Published in final edited form as:
J Abnorm Psychol. 2008 May ; 117(2): 342–354. doi:10.1037/0021-843X.117.2.342.

Context Processing in Schizotypal Personality Disorder: Evidence of Specificity of Impairment to the Schizophrenia Spectrum

Margaret M. McClure,
VA VISN-3 Mental Illness Research, Education and Clinical Center, Bronx, New York, and Department of Psychiatry, Mt. Sinai School of Medicine

Janine D. Flory,
Department of Psychiatry, Mt. Sinai School of Medicine and Department of Psychology, Queens College of the City University of New York.

Deanna M. Barch,
Departments of Psychology and Psychiatry, Washington University

Philip D. Harvey, and
VA VISN-3 Mental Illness Research, Education and Clinical Center, Bronx, New York, and Department of Psychiatry, Mt. Sinai School of Medicine

Larry J. Siever
VA VISN-3 Mental Illness Research, Education and Clinical Center, Bronx, New York, and Department of Psychiatry, Mt. Sinai School of Medicine

Abstract

Working memory abnormalities, which are particularly pronounced on context processing tasks, appear relatively specific to schizophrenia spectrum illnesses compared with other psychotic disorders. However, the specificity of context processing deficits to schizotypal personality disorder (SPD), a prototype of schizophrenia, has not been studied. The authors administered 3 versions of the modified AX Continuous Performance Test and an N-back working memory test to 63 individuals with SPD and 25 with other personality disorders, as well as 42 healthy controls. For the AX Continuous Performance Test standard and degraded versions, there was a significant Trial Type × Delay × Group interaction, as SPDs made significantly more errors reflecting poor maintenance of context and fewer errors reflecting good maintenance of context. SPDs also demonstrated poor performance on the N-back, especially at the 2-back condition. Context processing errors and N-back accuracy scores were related to disorganization symptoms. These findings, which are quite similar to those previously reported in patients with schizophrenia, suggest that context processing deficits are specific to the schizophrenia spectrum and are not a reflection of overall psychopathology.

Correspondence concerning this article should be addressed to Margaret M. McClure, VA VISN-3 Mental Illness Research, Education and Clinical Center, James J. Peters Veterans Affairs Medical Center, 130 West Kings-bridge Road, Bronx, NY 10468. Margaret.McNamara@mssm.edu.

Keywords

schizotypal personality; context processing; cognition; schizophrenia spectrum; personality disorders

Cognitive limitations are a core component of schizophrenia and other schizophrenia spectrum disorders, frequently preceding the onset of the illness and persisting even after the remission of most other symptoms (Bowie & Harvey, 2005; Heinrichs, 2005). Furthermore, cognitive deficits predict poorer medication (Burton, 2005) and overall treatment adherence (Prouteau et al., 2005), reduced everyday living and social skills (Bowie & Harvey, 2005), and increased tendency for relapse in first-episode patients (Chen et al., 1998). Individuals with schizophrenia demonstrate mild to moderate impairment across a broad range of neuropsychological abilities, with what appears to be more severe impairment in tasks related both to working and episodic memory and to executive functioning (Green, 2006; Mishara & Goldberg, 2004). Although these cognitive limitations in schizophrenia tend to be pervasive in nature, affecting many areas of neuropsychological functioning (Davidson et al., 1995; Heaton et al., 2001; Saykin et al., 1994), new research and theory suggest that working memory may be a core deficit that underlies some of the other cognitive impairments in schizophrenia (Cohen & Servan-Schreiber, 1992; Silver, Feldman, Bilker, & Gur, 2003), as well as other spectrum disorders, such as schizotypal personality disorder (SPD).

SPD is a schizophrenia spectrum disorder closely linked with the symptom profile of schizophrenia (Siever, 1995). In addition to demonstrating characteristic clinical symptoms, SPD patients demonstrate cognitive impairment in several ability areas, such as executive functioning (Diforio, Walker, & Kestler, 2000), verbal learning and abstraction (Bergman et al., 1998; Voglmaier, Seidman, Salisbury, & McCarley, 1997), recognition memory (Cadenhead, Perry, Shafer, & Braff, 1999), visual perception and spatial working memory (Farmer et al., 2000; Park, Holzman, & Lenzenweger, 1995; Roitman et al., 2000), cognitive inhibition (Beech, Baylic, Smithson, & Claridge, 1989; Moritz & Mass, 1997), dual-task information processing (Harvey, Reichenberg, Romero, Granholm, & Siever, 2006; Moriarty, Harvey, Granholm, Mitropoulou, & Siever, 2003), and sustained attention (Roitman et al., 1997). Although the profile of cognitive deficits in SPD is similar in scope to that seen in schizophrenia, the severity of impairment in SPD is generally less. However, relative to healthy controls (HCs), individuals with SPD have demonstrated impairments on tasks of working memory comparable with what is seen in schizophrenia patients (Barch et al., 2004; Mitropoulou et al., 2005).

