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Context-Processing Deficits in Schizophrenia: Diagnostic Specificity, 4-Week Course, and Relationships to Clinical Symptoms

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Previous research on schizophrenia suggests that context-processing disturbances are one of the core cognitive deficits present in schizophrenia. However, it is not clear whether such deficits are specific to schizophrenia as compared with other psychotic disorders. To address this question, the authors administered a version of the AX Continuous Performance Test designed to assess context processing in a sample of healthy controls, patients with schizophrenia, and patients with other psychotic disorders. Participants were tested at index (when medication naive and experiencing their first contact with psychiatric services) and 4 weeks later, following medication treatment. At index, patients with schizophrenia and the psychotic comparison group demonstrated similar impairments in context processing. However, context-processing deficits improved in the psychotic comparison group at 4 weeks but did not improve in patients with schizophrenia.

The goal of identifying and characterizing cognitive deficits that are specific to schizophrenia has long been a focus of research on this debilitating disorder. In previous work, Cohen and colleagues have examined the nature of cognitive deficits in schizophrenia by developing neural network models of performance in cognitive tasks known to elicit performance deficits in individuals with schizophrenia. On the basis of such work, Cohen and colleagues have put forth the hypothesis that the dorsolateral prefrontal cortex (DLPFC) is responsible for the processing of context and that a disturbance in this mechanism is responsible for a range of cognitive deficits seen in schizophrenia (Braver & Cohen, 1999; Cohen & Servan-Schreiber, 1992). However, little is known about the specificity of context-processing deficits of schizophrenia versus other psychotic disorders. This information is important for theory development in that the role attributed to cognitive deficits in the development of schizophrenia versus other disorders would differ if context-processing deficits were as severe in other psychotic disorders as in schizophrenia. Thus, the goal of the current project was to examine cognitive function in medication-naive individuals with schizophrenia and in individuals with other psychotic disorders to examine the specificity of context-processing deficits to schizophrenia.

The hypothesis concerning context-processing deficits in schizophrenia (Barch et al., 2001; Braver, Barch, & Cohen, 1999; Braver & Cohen, 1999; Cohen & Servan-Schreiber, 1992) suggests that at least a subset of cognitive deficits in this disorder reflect disturbances in the ability to represent and maintain context information due to a disturbance in the function of dopamine in DLPFC. Context refers to prior task-relevant information that is represented in such a form that it can bias selection of the appropriate behavioral response. Context representations can include task instructions, a specific prior stimulus, or the result of processing a sequence of prior stimuli (e.g., the interpretation that results from processing a sequence of words in a sentence). Because context representations are maintained online, in an active state, they are continually accessible and available to influence processing. Consequently, context can be viewed as the subset of representations within working memory that govern how other representations are used. 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 (online 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 but operating under different task conditions. As such, it is possible that disturbances in attention, working memory, and inhibition in schizophrenia can all be understood in terms of a deficit in context processing (Barch et al., 2001; Braver et al., 1999; Braver & Cohen, 1999; Cohen & Servan-Schreiber, 1992).

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The context processing hypothesis is not necessarily meant to be an alternative to the hypothesis that patients with schizophrenia have deficits in working memory (Goldman-Rakic, 1994; Park & Holzman, 1992), as context processing and working memory are not completely independent constructs. Context processing is one subcomponent of working memory. As such, the hypothesis regarding deficits in context processing in schizophrenia is a more specific version of the working memory hypothesis. In addition, the context processing hypothesis can explain why patients with schizophrenia demonstrate deficits on at least some tasks thought to tap working memory as well as deficits on other cognitive control tasks that may not involve a high working memory load (e.g., The Stroop Color and Word Test). The context processing hypothesis states that there is something important about abstracting out contextual meaning from prior events in order to maintain and utilize such information to bias future processing and behavior. It is this component of working memory that is theorized to most strongly elicit deficits among patients with schizophrenia. Thus, in working memory tasks in which patients simply have to maintain few items, with no distraction, but do not have to determine the contextual meaning of the stimuli, they tend to show little deficits (Cohen, Barch, Carter, & Servan-Schreiber, 1999). In contrast, the context processing hypothesis argues that patients would show deficits on tasks in which context information needs to be determined and maintained, even if this contextual information constitutes a relatively low working memory load.

A task that has been used to examine our hypotheses about context processing in numerous prior studies is a version of the classic Continuous Performance Test (CPT; Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956) known as the AX-CPT (Cohen et al., 1999; Servan-Schreiber, Cohen, & Steingard, 1996). In this task, sequences of letters are presented, one at a time, as a series of cue–probe pairs. The object of the task is to make a target response to an X (the probe), but only when it follows an A (the cue), and make a nontarget response in all other cases (hence, the name AX). Performance in this task relies on the representation and maintenance of context information in that the correct response to X depends on the cue stimulus (A or not-A). Target (AX) trials occur with high frequency (70%) in our version of the AX-CPT. This induces two types of biases in subjects. The first bias is to make a target response to the occurrence of an X probe because this is the correct response on the majority of trials (87.5%). On trials in which a target response should not be made to the X probe (i.e., BX trials, where B refers to any non-A cue), context information must be used in an inhibitory fashion to override the tendency to false alarm. The second bias is an expectancy to make a target response following the occurrence of an A cue. In this case, the context provided by the cue serves a predictive function that directs attention to a particular response (i.e., attention-to-action; Allport, 1989; Norman & Shallice, 1986). On those trials in which the cue is an invalid predictor of the response (i.e., AY trials, where Y refers to any non-X probe), this attentional function of context actually creates the tendency to false alarm.

The design of this version of the AX-CPT allows one to examine the integrity of context processing by examining performance on various types of nontarget trials as well as AX target trials. On BX trials, the internal representation of context should improve performance by inhibiting an inappropriate response bias. However, on AY trials, representation of context should impair performance by creating an inappropriate expectancy bias. Thus, if context representations are intact, AY performance should be worse than BX performance (in terms of both errors and reaction time [RT]). Conversely, if context representations are impaired, as hypothesized for patients with schizophrenia, BX performance should be worse than AY performance, including both increased errors and slower RTs. Thus, the context processing hypothesis predicts that on the AX-CPT, patients with schizophrenia should demonstrate more BX errors and slower BX RTs than controls. However, if context representations are impaired in schizophrenia, then individuals with this disorder should display normal or improved performance on AY trials, the condition in which intact context representations tend to induce false alarms and slow RTs in controls. Specifically, compared with controls, patients with schizophrenia should display equal (or even fewer) AY errors and equal (or even faster) AY RTs. Performance on AX target trials should also be poorer if context processing is impaired because determination of targets is dependent on the context provided by the cue. Finally, a third type of nontarget trial, BY, provides a useful internal control because in this condition the influence of context on performance should be relatively small (given that both the cue and the probe always map to a nontarget response). Thus, errors of this type reflect random responding or nonspecific lapses of attention.

