Context-Processing Deficits in Schizophrenia: Converging Evidence From Three Theoretically Motivated Cognitive Tasks

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To test the hypothesis that the ability to actively represent and maintain context information is a central function of working memory and that a disturbance in this function contributes to cognitive deficits in schizophrenia, the authors modified 3 tasks—the AX version of the Continuous Performance Test, Stroop, and a lexical disambiguation task—and administered them to patients with schizophrenia as well as to depressed and healthy controls. The results suggest an accentuation of deficits in patients with schizophrenia in context-sensitive conditions and cross-task correlations of performance in these conditions. However, the results do not definitively eliminate the possibility of a generalized deficit. The significance of these findings is discussed with regard to the specificity of deficits in schizophrenia and the hypothesis concerning the neural and cognitive mechanisms that underlie these deficits.

The effort to identify and characterize cognitive deficits that are specific to schizophrenia has been a long and challenging enterprise. Shakow (1962) was among the first to make use of modern methods in this endeavor, including structured laboratory tasks and the formulation of observations in information-processing terms. Based on the results of reaction time studies, Shakow (1962) observed that

we see particularly the various difficulties created by context. . . . It is as if, in the scanning process which takes place before the response to a stimulus is made, the schizophrenic is unable to select out the material relevant for optimal response. (p. 4)

Shakow's work inspired a large number of studies over the ensuing 2 decades, which generated several new theories concerning information-processing deficits in schizophrenia (e.g., Braff & Saccuzzo, 1981; Oltmanns & Neale, 1975) and produced a number of important laboratory measures that continue to be applied in studies of populations with vulnerability for schizophrenia (e.g., first-degree family members and children at risk; for a review, see Cornblatt & Keilp, 1994).

In our own work, we have drawn on computational modeling techniques to characterize the specific processing mechanisms involved in such cognitive tasks and on controlled experimental tasks to empirically test predictions generated by these simulation studies. This work has led to the hypothesis that an important subset of cognitive deficits in schizophrenia can be understood in terms of a disturbance in a single underlying mechanism, one that is responsible for the representation and maintenance of context information needed to select task-appropriate action. This hypothesis is closely related to Shakow's original proposal that patients with schizophrenia suffer from an impairment in the ability to use context. However, through the use of computational models, we have been able to make explicit the mechanisms involved in the processing of context (Cohen, Braver, & O'Reilly, 1996; Cohen & Servan-Schreiber, 1992). These models can be used to simulate performance in cognitive tasks and to make predictions about the specific patterns of deficits that should result from a context-processing disturbance. The primary aim of the present study was to test a set of such predictions empirically. As background, it will be useful to specify what we mean by context processing and how we believe this is disturbed in schizophrenia.

Context Processing

By context information, we refer to information that must be actively held in mind in such a form that it can be used to mediate task-appropriate behavior. In our models, representations of context are used to support task-relevant information against sources of interference (e.g., noise or competing processes). Context representations can be composed of different types of information, such as specific prior stimuli, the result of processing a sequence of prior stimuli, or more abstract information such as task instructions. This definition may seem overly inclusive. However, we have argued that context can be distinguished from related con-
cept in useful and important ways. For example, we have distin-
guished it from the construct of short-term memory (Cohen & Servan-Schreiber, 1992). The latter typically refers to processes involved in the temporary storage of recently presented information, which may or may not have relevance for later behavior. Conversely, by our definition, context representations may or may not correspond to the identity of previously presented information but always have relevance for later behavior. A recent neurophys-
ological study by Miller et al. supports such a distinction (Miller, Erickson, & Desimone, 1996). They observed sustained, stimulus-
specific activity in regions posterior to prefrontal cortex, but this occurred to cue stimuli and distractors alike and was disrupted by the presentation of each new stimulus. In contrast, they observed sustained activity in prefrontal cortical units that was specific to a cue to which a monkey had to respond. This pattern of activity survived the presentation of intervening distractors and persisted until a target stimulus appeared to which the monkey had to respond. These findings are consistent with our theory that the active maintenance of context information is subserved by mechanisms housed within prefrontal cortex and is distinguishable from other forms of activity-based, short-term storage. We also assume that context processing is one component of working memory, which is commonly defined as the collection of processes responsible for on-line maintenance and manipulation of information necessary to perform a cognitive task (Baddeley & Hitch, 1994). We view context representations as a subset of representations within working memory, which govern how other representations are used (Cohen et al., 1996). In this respect, they bear a close relationship to goal representations in production system models of cognition (e.g., Anderson, 1983).

One important insight that has emerged from this work is that three cognitive functions that are often treated as independent—attention (selection and support of task-relevant information for processing), active memory (on-line maintenance of such information), and inhibition (suppression of task-irrelevant information)—can all be understood in terms of a single mechanism responsible for the processing of context, operating under different task conditions. When a task involves competing, task-irrelevant processes (as in the Stroop task), it is often assumed that a dedicated inhibitory function is responsible for suppressing, or overriding, these irrelevant processes. However, in our models, there is no dedicated mechanism for inhibition. Rather, context representations accomplish the same effect by providing top-down support for task-relevant processes, allowing these to compete effectively against irrelevant ones. In contrast, when a task involves a delay between a cue and a later contingent response, it is usually assumed that a memory function is involved. Once again, there is no dedicated mechanism for this function in our models. Rather, the mechanism used to represent context information is used to maintain task relevant information against the interfering and cumulative effects of noise over time. Thus, both for tasks that tap inhibition and for those that tap memory, the same mechanism is involved; it is simply a matter of the behavioral conditions under which it operates (i.e., the source of interference) that lead us to label it as having an inhibitory or a memory function. Furthermore, under both types of conditions, context representations serve an attentional function, by selecting task-relevant information for processing over other potentially competing sources of informa-
tion. 2 In all circumstances, the same mechanism is involved. This perspective has led us to propose that many of the deficits observed in patients with schizophrenia in a variety of cognitive tasks can be explained in terms of a disturbance in a single underlying mechanism.

Context Processing in Schizophrenia

In Cohen and Servan-Schreiber (1992), we demonstrated that degrading the processing of context in our models produced quantitatively accurate simulations of data regarding the performance of patients with schizophrenia in three tasks that are commonly considered to tap different domains of cognitive function: the Stroop task (selective attention), the identical pairs version of the continuous performance test (signal detection and vigilance), and a lexical disambiguation task (language processing). Furthermore, we used these to identify two dimensions along which tasks could be modified, to permit a more direct and specific test of our hypothesis concerning a disturbance in the processing of context: (a) the relative strength of competing responses and (b) the delay between a contextual cue and the contingent response to a probe. Whenever dominant but task-inappropiate responses are possible, context becomes important for mediating the weaker but task-appropriate response. Thus, a task can be made sensitive to disturbances in context processing by increasing the strength of task-irrelevant responses. Tasks can also be made sensitive to the maintenance of context information, independent of response-strength asymmetries, by increasing the delay between context and response. A number of tasks commonly used in schizophrenia studies manipulate one or the other of these factors. However, to our knowledge, none have manipulated both simultaneously. For example, the Stroop task involves a strong response strength asymmetry but no manipulation of delay between context and response. Conversely, two versions of the continuous performance test (CPT; Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956) in common use (identical pairs [Correll & Keilp, 1994] and AX [Nuechterlein, 1991]) involve a delay between the context-providing cue (prior stimulus) and the one to be responded to. However, these tasks do not involve any response-strength asymmetry.

