < 0.05$]. These results suggest that increased Stroop facilitation among schizophrenia patients is not simply an artifact of their slower RTs.

4. Discussion

The primary purpose of the present research was to test the hypothesis that increased RT Stroop facilitation and increased error interference among schizophrenia patients reflect enhanced spreading activation. Consistent with previous research, we found that schizophrenia patients showed increased RT facilitation on the single trial Stroop task compared to normal controls (Carter et al., 1992, 1993; Taylor et al., 1996; Henik et al., 1998; Cohen et al., in press), as well as increased error interference (Taylor et al., 1996; Henik et al., 1998; Cohen et al., in press). However, these same patients did not show evidence of increased semantic priming. Such results are not consistent with the hypothesis that increased Stroop facilitation

reflects an effect of increased automatic activation of semantic, orthographic, or phonological information. As discussed in the Introduction, priming at SOAs less than 500 ms, at least in WP, is thought to be influenced only by spreading activation, and thus should provide the clearest test of abnormal spreading activation among schizophrenia patients. Further, this is the second study in which we have failed to observe evidence for increased "automatic" semantic priming effects in schizophrenia (Barch et al., 1996). This leads us to conclude that it is doubtful that schizophrenia is associated with enhanced spreading activation, or that disturbed spreading activation is the mechanism leading to enhanced Stroop facilitation in this group.

None the less, one might argue that schizophrenia patients do suffer from enhanced spreading activation, but that this effect was not apparent at the short SOA because of potential backward masking effects. It is well documented that schizophrenia patients are more impaired than healthy controls at detecting a target when it is followed in close succession by a masking stimulus. This effect, with targets as single letter or letter pairs, has been found up to 100 ms ISIs (Green and Walker, 1984). In addition, enhanced backward masking effects have also been found among schizophrenia patients at longer ISIs (e.g., 400 ms) with more complex stimuli such as pictures (Knight et al., 1985). Thus, it is possible that at the short SOA (300 ms) in our priming paradigms, the target served to interrupt processing of the prime, and impede spreading activation. However, if this hypothesis were correct (i.e., schizophrenia patients do suffer from enhanced spreading activation, but this is "masked" as short SOAs because of backward masking effects), then one would expect priming to increase among schizophrenia patients as the SOA increases, which would allow more time to process the prime before the target appears. However, contrary to this hypothesis, priming among schizophrenia patients decreased, rather than increased, at the longer SOA, while controls showed the opposite pattern. In fact, schizophrenia patients did not display a significant priming effect at the long SOA in either WP or LD, and there was a trend for patients to display significantly less priming than controls in these conditions. Thus, is seems unlikely that backward masking effects could explain the complete absence of any evidence for enhanced priming among schizophrenia patients at either the short or long SOAs in either task

The failure of schizophrenia patients to show significant priming at the longer SOA is consistent with several different hypotheses. First, as noted in the Introduction, priming at SOAs longer than 500 ms is thought to be influenced by strategic mechanisms, such as expectancy, that involve detecting and using relevant information about the stimuli (e.g., semantic relationships between primes and targets). In previous work, we have hypothesized that decreased priming at long SOAs among schizophrenia patients may reflect a disturbance in such mechanisms (Barch et al., 1996). However, although decreased long SOA priming in schizophrenia patients is consistent with a deficit in the strategic allocation of attention, such a result could also be explained by alternative hypotheses, such as poor encoding into short-term visual memory or the rapid decay of short-term visual memory. Thus, more work is needed to clarify the mechanisms underlying decreased priming at longer SOAs among schizophrenia patients.