The AX-CPT paradigm also provides a means for examining the mnemonic role of context information through the cue–probe delay duration. In conditions with a long cue–probe delay (e.g., 5 s), context must be actively maintained within working memory (supported by prefrontal cortex in many theories of working memory function). Thus, the same context-processing mechanism that subserves inhibitory and attentional functions also subserves working memory functions. Consequently, a prediction of the theory is that the effect of delay will interact with performance on AY and BX trials. If context maintenance is intact, then the strength of context representations should either hold constant or increase with delay (if it takes some period of time for context representations to reach full activation strength). Specifically, BX performance should remain constant or improve at long delays, whereas AY performance should remain constant or worsen. Conversely, if context maintenance is impaired, then context representations should lose strength over time. This should lead to a worsening of BX performance with a delay but to an improvement in AY performance.

As noted earlier, a number of prior studies have provided evidence consistent with several of these predictions and therefore provide support for the hypothesis concerning context-processing deficits in schizophrenia. For example, several behavioral studies have found selective patterns of performance deficits among patients with schizophrenia on the AX-CPT and other tasks measuring context processing (Barch, Braver, Cohen, & Servan-Schreiber, 1998; Barch et al., 2001; Cohen et al., 1999; Javitt, Shelley, Silipo, & Lieberman, 2000; Niznikiewicz et al., 1997; Salisbury, O'Donnell, McCarley, Nestor, & Shenton, 2000; Servan-Schreiber et al., 1996; Stratta, Daneluzzo, Bustini, Casacchia, & Rossi, 1998; Stratta, Daneluzzo, Bustini, Prosperini, & Rossi, 2000; Titone, Levy, & Holzman, 2000) as well as strong correlations of performance among task conditions specifically thought to assess context processing. In addition, medication-naive

first-episode patients with schizophrenia demonstrate impaired DLPFC activation associated with impaired context processing (Barch et al., 2001). Note that deficits in context processing seem to be particularly associated with the presence of disorganization symptoms in patients with schizophrenia (Barch, Carter, Hachten, & Cohen, 1999; Barch, Carter, Perlstein, et al., 1999; Cohen et al., 1999; Stratta et al., 2000). Further, unaffected siblings of patients with schizophrenia also demonstrate a selective deficit in context processing (MacDonald, Pogue-Geile, Johnson, & Carter, in press). Finally, research has provided some evidence for specificity to schizophrenia in that individuals with nonpsychotic major depression do not show the same pattern of performance deficits seen in schizophrenia (Cohen et al., 1999). However, research has not yet determined whether individuals with schizophrenia can be distinguished from individuals with other psychotic disorders in terms of context-processing deficits.

Several studies of cognitive function in schizophrenia have examined whether the pattern of deficits found in patients with schizophrenia is selective to this disorder or is also present in other psychotic disorders. For example, McGrath, Scheldt, Welham, and Clair (1997) examined executive function (verbal fluency, Wisconsin Card Sort, Stroop Color and Word Test) in chronic medicated patients with schizophrenia and mania (some of whom had psychotic symptoms) and found that both groups showed impaired executive function as compared with controls, with no significant difference between the patients with schizophrenia and with mania. Similarly, Dickerson, Sommerville, Origoni, Ringel, and Parente (2001) found few differences between chronic patients with schizophrenia and chronic patients with bipolar disorder on the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS) tests as well as on Trails A. However, patients with schizophrenia were significantly more impaired than bipolar patients on immediate verbal memory from the RBANS. Verdoux and Liraud (2000) compared cognitive function in patients with schizophrenia, with other psychotic disorders (i.e., delusional disorder, etc.), with bipolar disorder, and with major depression (half of whom had psychotic features). The four groups did not differ in performance on measures of executive function, such as the Wisconsin Card Sort and the Stroop test. However, patients with schizophrenia were significantly more impaired than the other three groups on measures of episodic memory function from the abbreviated Battery of Memory Efficiency (BEM-84). Zihl, Gron, and Brunnauer (1998) compared a large sample of patients with schizophrenia and with affective disorders and again found few differences between the two groups in terms of cognitive function.

However, other studies have found more evidence for greater cognitive impairment in patients with schizophrenia as compared with those with other disorders. For example, Nuechterlein, Dawson, Ventura, Miklowitz, and Konishi (1991) found that patients with schizophrenia performed worse than patients with bipolar disorder on a degraded version of the CPT. Park and Holzman (1992) found that patients with schizophrenia performed worse than bipolar patients on both occulomotor and haptic versions of a working memory task. Similarly, Gooding found that patients with schizophrenia performed significantly worse than bipolar patients on both working memory and antisaccade tasks, although the bipolar patients were also impaired compared with healthy controls on antisaccade accuracy and working memory RTs (Gooding & Tallent, 2001). Goldberg has found greater impairment in visual memory and executive function in patients with schizophrenia as compared with depressed and bipolar patients (Goldberg et al., 1993). Hawkins et al. (1997) also found more cognitive impairments in patients with schizophrenia as compared with bipolar disorder. Hobart, Goldberg, Bartko, and Gold (1999) found that patients with schizophrenia were more impaired than were bipolar patients on the Vocabulary subscale of the Wechsler Adult Intelligence Scale—Revised (WAIS–R; Wechsler, 1991), several measures of episodic memory, and Stroop performance, although patients with bipolar disorder were also impaired in a number of cognitive domains compared with controls. As one can see, the results of such studies examining the specificity of cognitive deficits to schizophrenia are mixed, with some studies finding greater deficits in patients with schizophrenia as compared with patients with other disorders, and other studies finding equally severe cognitive impairments in other psychotic and nonpsychotic disorders (for a review, see Goldberg, 1999). However, the vast majority of these studies have examined only chronic medicated patients at a single time point, often when all patients are acutely ill. Little is known about the comparability of cognitive function in unmedicated patients with other psychotic disorders early in the course of illness, and little is known about whether cognitive deficits in disorders other than schizophrenia endure when such patients are not acutely ill (as is true in schizophrenia).

The goal of the current study was to examine the specificity of context-processing deficits to patients with schizophrenia as compared with patients of other psychotic disorders. As noted earlier, this information is critical for developing a theory about the specific relationships between cognitive deficits and the development and maintenance of schizophrenia. The role attributed to cognitive deficits in the development of schizophrenia versus other disorders would differ if some or all cognitive deficits are as severe in other psychotic disorders as in schizophrenia. To address this question, we administered our version of the AX-CPT to patients with a range of psychotic disorders (a) at an index assessment, when they presented for the first time for psychiatric care and were unmedicated and acutely ill; and (b) at a 4-week follow-up, when all patient participants were medicated and less acutely ill. Testing first-episode medication-naive individuals with psychotic disorders other than schizophrenia provides an important comparison group for the participants with schizophrenia for several reasons: (a) They control for medication status (all unmedicated patients had an index testing) and chronicity (all patients were having their first contact with psychiatric services), and (b) they allow us to assess the relationship of specific cognitive deficits to diagnoses versus the presence of psychotic symptoms that may occur in a number of different disorders.