It has been proposed that a specific deficit in the ability to represent and maintain context information may help to explain deficits in working memory, as well as deficits in other cognitive domains, in the schizophrenia spectrum disorders (Cohen, Barch, Carter, & Servan-Schreiber, 1999). *Context information* is defined as information actively maintained in such a form that it can be used to mediate later task-appropriate behavior. *Context representations* can include task instructions, a specific prior stimulus, or the result of processing a sequence of prior stimuli. Representations of context are hypothesized to

support task-relevant information against interference (e.g., noise or competing processes) and decay over time. In this model, a single underlying mechanism, operating under different task conditions, can subserve 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). 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 this model, context representations accomplish the same effect by providing top-down support for task-relevant processes, allowing these to compete effectively against irrelevant ones. When a task involves a delay between a cue and a later contingent response, it is usually assumed that a working memory function is involved. In the context model, the mechanism used to represent context information is used to maintain task-relevant information against the interfering and cumulative effects of noise over time. 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 information. Cohen and colleagues have argued that disturbances in attention, working memory, and inhibition in schizophrenia can all be understood in terms of a deficit in context processing (Barch, Braver, Cohen, & Servan-Screiber, 1998; Barch et al., 2001; Braver, Barch, & Cohen, 1999; Braver & Cohen, 1999; Cohen et al., 1999; Cohen & Servan-Schreiber, 1992). Furthermore, they have hypothesized that the dorsolateral prefrontal cortex (DLPFC) is responsible for the processing of context, and that a disturbance in this mechanism underlies several of the cognitive limitations exhibited by individuals with schizophrenia (Barch et al., 2001; Cohen & Servan-Schreiber, 1992).

Although few studies have been specifically designed to determine whether individuals with SPD have a disturbance in context processing, there is some indirect evidence that they do. Individuals with SPD demonstrate increased N400s to congruent sentence endings or related words in semantic priming paradigms. As N400s are electroencephalogram potentials that are inversely related to the degree of semantic relatedness of a stimulus event, this suggests that individuals with SPD are not able to use the prior context (e.g., sentence or word) to prime or bias ongoing processing (Niznikiewicz et al., 1999, 2002). Additionally, amphetamine, which has been found to improve working memory and executive function in schizophrenia (Barch & Carter, 2005), has also been effective at improving performance in these domains in SPD (Kirrane et al., 2000; Siegel et al., 1996). Moreover, like schizophrenia patients (Weinberger, Berman, Suddath, & Torrey, 1992), individuals with SPD demonstrate underactivation of the prefrontal cortex (Koenigsberg et al., 2005), an area that is important for the performance of working memory tasks (Friedman, Temporini, & Davis, 1999) and tasks of context processing (Barch, Csernansky, Conturo, Snyder, & Ollinger, 2002; MacDonald, Pogue-Geile, Johnson, & Carter, 2003).

Furthermore, we (Barch et al., 2004) published a study of context processing in SPD patients and HC, using three different versions of the AX Continuous Performance Test (AX-CPT), a task specifically designed to assess context processing. During the AX-CPT, participants are presented with cue-probe pairs and are told to respond to an *X* (probe), but only when it

follows an *A* (cue). The task also includes three types of nontarget trials that allow one to selectively assess context processing deficits: AY trials (*A* cue followed by any letter other than *X*), BX trials (non-*A* cue followed by an *X* probe), and BY trials (non-*A* cue followed by a non-*X* probe). AX trials occur with high frequency (70%), creating two important response biases. First, this high AX frequency creates a bias to make a target response to any stimulus following an *A* cue (as a target *X* occurrence is highly likely following an *A* cue). In healthy individuals, maintenance of context is demonstrated by the tendency to make a false alarm after occurrence of the *A* cue (leading to increased AY errors), or a slowing of reaction times (RTs) on correct AY responses (as the prepotent bias to make a target response needs to be overcome). The second bias created by the high AX frequency is the tendency to make a target response to the *X* probe, as this is the correct response the majority of the time. On BX trials, maintenance of the context provided by the cue (non-*A*) is needed to prevent BX false alarms. Thus, on the AX-CPT, deficits in context processing are not indicated by an overall increase in any type of false alarm, but rather a specific pattern of errors (decreased AY and increased BX).