In previous work (Servan-Schreiber, Cohen, & Steingard, 1997) we used the AX version of the CPT to test this hypothesis. By introducing a response strength asymmetry and manipulating the delay between cue and probe, we corroborated our prediction that patients with schizophrenia would show a selective deficit when the appropriate response was context-dependent (i.e., competing against a stronger, acontexual response) and there was a delay between context and response (requiring maintenance of context over time). A primary goal of the current study was to test the

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1 We should note that this competition does involve inhibition; however, this occurs "locally" (i.e., directly between the specific processes involved in the task), rather than originating from a central source, as is assumed in many theories. This is consistent with recent theories concerning the neurobiological substrates of selective attention (see Desimone & Duncan, 1995).

2 In the context of an inhibitory task we might refer to this as selective attention, whereas we might refer to it as vigilance in the context of a memory task.
The primary goal of the present study was to test our hypothesis that a disturbance in the processing of context can account for performance deficits among patients with schizophrenia in a variety of seemingly disparate task domains. Accordingly, we predicted not only that participants with schizophrenia should show a specific pattern of deficits in each task but that these deficits should correlate across tasks. Establishing such cross-task correlations would be an important result, given historical difficulties finding such correlations among tasks that individually elicit performance deficits in schizophrenia (e.g., Asarnow & MacCrimmon, 1981; Kopfstein & Neale, 1972). In addition, we sought to test the hypothesis that a disturbance in the processing of context would be associated most strongly with the disorganization dimension of positive symptoms.

Method

Participants

Participants were 26 inpatients and 27 outpatients with schizophrenia or schizoaffective illness, who were all receiving neuroleptics; 25 patient controls with nonpsychotic major depression; and 31 healthy controls. Inpatients were housed either on the Schizophrenia Unit or Mood Disorders Unit of the Western Psychiatric Institute and Clinic (WPIC). Outpatients were recruited from the Schizophrenia Treatment and Research Center at WPIC. Patients were included if they received a diagnosis of schizophrenia or schizoaffective disorder from a staff psychiatrist and if the diagnosis was confirmed by trained research personnel using the Structured Clinical Interview for DSM-III-R (Diagnostic and Statistical Manual of Mental Disorders, 3rd ed., rev.; American Psychiatric Association, 1987)—Psychotic Disorders (SCID-PD). Spitzer, Williams, Gibbon, & First, 1990) and a thorough chart review. All interviewers participated in ongoing biweekly diagnostic reliability and training sessions. Healthy controls were recruited through local advertisements and were also evaluated using the SCID. Controls were excluded if they had any lifetime history of Axis I disorder or any first-order family history of a psychotic disorder. The healthy controls were matched with patients for age, gender, race, and parental education (to match approximately for socioeconomic status). Potential participants were excluded for (a) substance abuse within the prior 6 months, (b) neurological illness or history of head trauma with loss of consciousness, (c) mental retardation, or (d) non-English native language.

The Brief Psychiatric Rating Scale (BPRS; Overall, 1974) was used to evaluate clinical state. Ratings were completed by trained research team members who regularly participated in evaluation sessions to ensure reliability. All BPRS ratings were made within 1 week of admission to the study, and all raters were not informed of the performance of participants in the tasks. To test hypotheses regarding the clinical relevance of context processing deficits (described in detail below), we grouped symptoms into the following syndrome subscales (as suggested by Liddle, 1987) based on a review of the BPRS factor analysis literature (e.g., Brekke, DeBonis, & Graham, 1994): (a) disorganization (conceptual disorganization and mannerisms and posturing); (b) reality distortion (hallucinations, delusions); and (c) poverty symptoms (blunted affect, emotional withdrawal, motor retardation). Interrater reliability was measured using intraclass correlations with raters treated as random effects and the individual rater as the unit of reliability (Shrout & Fleiss, 1979). Interrater reliability was .74 for disorganization, .99 for reality distortion, .82 for poverty symptoms, and .88 for the total BPRS score.

The demographic and clinical characteristics of all participant groups are shown in Table 1. Focused contrasts indicated that the healthy controls did not differ from the patients with schizophrenia on age, gender, or years of parent’s education. However, the controls with depression did have fewer males and African Americans than all other groups. The patients with schizophrenia and depression did not differ significantly in total BPRS scores, but the patients with schizophrenia had an earlier age of first hospitalization, a greater number of prior admissions, and a longer length of illness. Oral doses of antipsychotics were converted to chlorpromazine equivalents using guidelines suggested by Davis, Janicak, Linden, Moloney, and Pavkovic (1983). Depot doses were converted to average daily

3 This hypothesis has recently received empirical support from a study demonstrating activation of the prefrontal cortex among healthy controls during performance of the long versus short delay conditions of the AX-CPT (Barch, Braver, Nystrom, Ferman, Noll, & Cohen, in press).
Table 1
Demographic and Clinical Characteristics of Each Group

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Participants with schizophrenia</th>
<th>Controls with depression</th>
<th>Healthy controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>M = 36.3, SD = 8.7</td>
<td>M = 32.7, SD = 10.0</td>
<td>M = 33.2, SD = 7.7</td>
</tr>
<tr>
<td>Sex (% female)</td>
<td>49</td>
<td>69</td>
<td>39</td>
</tr>
<tr>
<td>Race (% African American)</td>
<td>47</td>
<td>8</td>
<td>45</td>
</tr>
<tr>
<td>Education</td>
<td>12.7</td>
<td>2.3</td>
<td>14.6</td>
</tr>
<tr>
<td>Father's education</td>
<td>13.4</td>
<td>3.1</td>
<td>13.0</td>
</tr>
<tr>
<td>Total BPRS score</td>
<td>39.4</td>
<td>7.9</td>
<td>32.1</td>
</tr>
<tr>
<td>Age of first hospitalization</td>
<td>22.9</td>
<td>6.5</td>
<td>30.1</td>
</tr>
<tr>
<td>No. of previous admissions</td>
<td>8.9</td>
<td>10.1</td>
<td>7.9</td>
</tr>
<tr>
<td>Length of illness (years)</td>
<td>12.7</td>
<td>8.4</td>
<td>1.8</td>
</tr>
<tr>
<td>Chlorpromazine equivalents</td>
<td>733.7</td>
<td>742.8</td>
<td>0.0</td>
</tr>
<tr>
<td>% taking antiparkinsonians</td>
<td>38</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>% taking antidpressants</td>
<td>21</td>
<td>76</td>
<td></td>
</tr>
<tr>
<td>% taking mood stabilizers</td>
<td>19</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>% taking benzodiazepines</td>
<td>21</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

Note. Inpatients and outpatients with schizophrenia were recruited to comprise a single group. Nonetheless, we examined potential clinical differences between the populations and found no significant differences with regard to age, race, gender, parental education, age at first hospitalization, number of previous hospitalizations, chlorpromazine equivalents for neuroleptic dose, number receiving antiparkinsonian agents, or Wechsler Adult Intelligence Scale raw or standard scores. However, these analyses were post hoc and may not have addressed all variables that could have differed between inpatients and outpatients. BPRS = Brief Psychiatric Rating Scale.