Method

Participants

At the index assessment, participants included 72 healthy controls, 49 individuals with either schizophrenia ($n = 41$) or schizoaffective disorder ($n = 8$) on the basis of full diagnostic assessment and at 6-month follow-up, and 30 individuals with psychotic disorders other than schizophrenia (13 with major depression with psychotic features, 4 with delusional disorder, 8 with psychotic disorder not otherwise specified [NOS], 2 with brief psychotic disorder, and 3 with bipolar I disorder). We chose to combine the individuals with schizophrenia and the individuals with

schizoaffective disorder because of prior research suggesting that these disorders share similar cognitive deficits (Gooding & Tallent, 2002). Among the patients with schizoaffective disorder, 7 had the depressed subtype and 1 had the bipolar subtype. Of these individuals, the following completed the 4-week assessment: 61 healthy controls, 42 patients with schizophrenia ($n = 37$) or schizoaffective disorder ($n = 5$), and 21 individuals with psychotic disorder other than schizophrenia (11 with major depression with psychotic features, 3 with bipolar I disorder, 3 with delusional disorder, 3 with psychotic disorder NOS, and 1 with brief psychotic disorder). Controls were recruited through local advertisements and were evaluated using the Non-Patient version of the *Structured Clinical Interview for DSM–III–R* (*SCID–III*; Spitzer & Williams, 1987). All patients were neuroleptic naive at the time of index assessment and were recruited if they were experiencing any type of psychotic symptom (i.e., hallucination, delusion, or thought disorder) and it was their first episode of psychiatric hospitalization or contact with outpatient psychiatric services. Patients were followed longitudinally, and their diagnosis was confirmed 6 months after their participation in the index assessment. Diagnoses were determined through a diagnostic conference that included information from the *SCID–III* administered by trained research personnel and a thorough chart review. In addition, the Brief Psychiatric Rating Scale (BPRS; Overall, 1974), the Global Assessment Scale, and the Scales for the Assessment of both Positive and Negative Symptoms (SANS, Andreasen, 1983a; SAPS, Andreasen, 1983b) were used to evaluate symptom severity (Table 1). Ratings were completed by trained research team members who regularly participated in evaluation sessions to ensure reliability. All ratings were made within 1 week of testing, and all raters were blind to the performance of participants in the tasks. Consistent with our previous work, scores on the three major factors (see Table 1) often found in these scales were used to describe the clinical state of the participants (Andreasen, Arndt, Alliger, Miller, & Flaum, 1995; Brekke, DeBonis, & Graham, 1994; Shatasel et al., 1992; Silver et al., 1993; Van der Does, Linszen, Dingemans, Nugter, & Scholte, 1993):

1. The Reality Distortion factor ($\alpha = .77$) includes grandiosity, suspiciousness, hallucinations, and unusual thought content from the BPRS and hallucinations and delusions from the SAPS.

2. The Disorganization factor ($\alpha = .65$) includes conceptual disorganization, mannerisms and posturing, and disorientation from the BPRS and attention, positive formal thought disorder, and bizarre behavior from the SAPS/SANS.

3. The Poverty Symptoms factor ($\alpha = .83$) includes emotional withdrawal, motor retardation, and blunted affect from the BPRS and anhedonia/asociality, avolition/apathy, alogia, and affective flattening from the SANS.

Participants were excluded for the following reasons: (a) age greater than 40 or less than 14 years, (b) WAIS–R Full Scale IQ below 70, (c) non-English native language, (d) lifetime diagnosis of substance dependence or substance abuse disorder within 6 months of testing, (e) neurologic disorders or family history of hereditary neurologic disorder, or (f) pregnancy. Additional exclusion criteria for potential controls included (a) any lifetime history of Axis I disorder or any first-order family history of a psychotic disorder or (b) treatment with any psychotropic medication within 6 months prior to testing. The groups did not differ significantly on age, $F(2, 148) = 0.41$, $p > .50$; gender, $\chi^2(2, N = 151) = 3.4$, $p > .19$; or years of parental education (as a proxy for socioeconomic status), $F(2, 148) = 0.11$, $p > .89$. The groups did differ significantly on personal education, $F(2, 148) = 10.2$, $p < .001$, with controls having higher education than both patients with schizophrenia and the psychotic comparison group, who did not significantly differ. Demographic and clinical characteristics of both groups are shown in Table 1. All participants signed informed consent forms in accordance with the University of Pittsburgh Institutional Review Board.

Tasks and Apparatus

In the AX-CPT task, sequences of letters were visually presented one at a time in a continuous fashion on a computer display. Subjects were instructed to make a positive response on target trials and a negative response otherwise. Target trials were defined as a cue–probe sequence in which the letter A appeared as the cue and the letter X appeared as the probe. The remaining letters of the alphabet served as invalid cues (i.e., cues that were not As) and nontarget probes (i.e., probes that were not Xs), with the exception of the letters K and Y, which were excluded because of their similarity in appearance to the letter X. Letter sequences were presented in pseudorandom order such that target (AX) trials occurred with 70% frequency and nontarget trials occurred with 30% frequency. Nontargets were divided evenly (10% each) among the following trial types: BX trials, in which an invalid cue (i.e., non-A) preceded the target; AY trials, in which a valid cue was followed by a nontarget probe (i.e., non-X); and BY trials, in which an invalid cue was followed by a nontarget probe. The

Table 1
Clinical and Demographic Characteristics

	Healthy controls		Patients with schizophrenia		Psychotic comparison group	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*
Age (in years)	23.8	5.5	24.9	6.9	24	7.9
Gender (% male)	61		67		47	
Parent's education (in years)	15.0	2.5	14.8	3.2	14.8	3.5
Education (in years)	15.1	2.4	13.0	2.8	13.2	3.4
Index assessment						
Global Assessment Scale			32.9	10.5	39.9	11.1
Disorganization Symptoms			11.9	4.0	10.1	3.6
Reality Distortion Symptoms			20.9	6.7	16.0	5.9
Poverty Symptoms			19.8	5.3	16.7	5.4
4-week assessment						
Global Assessment Scale			40.5	11.0	51.4	15
Disorganization Symptoms			10.1	4.2	7.2	2.5
Reality Distortion Symptoms			15.8	7.3	10.0	5.1
Poverty Symptoms			18.9	6.0	14.7	3.7

delay between cue and probe was manipulated so that half of the trials had a short delay and half had a long delay. On short-delay trials, the cue–probe interval was 1 s, and the intertrial interval was 5 s. On long-delay trials, the cue–probe interval was 5 s, and the intertrial interval was 1 s. Thus, the total trial duration was equivalent across conditions, providing a means of controlling for general factors that might affect performance (e.g., pace of the task, response frequency, or total time on task).

Stimuli were presented centrally, for a duration of 300 ms, in 24-point, uppercase Helvetica font. Subjects were instructed to respond to both cue and probe stimuli, pressing one button for targets and another button for nontargets (cues were always considered nontargets). Responses were recorded on a specially constructed button box connected to the computer, which recorded response choice and RTs with 1-ms accuracy. For right-handed individuals, responses were made with the middle (nontarget, middle button) and index (target, right button) fingers of the right hand. For left-handed individuals, responses were made with the middle (nontarget, middle button) and index (target, right button) fingers of the left hand. Subjects were allowed a total of 1,300 ms from stimulus onset in which to respond. Responses slower than this limit were not recorded and elicited feedback (a "bloop" sound) as a prompt to increase speed. The task was run on Apple Macintosh computers, using PsyScope software (Cohen, MacWhinney, Flatt, & Provost, 1993) for stimulus presentation and data collection.