If enhanced spreading activation is not the mechanism underlying altered Stroop performance in schizophrenia, what alternative mechanism might be responsible for such cognitive deficits? One hypothesis is that this pattern of Stroop performance shown by schizophrenia patients reflects a disturbance in the strategic control of selective attention, which leads to a failure in modulating the relative influence of the relevant (color) dimension of the stimulus over the irrelevant (word) dimension of the stimulus (e.g., Callaway and Naghdi, 1982; Cohen and Servan-Schreiber, 1992). On face value it would seem that an increased influence of word reading should manifest as increases in *both* Stroop interference and facilitation in the RT analysis. In the congruent condition, the word should speed response times, resulting in greater facilitation. In the incongruent condition, the words should slow response times and increase interference. However, two factors may contribute to the absence of RT

interference in schizophrenia patients. First, facilitation and interference scores are expressed as differences from the neutral condition. Thus, how patients perform in the neutral condition may strongly influence the overall pattern of performance with respect to these difference scores (Barch et al., in press). If the irrelevant dimension is influencing color naming more in schizophrenia, patients may experience some degree of "interference" from the neutral stimulus, producing more slowing in this condition. Slower neutral RTs would contribute to an increased RT facilitation effect, but would contribute to no change, or even a decrease, in RT interference effects. This hypothesis is consistent with our finding that the significant group × condition effect for Stroop RTs was due primarily to group differences in the relationship between the congruent and neutral conditions, and not in the relationship between the neutral and incongruent conditions.

A second factor that may contribute to the absence of increased RT interference in schizophrenia patients is their pattern of accuracy performance (Barch et al., in press). Although increased facilitation, and not interference, was observed among schizophrenia patients in the RT analysis, increased interference was found in the error analysis. The increase in errors in the incongruent condition exhibited by patients may also contribute to an absence of RT interference (in conjunction with the slowing in the neutral condition). Slowing in the incongruent condition is thought to occur when the influence of the word interferes with the processing of the print color. Schizophrenia patients may be less able to inhibit the influence of the word, and therefore more likely to actually respond to the word instead of the color. This may occur in the incongruent condition (but not in the congruent and neutral conditions) because the incongruent condition contains the greatest amount of conflicting word information. This pattern of results (increased interference in errors, but not RT) has now been found in several studies (Taylor et al., 1996; Henik et al., 1998; Cohen et al., in press), and clearly warrants further theoretical and empirical investigation.

Our analysis of the relationship to specific symptoms suggests that Disorganization, which was more severe among undifferentiated patients, may be most strongly related to the cognitive dysfunction tapped by the Stroop task. This result is consistent with previous assertions that Disorganization is related to attentional impairment in schizophrenia (Liddle and Morris, 1991). However, the pattern of correlations was somewhat unusual, in that Disorganization was associated with decreased RT interference and increased error interference, but was not directly associated with facilitation. Although unexpected, this pattern of correlations does make sense when considered in relation to an analysis of the cognitive mechanisms underlying disturbed Stroop performance in schizophrenia, as described above. In other words, Disorganization symptoms may be related to increased error interference rather than increased RT interference, because Stroop deficits among patients with schizophrenia are expressed as increased error interference (and increased RT facilitation) rather than increased RT interference.

The present study was conducted with only medicated patients. However, it is unlikely that medication effects account for the pattern of results we obtained (e.g., increased Stroop RT facilitation, increased error interference, decreased long SOA priming, and the absence of increased priming at short SOAs). First, increased Stroop RT facilitation has previously been reported in unmedicated patients (Carter et al., 1992, 1993; Henik et al., 1998). Second, studies that have reported increased automatic semantic priming effects have included only medicated participants (e.g., Vinogradov et al., 1992; Spitzer et al., 1994), whereas Barch et al. (1996) have reported that unmedicated patients fail to show increased priming at short SOAs. Third, Barch et al. (1996) found evidence for decreased priming at a long SOA in both medicated and unmedicated patients.

In summary, the results of the present study suggest that a disturbance in spreading activation is not the mechanism underlying increased Stroop facilitation in schizophrenia patients. As an alternative hypothesis, we have proposed that abnormal Stroop performance among schizophrenia patients may reflect a disturbance in the strategic control of attention, leading to deficits in the ability to attend to the task relevant dimensions

of Stroop stimuli. More specifically, we have proposed that a deficit in the ability to attend to task relevant dimensions of the stimulus in schizophrenia could influence performance in all conditions of the Stroop task (e.g., congruent and neutral, as well as incongruent), a prediction that we explore in more detail in related work (Barch et al., in press).

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