The AX-CPT

In this version of the CPT, randomly chosen letters are presented sequentially in a visual display, and participants are instructed to respond only to the letter X, and only when it follows an A. Performance in this task relies on the representation and maintenance of context information, insofar as the correct response to X depends on the previous stimulus (A or not-A). Patients with schizophrenia consistently show increased errors of omission in this task (Comblatt & Keilp, 1994), that is, target misses. This finding is consistent with a failure to process context but could also be explained by a more generalized deficit (e.g., lack of motivation or difficulty initiating a response). To test our hypothesis of a specific deficit in context processing, we used a version of this task that we modified in two ways (Servan-Schreiber et al., 1997). First, we increased the frequency of target (A-X) sequences to 70% of trials, with the remaining 30% divided evenly between three distractor conditions: a non-A followed by an X (which we refer to as the BX condition); an A followed by a non-X (AY); and a non-A followed by a non-X (BY). We hypothesized that this frequency distribution would induce a strong tendency to respond to X, since that is the correct response for 87.5% of the trials in which X appeared. This tendency would have to be inhibited in the BX condition, relying on the use of context (non-A stimulus) to overcome the tendency to respond to X. Thus, in our variant of the task, a failure to process context can be indexed not only by misses in the AX (target) condition but also by false alarms in the BX condition. Our second manipulation was to vary the delay (1,000 or 5,000 ms) between the cue (A/non-A) and the probe (X/non-X) stimuli, in order to test for the ability to maintain context information. We predicted that increased errors in the AX and BX conditions should be greater at the long delay, consistent with our hypothesis that a disturbance in the processing of context should be sensitive to both delay (memory) and frequency (inhibition) manipulations.

Materials and Tasks

The Stroop Task

Stimuli in this task vary in two dimensions (e.g., color words displayed in color), and participants are instructed to respond to one of the two dimensions (read the word or name the color—Stroop, 1935; see MacLeod, 1991, for a review). Typically, there is a dominant tendency to respond to information in one dimension, as evidenced by faster responding to information in that dimension (e.g., word reading is faster than color naming), and by its ability to influence responding to the other dimension (words interfere with and facilitate color naming but not the reverse). Context...
provided by the task instructions must be used to overcome this influence of the stronger dimension when the task is to respond to the weaker one (Cohen, Dunbar, & McClelland, 1990). However, Stroop experiments do not usually place strong demands on the maintenance of context. This is because trials are almost always blocked by task (i.e., word reading or color naming), so that the same instructions (context) are repeatedly reinforced. However, reliance on context could be increased by (a) varying the task to be performed on each trial and (b) introducing a delay between the task instructions for each trial and the stimulus to be responded to. We predicted that such manipulations should further increase the sensitivity of this task to deficits among patients with schizophrenia.

At the beginning of each trial, participants heard an instructional cue (the word “color” or “word” presented auditorily by computer) followed by a stimulus presented on the monitor. Participants were instructed to respond verbally to the stimulus, as designated by the cue, as quickly and accurately as possible. Each stimulus remained on the screen until the participant responded. Reaction times (RTs) were automatically recorded by a voice-activated relay connected to the computer. Responses were also tape-recorded for later coding of accuracy. Two ISIs between the cue and the onset of the stimulus were used: 1 s and 5 s. Three colors and color words were used (red, green, and blue), presented in each of three conditions (congruent, neutral, and incongruent). Congruent stimuli consisted of one of the three color names presented in its own color. Incongruent stimuli consisted of a color name presented in one of the two remaining colors. Neutral stimuli were four colored XXXxs for color naming trials and color words displayed in black for word reading trials. A total of 180 experimental trials were distributed equally across tasks (color naming, word reading), cue-stimulus ISI (short, long), and conditions (congruent, neutral, incongruent) and, within conditions, across the different stimuli for that condition. Trials were pseudorandomly ordered for each participant, with the constraint that each trial type occurred twice in every 24 trials.

Our hypothesis predicts that participants with schizophrenia should show an increase in the influence of words on color naming in the Stroop task. This should result in degradation of performance in the incongruent condition of the color naming task (which relies most heavily on the use of context to support a weaker response against competition from a stronger one) and should manifest as a greater total Stroop effect. The total Stroop effect is the difference between performance measures in the incongruent and congruent conditions, or the sum of facilitation and interference effects. Given that interference effects (incongruent–neutral) are usually substantially greater than facilitation effects (neutral–congruent; MacLeod, 1991), it would seem natural to predict that patients with schizophrenia should exhibit a greater increase in interference than facilitation. Such increases have been observed using the traditional card version of the Stroop task (e.g., Abramczyn, Jordan, & Hegel, 1983; Wysocki & Sweet, 1985). However, this version precludes the use of congruent stimuli and thus the measurement of facilitation. To reliably measure facilitation effects, several recent studies have used tachistoscopic presentation of stimuli and automated measurement of verbal response latency, thereby permitting the randomization of trial types. These studies have consistently shown a characteristic pattern of performance among patients with schizophrenia, with increases in interference limited to accuracy and RT showing an increase in facilitation (Barch, Carter, Hachten, & Cohen, in press; Carter et al., 1997; Carter, Robertson, & Nordahl, 1992; Taylor, Kornblum, & Tandon, 1996). This pattern is consistent with our general prediction of a greater influence of words on colors for patients with schizophrenia (Cohen & Servan-Schreiber, 1992). However, it is a more complex pattern of deficits than was predicted by our original model of the Stroop task. Recently, we have shown that a more refined model of this task (Cohen & Huston, 1994), motivated by an independent set of considerations, can in fact explain this pattern of results (Usher & Cohen, 1997). Thus, for the current study, we predicted a replication of these effects (i.e., increased facilitation in RT and increased interference in accuracy). In addition, we also made the new prediction that these effects would be greater with longer delays between instructions and stimuli, as a consequence of a failure to maintain context. Finally, our design allows us to contrast our hypothesis with a competing interpretation of cognitive deficits in schizophrenia—that these cognitive deficits are due to a disturbance in switching sets. If the problem is primarily one of switching, we would expect patients to perform better, not worse, in the long delay condition, as they would have more time to adapt to the instructions for each trial before the stimulus appears.