Procedure

Participants were tested in a single testing session. For 21 of the participants (9 healthy controls, 10 patients with schizophrenia, and 2 with other psychotic disorders), conditions were run in 3 blocks of 100 trials, in which short- and long-delay trials were randomly intermixed, yielding a total of 150 trials each in the short- and long-delay conditions (105 AX, 15 BX, 15 AY, and 15 BY). For the remaining participants, conditions were run in 30 blocks of 10 trials each, with each block consisting of all short-delay or all long-delay trials (see later for reasons behind this change). The order of block (short vs. long delay) presentation was counterbalanced within and across participants with the constraint that within every 4 blocks, 2 blocks of short-delay and 2 blocks of long-delay trials were presented. The method of task presentation again yielded a total of 150 trials each in the short- and long-delay conditions. Prior to performance of the first task block, standardized instructions describing the task appeared on the computer, and the experimenter answered any remaining questions regarding the instructions. Participants were asked to respond as quickly as possible to each stimulus while maintaining accuracy. All participants were given practice with the task prior to administration of the experimental trials for that condition to ensure that subjects understood the task. A direct comparison of the two types of blocking procedures indicated that there were overall slightly more errors among those who received the mixed design (interleaved short- and long-delay trials), as compared with the blocked design, as well as somewhat faster overall RTs in the mixed as compared with the blocked design. However, design type did not interact with trial type or group for either errors or RTs. At 4 weeks, there were no significant main effects of or interactions with block type for either errors or RTs. Thus, data from each design type were combined for subsequent analysis.

Data Analysis

Data were analyzed using error rates (misses and false alarms), signal detection indices (d'), and RTs as the dependent measures of interest. The error data were normalized using an arcsine transformation (Neter, Wasserman, & Kutner, 1990). Median RTs were examined for correct responses only, unless otherwise noted. For the signal detection measures, a correction factor was applied in cases of a perfect hit rate (1.0) or false-alarm rate (0.0). This correction factor (hit rate $= 2^{-(1/N)}$; false alarm $= 1 - 2^{-(1/N)}$; N = number of target or nontarget trials) allows an unbiased estimation of d' in such cases (Nuechterlein, 1991). Instead of the traditional computation of d' (i.e., using hits and all false alarms), d' was computed using just BX false alarms. This measure, hereafter referred to as d' context, has been used in previous AX-CPT studies to provide a more specific index of sensitivity to context (Cohen et al., 1999; Servan-Schreiber et al., 1996).

Results

Index Assessment

Error rates. Error data (Table 2) were analyzed using a three-way analysis of variance (ANOVA), with diagnostic group (healthy controls, schizophrenia patients, psychotic patient controls) as a between-subjects factor, and delay (short, long) and trial type (AX, AY, BX, BY) as within-subjects factors. This ANOVA revealed main effects of diagnostic group, $F(2, 148) = 14.9$, $p < .001$, and trial type, $F(3, 444) = 60.3$, $p < .001$, modified by a Diagnostic Group × Trial Type interaction, $F(6, 444) = 4.5$, $p < .001$, and a Diagnostic Group × Trial Type × Delay interaction, $F(6, 444) = 3.5$, $p < .005$. Planned contrasts indicated that, as predicted, patients with schizophrenia made more AX, $F(1, 148) = 24.9$, $p < .001$, and BX errors, $F(1, 148) = 24.4$, $p < .001$, than healthy controls but not more AY errors, $F(1, 148) = 0.5$, $p > .50$. Further, as predicted, patients with schizophrenia made significantly more BX than AY errors, $F(1, 148) = 21.7$, $p < .001$, whereas BX and AY errors did not differ in healthy controls, $F(1, 148) = 0.0$, $p > .90$. However, the psychotic comparison group also made more AX, $F(1, 148) = 13.9$, $p < .001$, and BX, $F(1, 148) = 4.7$, $p < .05$, errors but not more AY errors, $F(1, 148) = 1.6$, $p > .20$, than healthy controls. Patients with schizophrenia and

Table 2
Index Testing: Error Rates and d' Context

	Healthy controls		Patients with schizophrenia		Psychotic comparison group	
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*
Short delay						
AX	.03	.03	.06	.06	.07	.09
AY	.08	.11	.11	.17	.13	.15
BX	.08	.10	.24	.25	.15	.18
BY	.005	.02	.05	.07	.02	.08
d' context	3.4	0.7	2.5	1.1	2.9	1.0
Long delay						
AX	.06	.07	.18	.16	.16	.17
AY	.07	.10	.07	.16	.08	.11
BX	.06	.08	.21	.24	.15	.16
BY	.004	.02	.02	.04	.02	.06
d' context	3.2	0.7	2.0	1.1	2.4	1.2

Note. Data are proportions of errors at the index assessment of context processing, using an AX version of the Continuous Performance Test. AX = an A cue followed by an X probe (target trial); BX = a cue that is any letter other than A or X, followed by an X probe (nontarget trial); AY = an A cue followed by a probe that is any letter other than A or X; BY = a cue that is any letter other than A or X, followed by a probe that is any letter other than A or X (nontarget trial). See text for their relationships to each other. d' context = signal detection index, using BX false alarms.

the psychotic comparison group did not differ significantly on either AX, $F(1, 148) = .3$, $p > .90$, or AY errors, $F(1, 148) = 0.4$, $p > .90$; however, there was a strong trend for patients with schizophrenia to make more BX errors than the psychotic comparison group, $F(1, 148) = 3.6$, $p = .06$. Planned contrasts to determine the source of the Group × Trial Type × Delay interaction indicated that, compared with healthy controls, patients with schizophrenia demonstrated a greater increase in AX errors from the short to the long delay, $F(1, 148) = 15.5$, $p < .001$. However, the psychotic comparison group also showed a greater increase in AX errors from the short to the long delay than did controls, $F(1, 148) = 7.1$, $p < .01$, an effect that did not differ significantly from patients with schizophrenia, $F(1, 148) = .42$, $p > .50$. In addition, the controls demonstrated a significant decrease in BX errors from the short to the long delay, $F(1, 148) = 5.7$, $p < .05$, and no change in AY errors from the short to the long delay, $F(1, 148) = 0.8$, $p > .40$. In contrast, patients with schizophrenia demonstrated a significant decrease in AY errors from the short to the long delay, $F(1, 148) = 4.6$, $p < .05$, but no significant change in BX errors, $F(1, 148) = 1.9$, $p > .15$. Similarly, the psychotic comparison group showed a marginally significantly decrease in AY errors from the short to the long delay, $F(1, 148) = 3.5$, $p = .07$, but no significant change in BX errors, $F(1, 148) = .13$, $p > .70$.