### Lexical Disambiguation Task

In this task, participants listened to pairs of sentences presented sequentially and then responded to a visually presented probe by reading it aloud as quickly as possible. The probe was a letter string with one blank space (e.g., SH_FT) that could be completed to make either of two words, one of which was a more frequent completion (dominant: SHIFT) than the other (subordinate: SHAFT). Responses were tape-recorded and later coded manually by the experimenter. Response latency was recorded by computer using a voice-activated relay. In each trial, one of the two preceding sentences provided context favoring either the dominant or the subordinate completion, while the other was neutral with respect to the completion. The participant’s verbal response was used to measure the influence of context on their interpretation of the probe. Delay was manipulated by providing context in either the first or the second of the two preceding sentences. In the long delay condition, the first sentence provided context, followed by the neutral sentence (“Jerry was surprised to find the mine tunnel deserted. It was unusual that there was no one around.”). In the short delay condition, this order was reversed, so that context was presented just prior to the probe (“Jerry was surprised to find the area deserted. It was unusual that there was no one in the mine tunnel.”). To ensure that participants attended to all of the sentences presented, comprehension questions appeared following the participant’s response to the probe on one third of the trials. Finally, a norming condition was included at the end of the experiment, in which participants responded to probe stimuli in the absence of the sentences. This was used to compare the baseline distribution of response frequencies to the probes for patients and controls in the absence of context. Previous studies have indicated that patients with schizophrenia show normal frequency distributions for responses to isolated words (e.g., Lishman & Cohen, 1972; Mefferd, 1978), which we felt was important to confirm using our items. A total of 180 trials were conducted, which included 120 experimental trials and 60 norming trials. Each probe was seen in only one condition, the assignment of probes to conditions was counterbalanced across participants, and the experimental trials were always presented first, followed by a final block of norming trials. During the experimental trials, conditions were presented in random order with the constraint that each of the four conditions occurred twice in every eight trials.

We predicted that, compared with controls, participants with schizophrenia would show a reduced tendency to respond with the context-related interpretation of the probe and that the distribution of their responses (dominant vs. subordinate) would be closer to the distribution observed in the absence of context. In norming studies, we observed approximately 70% dominant responses to these items in the absence of context. This partial but not overwhelming bias toward dominant responses allowed us to make two important predictions. First, we predicted that when context favored the subordinate completion, participants with schizophrenia would produce a greater number of dominant completions than controls. More
important, we predicted that when context favored the dominant completion, participants with schizophrenia would actually make fewer dominant (and more subordinate) completions than controls. This would occur because controls would be biased by the dominant context to produce more dominant responses than would be produced in the absence of context (i.e., >70%), whereas participants with schizophrenia should be less influenced by the biasing context. Thus, this prediction differentiates our hypothesis from the hypothesis of a generalized dominant response tendency (e.g., Chapman, Chapman, & Daut, 1976). Finally, as with our other tasks, we predicted that participants with schizophrenia would show a greater tendency toward acontextual responding in the long vs. short delay conditions.

**Short-Term Memory Measures**

Our theory suggests that a disturbance in context processing can be dissociated from a disturbance in short-term memory (STM). To test this, we administered two standard STM measures, the digit span and the word span recall tasks. We used digit sequences ranging from 2 to 11 items and word sequences ranging from 2 to 8 items. Digit sequences were made up of random numbers (rejecting all regular arrangements and local telephone prefixes). Word sequences avoided obvious semantic relations. The sequences were digitized and presented by computer at the rate of 1 per s, with a fall of voice inflection on the last item. Participants were instructed to respond verbally by attempting to repeat each sequence. Two trials of each length were presented until the participant failed both trials at a particular length. Responses were scored 1 if exactly correct and 0 otherwise. The dependent variable was the longest sequence length at which a participant completed at least one correct trial.

**Procedure**

Each participant was tested individually and was administered the three primary tasks as well as the two measures of STM. Order of task administration was counterbalanced across participants. All tasks were administered within 1 week, typically in two testing sessions. The stimuli were presented on an Apple Macintosh computer, using PsyScope software (Cohen, MacWhinney, Flatt, & Provost, 1993). All visual stimuli appeared in the middle of the computer screen in 14-point Geneva font, subtending an average visual angle of approximately 3 degrees. A short practice period preceded each test to ensure that participants understood the instructions, were comfortable with the apparatus, and were performing the task appropriately.

**Data Analysis**

For each task, analyses were conducted using repeated measures analyses of variance (ANOVAs), with diagnostic group as a between-subjects factor and all experimental conditions as within-subjects factors. When necessary, degrees of freedom were adjusted according to the Geisser-Greenhouse procedure (Neter, Wasserman, & Kutner, 1990). For dependent measures involving accuracy or response proportions, the raw data were normalized using an arcsine transformation (Neter et al., 1990). For measures involving reaction time, we confirmed traditional analyses with analyses using an inverse transformation of the data, to rule out spurious effects of outliers (Ratcliff, 1993). Significant effects that conformed to a priori predictions were evaluated with planned orthogonal contrasts adjusted for unequal sample sizes. Other significant effects were evaluated with post hoc contrasts using Tukey’s HSD for repeated measures, also adjusted for unequal sample sizes.

**Results**

**Performance on Individual Cognitive Tasks**

**AX-CPT**

Within each group, accuracy and RT were negatively correlated, indicating that speed-accuracy tradeoffs were not a factor in this task. RT was not analyzed further given that participants who did not make false alarms in a particular condition (e.g., BX, AY, BY) had no RT data for that condition. Accuracy data were analyzed using planned orthogonal contrasts for each of the four conditions (AX, BX, AY, and BY). As shown in Figure 1, planned contrasts indicated that participants with schizophrenia displayed the predicted pattern of performance when compared with healthy and patient controls—impairments in the AX, $F(1, 106) = 30.33, p < .001,$ and BX, $F(1, 106) = 10.82, p < .001,$ but not the AY, $F(1, 106) = 1.80, p < .10,$ or BY, $F(1, 106) = 2.24, p < .10,$ conditions of the task. Healthy controls and patient controls did not differ in any of the four conditions. This increase in AX misses among patients with schizophrenia cannot be accounted for by a general decrease in responding because there was an increase in responding in the BX condition (false alarms). Conversely, this increase in BX errors cannot be explained by a tendency to respond indiscriminately because no increase was observed in the AY or BY conditions and responding decreased in the AX condition.

We next examined the effects of ISI on performance, again using planned contrasts. As predicted, patients with schizophrenia demonstrated significantly more AX misses, $F(1, 106) = 38.00, p < .001,$ and BX false alarms, $F(1, 106) = 26.41, p < .001,$ at the

![Figure 1](https://example.com/figure1.png)
long than the short ISI. Further, the increase in AX misses at the long ISI among patients with schizophrenia was significantly greater than among healthy and patient controls, $F(1, 106) = 7.30, p < .01$ (see Figure 1). However, contrary to our predictions, there was no such interaction with group for BX false alarms, $F(1, 106) = 7.30, p < .01$. This was because the healthy controls, $F(1, 106) = 15.51, p < .001$, and the patients controls, $F(1, 106) = 10.5, p < .01$, also demonstrated significant increases in BX errors from the short to long ISI. Thus, although participants with schizophrenia showed the predicted increase in both AX and BX errors at the long versus short delay, and this effect was greater than in controls for the AX errors, it was not for the BX errors.

Similar results were obtained using d-prime ($d'$; Swets & Sewall, 1963) computed from the AX hits and BX false alarms. This measure, which we refer to as $d'$-context, compares responses to X in the presence (AX condition) and absence (BX condition) of context and provides a more focused measure of sensitivity to context. As predicted, $d'$-context was overall lower for participants with schizophrenia than controls and decreased from the short to long ISI among participants with schizophrenia (short ISI: $M = 2.69, SE = .08$; long ISI: $M = 1.79, SE = .13$). However, it also decreased with delay for healthy controls (short ISI: $M = 3.83, SE = .09$; long ISI: $M = 2.98, SE = .18$) and patients with depression (short ISI: $M = 3.86, SE = .20$). This pattern was confirmed by an ANOVA that demonstrated main effects of group, $F(2, 106) = 21.73, p < .001$, and ISI, $F(2, 106) = 139.53, p < .001$, but no Group X ISI interaction. This was contrary to our prediction of a differential effect of delay for participants with schizophrenia versus controls.

**Stroop Task**

Accuracy and RT data were analyzed using 4-way ANOVAs, with group as the between-subjects factor and task (color naming, word reading), condition (congruent, neutral, incongruent), and delay (1, 5 s) as within-subjects factors. Within each group, accuracy and RT were negatively correlated, indicating that speed-accuracy tradeoffs were not a factor.

**Accuracy.** Means and standard deviations for the three conditions of each task at each delay for each group are shown in Figure 2. Analysis revealed the standard Stroop effects: a main effect of task, $F(1, 106) = 13.91, p < .001$, with word reading more accurate than color naming; a main effect of condition, $F(2, 212) = 31.01, p < .001$, with more errors in the incongruent than neutral condition, $F(1, 106) = 30.7, p < .001$, and fewer in the congruent than neutral condition, $F(1, 106) = 4.55, p < .05$. There was also a Task X Condition interaction, $F(2, 212) = 13.38, p < .001$, with a greater increase in errors from the neutral to incongruent condition for color naming than word reading, $F(1, 106) = 15.29, p < .001$, but no difference in error facilitation (neutral errors–congruent errors) between color naming and word reading.

There was also a main effect of diagnostic group, $F(2, 106) = 15.74, p < .001$, and both Group X Task, $F(2, 106) = 7.22, p < .01$, and Group X Condition, $F(4, 212) = 11.13, p < .001$, interactions. Planned contrasts indicated that participants with schizophrenia made more errors overall than both control groups, $F(1, 106) = 31.48, p < .001$, who did not differ. Consistent with our hypothesis, participants with schizophrenia displayed a greater increase in errors between the congruent and incongruent conditions (i.e., total Stroop effect; $F(1, 106) = 26.02, p < .001$) than controls. More specifically, interference (i.e., the decrease in accuracy from the neutral to incongruent condition; $F(1, 106) = 20.37, p < .001$) and facilitation (increase in accuracy from the neutral to congruent condition; $F(1, 106) = 6.31, p < .05$) were significantly greater among participants with schizophrenia than both control groups. The difference between participants with schizophrenia and controls was significantly greater for color naming than word reading, $F(1, 106) = 14.4, p < .001$. These two-way interactions were modified by a three-way interaction between group, task, and condition, $F(4, 212) = 5.72, p < .01$. Planned contrasts indicated that, as predicted, the difference between participants with schizophrenia and controls was greatest for color naming than word reading, $F(1, 106) = 11.77, p < .001$. However, the predicted four-way interaction between group, task, condition, and delay was not significant, $F(4, 212) = 0.14, p < .10$, with participants with schizophrenia comparably impaired at the short and long delays.

**RT.** Means and standard deviations are shown in Figure 2. As with the accuracy data, analyses revealed the standard Stroop effects: a main effect of task, $F(1, 106) = 46.99, p < .001$, with word reading faster than color naming; a main effect of condition,
$F(2, 212) = 177.84, p < .001$, with slower RTs in the incongruent than neutral condition (i.e., interference; $F[1, 106] = 252.25, p < .001$); and a Task $\times$ Condition interaction, $F(2, 212) = 62.57, p < .001$, with stronger interference effects for color naming than word reading, $F(1, 106) = 93.94, p < .001$. We did not find significant overall facilitation effects (congruent faster than neutral) in either color naming or word reading.

There was a significant main effect of diagnostic group, $F(1, 106) = 8.52, p < .001$, with participants with schizophrenia slower than the controls, $F(1, 106) = 17.03, p < .001$. Furthermore, the predicted three-way interaction of group, task, and condition was significant, $F(4, 212) = 2.37, p = .05$, with participants with schizophrenia showing a significantly greater difference than controls between the incongruent and congruent conditions (i.e., total Stroop effect; $F[1, 106] = 20.21, p < .001$, but only for color naming, $F(1, 106) = 7.07, p < .01$. Also as predicted, participants with schizophrenia exhibited a significantly greater facilitation effect (congruent vs. neutral) than controls for color naming, $F(1, 106) = 5.45, p < .05$, while there were no group differences in interference effects (incongruent vs. neutral). There was a trend toward a three-way interaction of Task $\times$ Condition $\times$ ISI, $F(2, 212) = 2.40, p = .09$, but the predicted four-way interaction between group, task, condition, and ISI was not significant. Planned contrasts indicated that participants with schizophrenia displayed a significantly greater facilitation effect at the long ISI than at the short ($p < .01$; short ISI: $M = 22$ ms, $SE = 63$; long ISI: $M = 129$ ms, $SE = 74$). However, normal controls also showed a tendency for facilitation to increase from the short to long ISI (short ISI: $M = -68$ ms, $SE = 30$; long ISI: $M = -16$ ms, $SE = 26$), although patient controls did not (short ISI: $M = -85$ ms, $SE = 25$; long ISI: $M = -84$ ms, $SE = 35$).

**Lexical Disambiguation Task**

**Norming data.** The proportion of dominant completions was 0.71 for participants with schizophrenia and 0.73 for controls, indicating that the groups did not differ in their distribution of responses to stimuli in the absence of context. Furthermore, the item-wise correlation of completions between groups was 0.72 ($p < .001$), indicating that the groups tended to show similar response distributions for a given stimulus.