Reaction time. The RT data (Table 3) were analyzed using a three-way ANOVA, with group as a between-subjects factor, and delay and trial type as within-subjects factors. The ANOVA revealed main effects of diagnostic group, $F(2, 148) = 20.6$, $p < .001$; trial type, $F(3, 444) = 92.8$, $p < .001$; and delay, $F(1, 148) = 20.7$, $p < .001$, modified by a Diagnostic Group × Trial Type interaction, $F(6, 399) = 5.9$, $p < .005$. Planned contrasts indicated that healthy controls were faster than both the patients with schizophrenia (all ps $< .001$) and the psychotic comparison group (all ps $< .05$), who did not differ significantly from each other. However, as predicted, healthy controls were significantly slower on AY as compared with BX trials, $F(1, 148) = 13.1$, $p < .01$, whereas patients with schizophrenia were slower on BX as compared with AY trials, $F(1, 148) = 6.2$, $p < .05$. The psychotic comparison group also tended to be slower on BX as compared with AY trials, but this effect was not significant, $F(1, 148) = 0.8$, $p > .30$.

Table 3
Index Testing: Reaction Times

	Healthy controls		Patients with schizophrenia		Psychotic comparison group	
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*
Short delay						
AX	418	88	510	130	476	116
AY	566	82	629	114	606	122
BX	503	164	708	208	626	231
BY	407	79	511	135	478	105
Long delay						
AX	445	93	558	132	543	149
AY	572	79	651	108	616	116
BX	507	166	680	198	646	243
BY	448	85	541	97	492	102

Note. Data are reaction times (in milliseconds) at the index assessment of ontext processing, using an AX version of the Continuous Performance Test. AX = an A cue followed by an X probe (target trial); BX = a cue that is any letter other than A or X, followed by an X probe (nontarget trial); AY = an A cue followed by a probe that is any letter other than A or X; BY = a cue that is any letter other than A or X, followed by a probe that is any letter other than A or X (nontarget trial). See text for their relationships to each other. d' context = signal detection index, using BX false alarms.

d' context. The d' context data (see Table 2) were analyzed using a two-way ANOVA, with diagnostic group as a between-subjects factor and delay as a within-subjects factor. This ANOVA indicated main effects of diagnostic group, $F(2, 148) = 23.8$, $p < .001$, and delay, $F(1, 148) = 61.6$, $p < .001$, as well as a Diagnostic Group × Delay interaction, $F(2, 148) = 6.8$, $p < .01$. Both the patients with schizophrenia and the psychotic comparison group had lower d' contexts than healthy controls at both the short and long delays. All groups had lower d' contexts at the long compared with the short delay (all ps $< .05$). However, the Group × Delay interaction reflected the fact that the decrease in d' context as a function of delay was greater in the patients with schizophrenia, $F(1, 148) = 11.7$, $p < .001$, and the psychotic comparison group, $F(1, 148) = 6.4$, $p < .05$, as compared with the healthy controls.

4-Week Assessment

Error rates. The error data (Figure 1) were analyzed using a three-way ANOVA, with diagnostic group as a between-subjects factor, and delay and trial type as within-subjects factors. This ANOVA revealed a main effect of diagnostic group, $F(2, 121) = 7.1$, $p < .001$; trial type, $F(3, 363) = 55.3$, $p < .001$; and delay, $F(1, 121) = 5.3$, $p < .01$, modified by Group × Trial Type, $F(6, 363) = 4.8$, $p < .05$. Planned contrasts indicated that patients with schizophrenia continued to make more AX, $F(1, 121) = 39.56$, $p < .01$, and BX errors, $F(1, 121) = 9.5$, $p < .01$, than healthy controls at 4 weeks but did not make more AY errors, $F(1, 121) = .10$, $p < .40$. Further, as predicted, patients with schizophrenia made significantly more BX than AY errors, $F(1, 121) = 7.2$, $p < .05$, whereas BX and AY errors did not differ in healthy controls, $F(1, 121) = 1.5$, $p > .20$. Note that, at 4 weeks, there was evidence that this pattern of performance (consistent with a context-processing deficit) was selective to patients with schizophrenia as compared with the psychotic comparison group. For example, the psychotic comparison group made more AX errors than healthy controls ($p < .01$) but did not differ significantly from controls on either AY or BX errors (ps $> .15$). Further, AY and BX errors did not differ in the psychotic comparison group, $F(1, 121) = .03$, $p > .90$. In addition, the patients with schizophrenia made significantly more AX errors, $F(1, 148) = 3.8$, $p < .05$, than the psychotic comparison group, although AY and BX errors did not differ significantly between the two patient groups. The Group × Trial Type × Delay interaction was only significant at a trend level ($p = .11$). However, because we had a specific a priori hypothesis about the pattern of errors as a function of delay, we conducted planned contrasts that indicated that, compared with healthy controls, the patients with schizophrenia, $F(1, 121) = 14.2$, $p < .01$, and the psychotic comparison group, $F(1, 121) = 4.9$, $p < .05$, demonstrated a greater increase in AX errors from the short to the long delay. Further, patients with schizophrenia demonstrated a significant increase in BX errors from the short to the long delay, $F(1, 148) = 4.0$, $p < .05$, and a

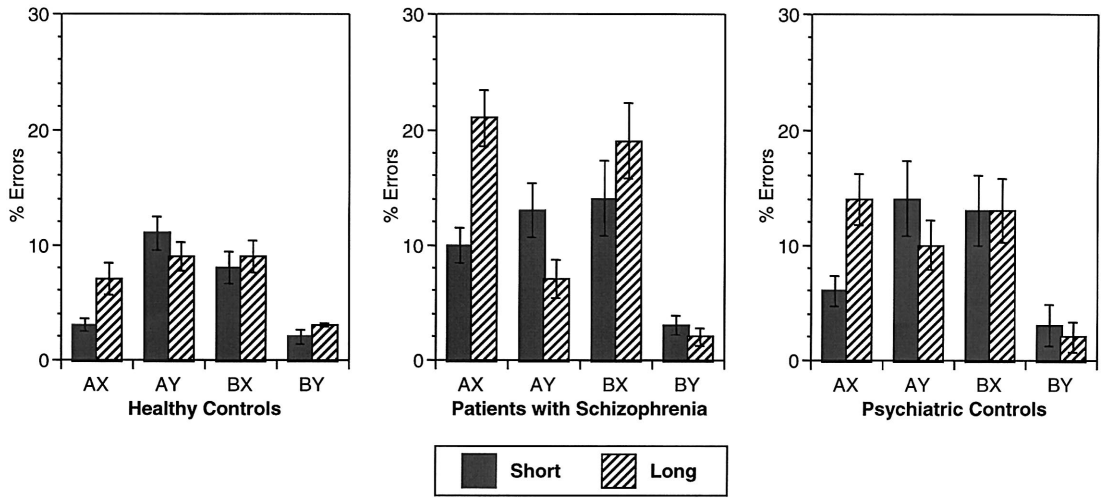

Figure 1. Graph plotting error rates (proportions) in all three groups at the 4-week follow-up assessment of context processing, using an AX version of the Continuous Performance Test. AX = an A cue followed by an X probe (target trial); BX = a cue that is any letter other than A or X, followed by an X probe (nontarget trial); AY = a cue followed by a probe that is any letter other than A or X; BY = a cue that is any letter other than A or X, followed by a probe that is any letter other than A or X (nontarget trial). See text for their relationships to each other.

significant decrease in AY errors from the short to the long delay, $F(1, 148) = 9.0$, $p < .05$. In contrast, neither the healthy controls nor the psychotic comparison group demonstrated any significant change in either AY or BX errors from the short to the long delay (all ps $> .20$).