*Experimental data.* Means and standard deviations for proportion of dominant completions are shown in Figure 3. A three-way ANOVA was conducted with group as the between-subjects factor and condition (dominant context, subordinate context) and delay (context in first sentence, context in second sentence) as within-subjects factors. The dependent variable was the arcsine transformation of the proportion of responses in each condition consistent with the biasing context. This analysis revealed main effects of condition, $F(1, 106) = 668.98, p < .001$, and delay, $F(1, 106) = 75.70, p < .001$. As shown in Figure 3, the proportion of dominant responses was higher in the dominant versus the subordinate context, and the proportion of context-biased responses was higher when context was in the second sentence (no delay) versus the first sentence (delay).

The predicted main effect of group was also significant, $F(2, 106) = 17.97, p < .001$, with participants with schizophrenia producing significantly fewer context-biased responses than healthy controls or those with depression, $F(1, 106) = 27.19, p < .001$, who did not significantly differ from one another. As shown in Figure 3, participants with schizophrenia made more dominant responses than controls in the subordinate context condition, $F(1, 106) = 25.19, p < .001$, but fewer dominant responses than controls in the dominant context condition, $F(1, 106) = 25.43, p < .001$. This is what would be predicted if their responses were closer to those produced in the absence of context. This finding is not consistent with the overall dominant response tendency, which would predict more dominant responses in all conditions. Finally, the predicted three-way interaction between group, condition, and delay was significant, $F(2, 214) = 4.60, p < .01$. Compared with controls, participants with schizophrenia exhibited a greater decrease in context-biased responses from the short to long delay, but only in the subordinate context condition, $F(1, 106) = 3.70, p < .05$, which required maintenance of context information to override the more potent (i.e., dominant) completion.

**Differential Versus Generalized Deficits**

The results reported above are consistent with a number of the predictions made by our hypothesis concerning disturbances in the processing of context in schizophrenia. However, the conditions in which patients with schizophrenia showed predicted impairments were, in several cases, also the most difficult for controls, raising the possibility that impairments reflected a generalized deficit, rather than a specific disturbance in the processing of context. To address this concern, we conducted post hoc regression procedures proposed by Chapman and Chapman (Chapman & Chapman, 1989; Chapman, Chapman, Curran, & Miller, 1994). For each task, we used data from control participants to compute the regression equation predicting performance in context-sensitive conditions from that in control conditions. We used this regression equation to compute the predicted score on each measure of interest for each participant in every group, based on that individual’s performance in the control conditions. We then calculated a standardized resid-
ual score for each participant (observed−predicted)/(SE of regression), and conducted focused contrasts on these scores comparing participants with schizophrenia to controls. These procedures were applied to both the accuracy and RT data, as appropriate for each task.

For accuracy measures, participants with schizophrenia displayed (a) more AX errors at the long delay than would be predicted from their AX performance at the short delay \( p < .01 \), although this was not true for BX errors; (b) more color naming errors in the incongruent condition than would be predicted from their color naming performance in the neutral condition \( p < .01 \); and (c) fewer context-biased completions in the subordinate context, long delay condition than would be predicted from their performance in the corresponding short delay condition \( p < .01 \). For RT measures, participants with schizophrenia displayed (a) significantly greater color naming facilitation in the Stroop task than would be predicted from their overall RT and (b) a trend toward greater facilitation in the long delay condition than would be predicted from the short delay \( p < .07 \).

**Cross-Task Correlations**

A central claim of our theory is that a disturbance in the processing of context can provide a common account for the pattern of deficits in each of the tasks described above. Each of the three tasks included one or more primary measures predicted to be sensitive to such a disturbance, for example: (a) BX false alarms at the long delay in the AX-CPT; (b) color naming errors to incongruent stimuli and RT facilitation at the long delay in the Stroop task; and (c) proportion of dominant responses in the subordinate context, long delay condition of the lexical disambiguation task. If impairments of performance among participants with schizophrenia in each of these conditions reflects a disturbance in the same underlying mechanism, then these measures should all be strongly associated with one another. To test this, we computed within-subject correlations among these measures and compared this with correlations among control measures from each task that, according to our theory, should not be sensitive to the processing of context (e.g., conditions in which the correct response was the dominant one or in which there was only a brief delay between context and response). As shown in the Table 2, there were strong correlations among all of the primary measures, both for the total sample and for participants with schizophrenia alone. In contrast, the control measures were much less strongly and consistently associated.

Although these results are consistent with our hypothesis, some of the control measures had lower variance than the primary measures.
Table 3
Reliability and Variance of Primary and Control Measures for Each Task

<table>
<thead>
<tr>
<th>Measure</th>
<th>All participants</th>
<th>Participants with schizophrenia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Alpha</td>
<td>Variance</td>
</tr>
<tr>
<td>Primary measures</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AX-CPT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BX errors—long delay</td>
<td>.88</td>
<td>.4734</td>
</tr>
<tr>
<td>Stroop</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incongruent color naming errors—long delay</td>
<td>.90</td>
<td>.4911</td>
</tr>
<tr>
<td>Color naming facilitation—long delay</td>
<td>.50</td>
<td>20.1822</td>
</tr>
<tr>
<td>Lexical disambiguation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% dominant responses in subordinate/neutral condition</td>
<td>.62</td>
<td>.4212</td>
</tr>
<tr>
<td>Control measures</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AX-CPT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>d-prime—short delay</td>
<td>.96</td>
<td>.3760</td>
</tr>
<tr>
<td>Stroop</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incongruent color naming errors—short delay</td>
<td>.90</td>
<td>.5104</td>
</tr>
<tr>
<td>Color naming interference—short delay</td>
<td>.50</td>
<td>19.8808</td>
</tr>
<tr>
<td>Lexical disambiguation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% subordinate responses in dominant/subordinate condition</td>
<td>.45</td>
<td>.3678</td>
</tr>
</tbody>
</table>

Note. AX-CPT = AX version of the Continuous Performance Test. BX indicates a distractor condition. These control measures are labeled as Set 2 in Table 2.

measures. Thus, the lower correlations among the control measures could simply be an artifact of reduced variance. To address this issue, we chose a second set of control measures that were matched to the primary measures on reliability and variance (see Table 3). Although similar in reliability and variance, the second set of control measures were still much less strongly and consistently associated than the primary measures (see Table 2). For example, among participants with schizophrenia, all six of the correlations for the primary measures were in the predicted direction, and five of the six were significant. In contrast, none of the six correlations for the control measures were significant, and several were in the opposite direction. These findings provide strong evidence for the construct validity of our primary measures. They are also consistent with our hypothesis that a disturbance in a single underlying mechanism contributed to the predicted pattern of deficit across all three tasks.