Reaction time. The RT data (Figure 2) were analyzed using a three-way ANOVA, with diagnostic group as a between-subjects factor, and delay and trial type as within-subjects factors. The ANOVA revealed main effects of group, $F(2, 121) = 13.0$, $p < .001$; trial type, $F(3, 363) = 63.3$, $p < .001$; and delay, $F(1, 121) = 26.3$, $p < .001$, modified by a Diagnostic Group × Trial Type interaction, $F(6, 363) = 4.8$, $p < .005$. Planned contrasts indicated that the patients with schizophrenia were slower than the healthy controls as well as the psychotic comparison group for AX, BX, and BY trials (all ps $< .05$), with no significant differences between the psychotic comparison group and healthy controls (all ps $< .10$). For AY trials, RTs for patients did not differ from healthy controls and the psychotic comparison group, $F(1,$

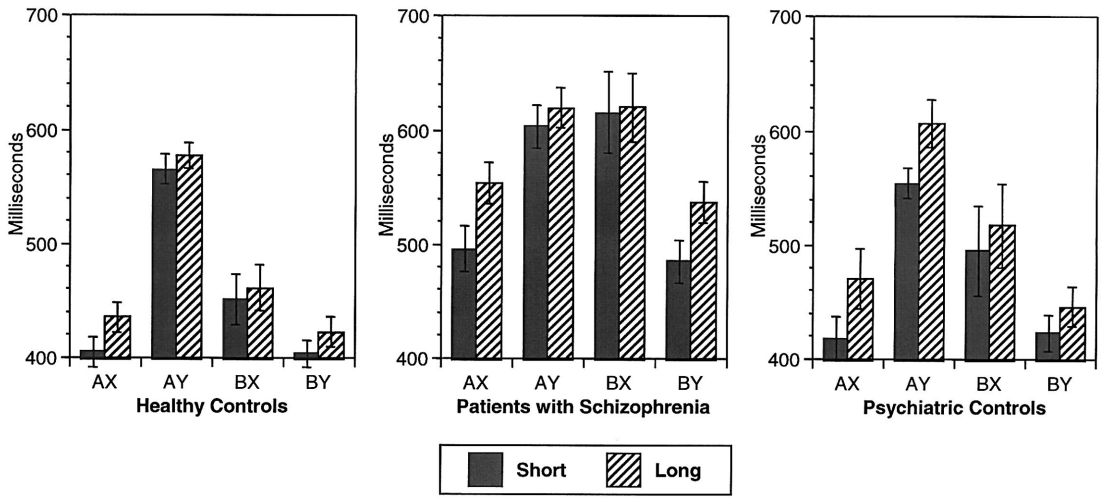

Figure 2. Graph plotting reaction times for short and long delays in all three groups at the 4-week follow-up assessment of context processing, using an AX version of the Continuous Performance Test. AX = an A cue followed by an X probe (target trial); BX = a cue that is any letter other than A or X, followed by an X probe (nontarget trial); AY = an A cue followed by a probe that is any letter other than A or X; BY = a cue that is any letter other than A or X, followed by a probe that is any letter other than A or X (nontarget trial). See text for their relationships to each other.

121) = 2.2, $p > .20$, suggesting that they were not experiencing the same level of context-induced slowing. Consistent with this hypothesis, at 4 weeks, both healthy controls and the psychotic comparison group were significantly slower on AY as compared with BX trials ($p < .001$), whereas the patients with schizophrenia were as slow on BX as on AY trials ($p < .50$). Further, the RT difference between AY and BX trials was significantly greater in both the healthy controls, $F(1, 121) = 20.0$, $p < .001$, and the psychotic comparison group, $F(1, 121) = 5.0$, $p < .05$, as compared with the patients with schizophrenia. In addition, a two-way ANOVA specifically comparing only psychotic patient controls and patients with schizophrenia on AY and BX RTs revealed a significant Group × Trial Type interaction, $F(1, 61) = 4.0$, $p = .05$, reflecting the fact that AY RTs were slower than BX RTs in the psychotic comparison group but not among the individuals with schizophrenia. The Group × Trial Type × Delay interaction was not significant.

d′ context. The *d′* context data (Table 4) were analyzed using a two-way ANOVA, with diagnostic group as a between-subjects factor, and delay as a within-subjects factor. This ANOVA indicated main effects of diagnostic group, $F(2, 121) = 19.0$, $p < .001$, and delay, $F(1, 121) = 56.4$, $p < .001$, as well as a Diagnostic Group × Delay interaction, $F(2, 121) = 5.5$, $p = .05$. Both patients with schizophrenia and the psychotic comparison group had lower *d′* contexts than healthy controls at both the short and long delays, and all groups had lower *d′* contexts at the long compared with the short delay. However, the decrease in *d′* context as a function of delay was significantly greater in patients with schizophrenia than in healthy controls, $F(1, 121) = 11.0$, $p < .01$, with a trend to also be greater than in the psychotic comparison group, $F(1, 121) = 3.1$, $p = .08$. The healthy controls and the psychotic comparison group did not differ in the magnitude of the delay effect, $F(1, 121) = .6$, $p > .20$.

Change From Index to 4 Weeks

The analyses reported here suggest that context-processing deficits in the psychotic comparison group improved from index to 4 weeks (particularly in RTs and the delay effect in *d′* context) but did not change in patients with schizophrenia. To address this question more directly, we conducted three-way ANOVAs separately for each group, with assessment (index, 4-week), delay (short, long), and trial type (AX, AY, BX, BY) as factors. For errors, healthy controls demonstrated a significant main effect of assessment, $F(1, 57) = 5.1$, $p < .05$, with slightly more errors overall at 4 weeks. Healthy controls also showed a significant Assessment × Delay × Trial Type interaction, $F(3, 171) = 2.9$, $p < .05$. At the short delay, healthy controls showed equal AYand BX errors at index but more AY than BX errors at 4 weeks. Neither the psychotic comparison group nor the patients with schizophrenia demonstrated any main effects of assessment or any interactions with assessment for errors (all *p*s > .15). For RTs, healthy controls demonstrated a significant Assessment × Trial Type interaction, $F(3, 171) = 5.1$, $p < .05$, such that RTs for AX, AY, and BY trials did not differ between index and 4 weeks, whereas BX RTs were significantly faster at 4 weeks. The psychotic comparison group also demonstrated a significant main effect of assessment, $F(1, 19) = 18.2$, $p < .001$, and a significant Assessment × Trial Type interaction, $F(3, 57) = 2.9$, $p < .05$. The psychotic comparison group was faster overall at 4 weeks as compared with index, with a disproportionate speeding of RTs on BX trials (similar to healthy controls). The patients with schizophrenia did not demonstrate any main effects of assessment or interactions with assessment for RTs (all *p*s > .15). Thus, these analyses confirm that context-processing deficits improved in the psychotic comparison group from index to 4 weeks (at least with regard to RT) but not in the patients with schizophrenia.

As described in the Method section, the patient control group included individuals with both psychotic mood disorders and nonmood psychotic disorders. An ANOVA on errors comparing these two groups, with both trial type and delay as within-subjects factors, indicated a main effect of group, $F(1, 29) = 4.8$, $p < .05$. Individuals with psychotic mood disorders made more errors overall than individuals with nonmood psychotic disorders, but there were no other significant interactions between group and any other factor (all *p*s > .60). No main effects of group or interactions with group were found for RTs or *d′* context. At the 4-week assessment, there were no significant main effects of group (psychotic mood disorder vs. nonmood psychotic disorder) or interactions involving group for errors, RT, or *d′* context (all *p*s < .20).

Table 4
Four-Week Testing: d′ Context

	Healthy controls		Patients with schizophrenia		Psychotic comparison group	
d′ context	*M*	*SD*	*M*	*SD*	*M*	*SD*
Short delay	3.4	0.7	2.6	1.0	2.9	0.9
Long delay	3.1	0.9	1.9	0.9	2.4	0.9

Note. Data are proportions of errors at the 4-week follow-up assessment of context processing, using an AX version of the Continuous Performance Test. *d′* context = signal detection index.

Clinical Symptoms

We next examined the relative severity of clinical symptoms between groups as well as change in symptoms from index to 4 weeks. Compared with the psychotic comparison group, patients with schizophrenia (Table 1) had significantly higher levels of reality distortion, $t(77) = 3.2$, $p < .01$, and poverty symptoms, $t(77) = 2.5$, $p < .05$, and a trend toward higher disorganization symptoms, $t(77) = 1.9$, $p = .06$, at index. Similarly, at 4 weeks, the patients with schizophrenia again had higher levels of disorganization, $t(60) = 2.8$, $p < .05$; reality distortion, $t(60) = 3.2$, $p < .05$; and poverty symptoms, $t(60) = 2.8$, $p < .05$. Patients with schizophrenia demonstrated a significant decrease in disorganization, $t(38) = 4.7$, $p < .001$, and reality distortions symptoms, $t(38) = 5.0$, $p < .001$, from index to 4 weeks and a trend-level change in poverty symptoms, $t(38) = 1.8$, $p = .07$. The psychotic comparison group also demonstrated significant decreases in disorganization, $t(19) = 3.6$, $p < .01$; reality distortion symptoms, $t(19) = 4.4$, $p < .01$; and poverty symptoms, $t(19) = 3.0$, $p < .01$, from index to 4 weeks.

We next examined the relationships between performance on the AX-CPT and clinical symptoms in each patient group. On the basis of prior research (Cohen et al., 1999), we specifically exam-

ined BX errors at the short and long delays as well as d' context at the short and long delays. Consistent with our prior research, disorganization symptoms among patients with schizophrenia at index were strongly associated with performance on the AX-CPT (see Table 5) at both the short and long delays, whereas the severity of reality distortion symptoms was unrelated to AX-CPT performance. However, poverty symptoms among patients with schizophrenia were also strongly correlated with AX-CPT performance, again at both the short and long delays. Using Z tests for comparing correlated correlation coefficients (Meng, Rosenthal, & Rubin, 1992), we found that all of the AX-CPT performance indices were significantly more correlated with disorganization symptoms than with reality distortion symptoms (all Zs > 2.1, all ps $< .05$) among the patients with schizophrenia at index, with the exception of BX errors at the short delay ($Z = 1.5$, $p = .08$). In addition, among the patients with schizophrenia, all of the AX-CPT performance indices were significantly more correlated with poverty symptoms than with reality distortions symptoms (all Zs > 1.7, all ps $< .05$).

Note that among the psychotic comparison group at index, disorganization symptoms were not significantly associated with performance on most of the AX-CPT indices, with the exception of d' context at the long delay (see Table 4). In addition, unlike for the patients with schizophrenia, poverty symptoms were only significantly correlated with d' context at the short delay. Similarly to patients with schizophrenia, reality distortion symptoms were not correlated with AX-CPT performance. At 4 weeks, none of the clinical symptoms ratings were significantly correlated with AX-CPT performance among either the patients with schizophrenia or the psychotic comparison group ($-.17 < r$s $< .19$).

Medication Status

As described earlier, the psychotic comparison group demonstrated more evidence of improvement at 4 weeks than did the patients with schizophrenia. One concern that might be raised about this result is that it could be influenced by differences in the types of medications that the individuals in the different groups were taking at the 4-week testing. Among the individuals with schizophrenia, 17% were not taking any medications, 6% were taking typical antipsychotics, and 77% were taking atypical antipsychotics. Among the individuals in the psychotic comparison group, 32% were not taking any medications, 5% were taking typical antipsychotics, and 63% were taking atypical antipsychotics. These percentages did not differ significantly across groups, $\chi^2(2, N = 73) = 1.5$, $p > .70$. Only one individual was taking an anti-Parkinsonian agent (who had a diagnosis of schizophrenia). There was a trend for group differences in the percentage of individuals taking antidepressants, $\chi^2(1, N = 73) = 3.1$, $p = .08$ (11% of the individuals with schizophrenia, 0% of the individuals with other psychotic disorders, and 29% of the individuals in the psychotic comparison group).

Completers Versus Noncompleters

Not all participants who provided data at index also provided data at 4 weeks. To determine whether there were systematic differences at index between those who did or did not provide data at 4 weeks, we conducted ANOVAs separately for each group, with retention (4-week, no 4-week), trial type, and delay as factors. There were no significant main effects of retention, or interactions of retention and any other factor, for any of the three groups for either accuracy or RT (all ps $> .20$). Those who did and did not provide data at 4 weeks were not different on age or parental education for either the healthy controls or the psychotic comparison group (all ps $> .30$), although the patients with schizophrenia who did not provide 4-week data tended to be somewhat older than those who did ($M = 28.6$ vs. $M = 23.2$), $t(47) = 2.7$, $p = .02$. The severity of disorganization, $t(29) = 1.3$, $p > .15$, and reality distortion symptoms, $t(29) = 0.1$, $p > .90$, at index did not differ for those with and without 4-week data among the psychotic comparison group, although poverty symptoms tended to be somewhat higher among those with 4-week data, $t(27) = 1.7$, $p = .09$. Those without 4-week data among the patients with schizophrenia had lower ratings of disorganization symptoms at index ($M = 10.1$ vs. $M = 12.7$), $t(47) = 2.1$, $p = .03$, but did not differ on poverty, $t(47) = 1.4$, $p > .15$, or reality distortion symptoms, $t(47) = 1.3$, $p > .15$.