**STM Assessment**

Data from the two measures of STM, digit span and word span, were analyzed using one-way ANOVAs, with diagnostic group as a between-subjects factor. These ANOVAs did not indicate any significant differences among groups, either for the digit span ($p > .10$; healthy controls, $M = 6.0$, $SD = 1.5$; controls with depression, $M = 6.5$, $SD = 1.3$; participants with schizophrenia, $M = 5.7$, $SD = 1.0$) or the word span ($p > .05$; healthy controls, $M = 4.8$, $SD = 0.6$; controls with depression, $M = 4.8$, $SD = 0.9$; participants with schizophrenia, $M = 4.4$, $SD = 0.8$).

**Clinical Symptoms**

We predicted that cognitive deficits would be associated with the disorganization dimension of positive symptoms. To test this, we conducted multiple regression analyses examining the association between the Disorganization subscale of the BPRS and the primary measures from each of our cognitive tasks. To assess the specificity of any positive findings, similar analyses were conducted using the Reality Distortion and Poverty symptoms subscales, as well as the total BPRS score. Cognitive performance was significantly associated with disorganization ($R^2 = .17$, $p < .05$) but not reality distortion ($R^2 = .07$, $p > .10$), negative symptoms ($R^2 = .04$, $p > .10$), or total BPRS score ($R^2 = .07$, $p > .10$).

**Psychopharmacological Variables**

Medication effects pose a difficult problem for research involving patients with schizophrenia. There are two issues in using only medicated patients. Negative findings could be attributed to the therapeutic effects of medication. Conversely, positive findings might reflect the effects of medication and not disease-specific deficits. Although we studied only medicated patients, and so could not address these problems directly, we explored them indirectly as follows. First, we examined correlations between dose of antipsychotic medication (in chlorpromazine equivalents) and our cognitive measures. None of these correlations were significant (average $r = .05$; range $= -.14$ to .07). Second, we examined the influence of anticholinergic medications (known to affect memory performance; Spohn & Strauss, 1989) by dividing participants with schizophrenia into those on such medications and those not, and then testing for group differences in cognitive performance using independent sample $t$ tests. There were no significant differences for any of our primary cognitive measures.

**Discussion**

The results of this study provide support for several of the predictions made by our theory concerning a disturbance in
context processing in schizophrenia. First, participants with schizophrenia displayed the greatest deficits in the task conditions that drew most heavily on the use of context information to support the execution of weaker but task-appropriate responses. In the CPT, this was evidenced by significantly more AX misses and BX false alarms but no increase in other types of errors as compared with controls. In the Stroop task, this was evidenced by a disproportionately greater number of errors in the color naming incongruent condition and by a disproportionately greater RT facilitation effect. In the lexical disambiguation task, this was evidenced by fewer context mediated responses in both the subordinate and dominant context conditions. Furthermore, these effects were accentuated by an increase in the delay between context and probe in both the AX-CPT and lexical disambiguation tasks. At the same time, our findings raise several questions, including the specificity of the deficits we observed, a failure to corroborate some of our theoretical predictions, and the relationship of these cognitive deficits to the clinical symptomatology of schizophrenia. We consider each of these issues in the following sections.

Specificity of Deficits to the Processing of Context

It could be argued that the cognitive deficits we observed among patients with schizophrenia reflect a generalized deficit, rather than a disturbance in a specific cognitive mechanism such as we have proposed. This is a concern because some of the conditions in which participants with schizophrenia differed most from controls were also the most difficult conditions for controls. However, three factors weigh against such an interpretation. First, in at least some cases performance was matched across conditions for controls, but elicited predicted differential effects among patients with schizophrenia. For example, in the AX-CPT, the performance of controls in the AX condition did not differ from the AY, whereas patients with schizophrenia showed the predicted pattern: a significant increase in AX but not AY errors. This finding is consistent with a specific deficit in context processing, although this finding could be subject to concerns about a ceiling effect in such errors for control participants. However, another source of support for the presence of a differential deficit is provided by the regression analyses we conducted, as suggested by Chapman and Chapman (1989). The deficits we observed in patients with schizophrenia were greater than would be predicted based on their performance in control conditions. Finally, we found that these deficits were selective to patients with schizophrenia, inasmuch as the performance of patients with depression was virtually identical to that of healthy controls, even though their overall degree of symptomatology did not differ significantly from patients with schizophrenia.

We should note, however, some potential concerns about the regression procedures referred to above. The range of scores among controls for the predictor variables was relatively restricted in some cases (e.g., AX-CPT and Stroop accuracy). In addition, the range of performance shown by controls did not fully span the range of performance shown by patients with schizophrenia. For both of these reasons, it is possible that the regressions derived from control participant data are biased estimates and do not accurately predict performance in the range relevant to patients with schizophrenia. If these were underestimates, this would lead to an overestimate of the magnitude of deficits among patients with schizophrenia and to a misinterpretation of the specificity of these deficits. One way of addressing this issue would be to use a control population exhibiting greater overlap in performance with schizophrenia patients but who are not thought to have the same cognitive deficits. For example, one possibility for future research would be to use older healthy adults as an additional control population to age-matched controls. Although the use of older controls introduces age-confounds, older controls often do perform closer to the range of schizophrenia patients.

Although psychometric issues remain, other findings from our study support the specificity of deficits observed among patients with schizophrenia. For example, we found strong correlations in performance across tasks in conditions selected to be maximally sensitive to context. In contrast, we did not find significant correlations for performance in other task conditions, even when these were matched for reliability and variance. This finding suggests that there is more structure to the pattern of deficits observed for participants with schizophrenia than would be predicted by a generalized deficit, and it is consistent with our hypothesis that a single underlying disturbance in the processing of context is responsible for the performance deficits observed in each task. The cross-task correlations we observed are noteworthy, in light of the difficulties that previous studies have faced trying to elicit such relationships among deficits across cognitive tasks in schizophrenia (e.g., Asarnow & MacCrimmon, 1981; Kopfstein & Neale, 1972). These studies may have used tasks that included context-sensitive conditions (thus eliciting schizophrenia-related deficits on individual tasks) but measures of performance that were not sufficiently specific to the context-sensitive conditions of these tasks (thus failing to elicit cross-task associations in the pattern of deficit). Our theory allowed us to decompose a set of disparate tasks, identify the conditions maximally sensitive to the processing of context, and demonstrate a predicted and significantly correlated pattern of deficits among participants with schizophrenia across these tasks.