Discussion

The results of the current study provide mixed evidence for the specificity of context-processing deficits to medication-naive, first-episode patients with schizophrenia. The results indicate that

Table 5
Correlations Between Clinical Symptoms and AX-CPT Performance at Index

	BX errors		d' context	
Group/factor	Short delay	Long delay	Short delay	Long delay
Patients with schizophrenia				
Disorganization Symptoms	.38**	.38**	−.41**	−.39**
Poverty Symptoms	.40**	.49**	−.28	−.30*
Reality Distortion Symptoms	.11	.003	.04	.18
Psychotic comparison group				
Disorganization Symptoms	.08	.27	−.20	−.36*
Poverty Symptoms	.32	.02	−.35*	.13
Reality Distortion Symptoms	.07	.08	−.24	−.18

Note. AX-CPT = AX version of the Continuous Performance Test.
* $p < .05$, two tailed. ** $p < .01$, two tailed.

during a first acute psychotic episode, when not medicated, both patients with schizophrenia and patients with other psychotic disorders demonstrated a pattern of behavioral performance on the AX-CPT consistent with a deficit in context processing. For example, both the patients with schizophrenia and the psychotic comparison group demonstrated more BX errors than the healthy controls (the type of errors thought to reflect a failure to represent context) but did not make more AY errors (the type of error thought to be caused by intact context representation). In addition, both patients groups demonstrated reduced d' context compared with healthy controls, with a larger decrease in d' context from the short to the long delay, also consistent with a deficit in context processing. Further, RTs among controls were significantly slower for AY than BX trials, whereas AY and BX RTs did not differ among either patient group. At 4 weeks, patients with schizophrenia continued to display this same pattern of performance deficits. However, at that point, the psychotic comparison group was performing more like the healthy controls. For example, at 4 weeks, the psychotic comparison group did not differ from the healthy controls on either BX or AY errors and demonstrated significantly slower AY than BX RTs. Thus, at index, there was little evidence for specificity of context processing to schizophrenia per se, whereas at 4 weeks, evidence for specificity to schizophrenia began to emerge.

There are several possible explanations for this pattern of performance as a function of time in the patients with schizophrenia and the psychotic comparison group. One possible explanation is that deficits in context processing are related to the presence of specific symptoms (i.e., disorganization, poverty), irrespective of diagnosis. As such, both the patients with schizophrenia and the psychotic comparison group may have shown evidence of impaired context processing at index because both groups displayed high levels of disorganization symptoms. However, the psychotic comparison group may have begun to display better AX-CPT performance than patients with schizophrenia at 4 weeks as their symptoms resolved more quickly than those of the patients with schizophrenia. This explanation would be consistent with our previous findings that patients with nonpsychotic major depression do not show evidence of context-processing deficits because these patients tend to have few disorganization symptoms (although they do show some poverty symptoms; Cohen et al., 1999).

However, several aspects of the current results are not consistent with this hypothesis. First, at index, the patients with schizophrenia and the psychotic comparison group did not show any significant differences in AX-CPT performance. However, the patients with schizophrenia had significantly higher levels of all symptom types than the psychotic comparison group. If AX-CPT performance deficits were determined only by the severity of either disorganization or poverty symptoms, one might have expected the patients with schizophrenia to display worse AX-CPT performance than the psychotic comparison group, even at index. Second, disorganization symptoms improved significantly from index to 4 weeks in patients with schizophrenia, but AX-CPT performance showed no change. Again, if AX-CPT performance were specifically related to the severity of disorganization symptoms, one would have expected to see improvement in AX-CPT performance among the patients with schizophrenia that paralleled the improvement in disorganization symptoms. We note, however, that unlike our previous work, AX-CPT performance was significantly related to poverty symptoms in both the patients with schizophrenia and the psychotic comparison group. However, at 4 weeks, the severity of AX-CPT deficits was not related to the severity of poverty or disorganization symptoms. The lack of correlation with disorganization symptoms among patients with schizophrenia at 4 weeks may be related to a reduced range of disorganization symptoms as patients improve. However, poverty symptoms did not improve among patients with schizophrenia at 4 weeks. Thus, such an explanation for a lack of AX-CPT correlations with poverty symptoms at 4 weeks seems less likely. Taken together, these results are not consistent with the hypothesis that context-processing deficits primarily reflect the severity of specific symptom dimensions, irrespective of diagnosis.

An alternative explanation for the pattern of results is that context-processing deficits represent a different aspect of the illness in patients with schizophrenia as compared with patients with other psychotic disorders. For example, Nuechterlein et al. (1992) have distinguished among three types of abnormalities that can be present in an illness: (a) episode indicators (processes that are impaired during acute psychotic episodes but unimpaired during clinical remission); (b) mediating vulnerability factors (processes that are impaired, even during clinical remission, but are further worsened during acute psychotic episodes); and (c) stable vulnerability indicators (processes that are impaired during clinical remission and do not worsen further during acute psychotic episodes). The pattern of AX-CPT performance shown by individuals with psychotic disorders other than schizophrenia suggests that context-processing deficits are episode indicators in these disorders. These patients displayed impaired context processing during acute psychotic episodes but began to perform much more like healthy controls as their symptoms remitted. In contrast, the pattern of AX-CPT performance shown by individuals with schizophrenia suggests that context-processing deficits reflect either stable or mediating vulnerability factors in schizophrenia. Patients with schizophrenia displayed impaired context processing at index and 4 weeks, even though they displayed a significant reduction in psychotic symptoms at 4 weeks. The hypothesis that cognitive deficits are stable or mediating vulnerability factors in schizophrenia is consistent with prior work by Nuechterlein et al. (1992). These researchers have shown that impairments on a 10-letter span of apprehension test and a degraded CPT reflect stable vulnerability indicators in schizophrenia, whereas performance on a different version of the AX-CPT (with a low target percentage) reflects a mediating vulnerability factor.

The current data are consistent with the hypothesis that context-processing deficits represent a stable or mediating vulnerability factor in schizophrenia but an episode indicator in other psychotic disorders. However, further research is needed to confirm this hypothesis. Specifically, performance on the AX-CPT at additional time points should be examined in these individuals to further clarify task performance as a function of clinical state. Such information would determine whether AX-CPT performance becomes identical to that of healthy controls among patients with psychotic disorders other than schizophrenia as they fully recover from their psychotic symptoms (assuming this does occur). Such a result would confirm that context-processing deficits represent an episode indicator in these individuals. Further, such information would help determine whether context-processing deficits in schizophrenia represent a stable vulnerability factor or a mediating

vulnerability factor. If AX-CPT performance does eventually improve in patients with schizophrenia as their symptoms resolve further, then such a result would suggest that context-processing deficits represent a mediating vulnerability factor. However, if AX-CPT performance remains consistently impaired, even in those individuals with a full remission of psychotic symptoms, then context-processing deficits likely represent a stable vulnerability factor. We are currently collecting such data, which we hope will shed further light on these critical issues.[1]

[1] One patient with schizophrenia and one patient control were missing clinical ratings at the 4-week follow-up.

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