Finally, two other findings are consistent with the specificity of the observed deficits to a disturbance in the processing of context. First, we hypothesized that a disturbance in the processing of context can be dissociated from STM disturbances. Consistent with this hypothesis, participants with schizophrenia did not display deficits on standard measures of STM. Second, with an increase in the delay between context and response, participants with schizophrenia displayed either no change or worsening of their performance. This pattern cannot be easily accounted for by alternative hypotheses about cognitive deficits in schizophrenia, such as impairments in stimulus processing (Nuechterlein & Dawson, 1984) or switching preparatory sets (Shakow, 1962). These hypotheses would predict improved performance with a delay by providing participants with more time to process the stimuli, or to switch sets, or both. Nevertheless, our predictions concerning a differential worsening of performance among participants with schizophrenia in the long delay conditions of each task, relative to control participants, received only partial corroboration. This was an important prediction, and our failure to fully corroborate it warrants further consideration.
Failure to Corroborate Predictions Concerning Delay Effects

The predicted effects of the delay manipulation were observed for participants with schizophrenia in the AX-CPT and language tasks and to some extent in the Stroop task. However, similar effects of delay were observed for control participants in the AX-CPT and Stroop tasks. This could be taken as evidence against the specificity to schizophrenia of the deficits that were observed. However, there are other possible explanations for this finding. First, the manner in which delay was manipulated may have spuriously produced greater delay effects among control participants in the AX-CPT and Stroop tasks than the language task. Delay was manipulated in a mixed design in all tasks (i.e., varied randomly across trials). However, the predictability of the probe onset time differed across tasks. In the language task, the probe always appeared at a predictable time, immediately following the second sentence. In the AX-CPT and Stroop tasks, the probe followed the cue after a delay that alternated randomly between 1 and 5 s. Thus, participants were uncertain about the timing of the probe presentation from trial to trial. This unpredictability may have interfered with the processing of context among controls, producing decrements in performance similar to those we predicted for participants with schizophrenia, and thus reducing our ability to detect a differential effect of delay associated with schizophrenia. This interpretation of our results suggests that the effect of delay in control participants should be eliminated in the AX-CPT and Stroop tasks if trials were presented blocked by delay, making target presentation predictable. However, patients with schizophrenia should continue to show delay effects under such conditions. These predictions are consistent with results from a previous study we conducted using the AX-CPT with delay manipulated in a blocked design (Servan-Schreiber et al., 1997), in which participants with schizophrenia showed delay effects similar to those reported here, while controls with depression did not. Results from recent pilot studies in our lab provide additional support for this interpretation. Twelve control participants were tested in both mixed and blocked design versions of our AX-CPT. Both AX misses and BX false alarms increased with delay when this was manipulated in a mixed design but not in a blocked design.

A second factor that may have influenced our ability to detect a differential effect of delay among participants with schizophrenia is medication status. That is, medications may have ameliorated the effect of delay in participants with schizophrenia, attenuating the difference from controls. Our post hoc analyses did not demonstrate any associations between medication status and task performance. However, chlorpromazine equivalents are not an ideal measure of neuroleptic levels (e.g., Spohn & Strauss, 1989). Thus, it is possible that medication effects may have gone undetected in our analyses. In our previous work with the AX-CPT (Servan-Schreiber et al., 1997), we found that the presence of a delay effect among participants with schizophrenia was in fact closely related to medication status: Unmedicated participants with schizophrenia displayed a disproportionate increase in BX errors at the long ISI, whereas medicated participants (and controls with depression) did not. Although the medicated participants with schizophrenia in our previous study did not display a delay effect, they did exhibit an overall worsening of performance. We interpreted these findings as suggesting that chronic administration of antipsychotic medications may serve to ameliorate context processing deficits (diminishing the effect of delay in medicated participants), while at the same time inducing a generalized degradation in performance. In the present study, medicated participants with schizophrenia did display a decrease in performance from the short to long delay; however, this effect was not significantly different from a similar one observed for control participants. As noted above, it is unclear whether the effects of delay in the present study were more closely related to disturbances in the processing of context or to unintended task-related factors such as the use of a mixed design to manipulate delay. This question would be best addressed by a study that prospectively controls for medication status and manipulates delay in a blocked design.

Clinical Relevance of Deficits in Context Processing

The results of this study corroborated our prediction that a disturbance in the processing of context would be associated with the disorganization rather than the reality distortion component of positive symptoms. However, context processing deficits accounted for a relatively small proportion of the variance in disorganization scores. In part, this may be due to the fact that the disorganization subscale includes several items that may themselves be differentially related to context processing. In particular, we hypothesize that the conceptual disorganization item may be more strongly related to context processing deficits than the mannerisms and posturing item. This hypothesis is consistent with data from other recent studies suggesting an association between context processing and formal thought disorder in schizophrenia (Barch & Berenbaum, 1996; Docherty et al., 1996; Kuperberg, McGuire, Tyler, & David, 1997). Further, this hypothesis is consistent with the suggestion that language production disturbances among patients with schizophrenia—the primary overt manifestation of formal thought disorder—arise from an inability to maintain prior discourse context (Barch et al., 1996). We were not able to address this more specific hypothesis in the present study because the BPRS provides only a single item with which to assess formal thought disorder (conceptual disorganization). In future work, the use of expanded assessments will allow us to examine this issue more fully.

Notably, we did not find any reliable association between deficits in context processing and either negative symptoms or global psychopathology. This lack of an association with negative symptoms replicates a similar finding in our previous work (Servan-Schreiber et al., 1997). As noted previously, the empirical support for a hypothetical link between negative symptoms, hypofrontality, and cognitive deficits has been mixed. Such equivocal findings might lead one to conclude that in schizophrenia, negative symptoms are not associated with disturbances in frontal function or the processing of context. However, two potential methodological confounds require further examination before such a conclusion can be drawn. First, like most other studies, our experimental design was cross-sectional, which may not provide the most sensitive test of relationships between clinical symptoms and experimental variables. Second, medication effects may have confounded assessment of clinical symptoms and of negative symptoms in particular. Our study, like several others that have not found such correlations, used only medicated patients (e.g., Pan-
Conclusion

The results of the present study provide support for several of the predictions made by our primary hypothesis—that a variety of cognitive deficits in schizophrenia reflect a disturbance in a single underlying information-processing mechanism: a degradation in the processing of context needed to select task-appropriate action. The support for this hypothesis includes corroboration of a number of detailed predictions concerning patterns of cognitive performance in both normal participants and those with schizophrenia and correlations in performance across seemingly disparate tasks. Further, these findings contribute to our understanding of the relationship between cognitive and clinical variables, suggesting an association between disturbances in the processing of context and symptoms of formal thought disorder. However, a number of important issues remain to be addressed in future work, the most important of which are the specificity of cognitive deficits shown by schizophrenia patients and clarification of the role played by neuroleptic medications. Nevertheless, the present study helps illustrate the power that modern cognitive psychological techniques can bring to bear in the study of schizophrenia, both at the theoretical and empirical levels. We strongly believe that further work along these lines will lead not only to a deeper understanding of the psychological processes disrupted in schizophrenia but also to the development of more sensitive measures for detecting and evaluating these processes that will be valuable in both clinical research and practice.

References


Andreasen, N. C., Pfefferbaum, R., & Schwartz, S. (1982). Increased Stroop facilitation effects in schizophrenia are not due to increased automatic spreading activation. Manuscript submitted for publication.


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