We (Barch et al., 2004) found that for the three AX-CPT tasks, patients with SPD manifested reduced performance consistent with abnormalities in context processing compared with HC. These included making more errors reflecting poor maintenance of context (BX errors) and fewer errors that reflect maintenance of strong contextual representations (AY errors). The magnitude of these impairments was substantial, with SPD participants performing more than one full standard deviation worse than HC participants on all context-relevant measures.

It is interesting to note that individuals with other psychotic disorders have not exhibited the same pattern of impaired context processing as that seen in individuals with schizophrenia. For example, Brambilla et al. (2007) found that individuals with bipolar disorder demonstrated some impairment on the AX-CPT, such as making more AX and BY errors compared with HC participants, although the errors of the bipolar participants were more generalized than that seen in schizophrenia. In addition, Nuechterlein (1991) found that individuals with schizophrenia performed worse than individuals with bipolar disorder on a degraded version of the AX-CPT. Furthermore, Barch, Carter, Mac-Donald, Braver, and Cohen (2003) found that individuals with schizophrenia exhibited impaired context processing following 4 weeks of medication treatment, whereas those with psychotic disorders other than schizophrenia and schizoaffective disorder did not, even though both groups were comparable in their baseline performance on the AX-CPT.

Although individuals with SPD exhibit impaired context processing compared with HCs, the specificity of these deficits to SPD has not been established using a nonschizophrenia spectrum comparison group. SPD has been characterized as a prototype for schizophrenia (Siever & Davis, 1991) and is universally accepted as a schizophrenia spectrum disorder. Thus, identifying an area of core impairment in schizophrenia that is shared by individuals with SPD lends further weight to the argument that individuals with SPD do in fact experience what Meehl termed *schizotypy*, or a schizoptypal personality organization resulting from the interaction of the schizotaxic individual's genes and his environment (Lenzenweger, 2006). Establishing the specificity of context processing deficits, which most

likely underlie many of the cognitive abnormalities of schizophrenia, to disorders within the spectrum could therefore have important implications for understanding the relationships between cognitive deficits and the schizophrenia disease process. This would also provide insight into why some schizotaxic individuals develop schizophrenia and others are spared.

Understanding context processing abnormalities of individuals with SPD has important clinical implications as well. Context processing abnormalities have been linked with the disorganized symptoms of schizophrenia (Barch et al., 2003; MacDonald, Carter, et al., 2005), symptoms that are also quite pronounced in SPD. Individuals with schizophrenia who exhibit impaired emotional context processing tend to misinterpret ambiguous situations as threatening (Green, Waldron, & Coltheart, 2007), as they are not able to effectively integrate details of context. Individuals with SPD are notorious for their inappropriate social interactions, and this aspect of the illness is one of the most debilitating of the disorder. Identifying a specific deficit in context processing abilities, and understanding its relationship to clinical symptoms, could lead to potential avenues of treatment approaches for such poor social skills.

The goal of the current study was to extend the examination of context processing deficits in individuals with SPD using two comparison groups: a group of individuals with other, nonschizophrenia spectrum personality disorders (OPD) and a group of HCs, with a much larger sample size than any other study to date. We administered three versions of the modified AX-CPT task: standard, interference, and degraded. Across all three versions of the task, we predicted that individuals with SPD would be unable to integrate contextual information, therefore making more BX errors and fewer AY errors than the other groups. In addition, we predicted that as delay increased from short to long, individuals in our OPD and HC groups would make more AY errors, whereas individuals with SPD would make fewer AY errors. The second version of the AX-CPT, an “interference” version, was designed to challenge the ability to maintain context. We predicted that the addition of interference should make it more difficult for individuals in both control groups to represent and maintain context, potentially leading them to perform more similarly to the SPD participants (i.e., increasing their BX errors, but not AY errors). For the SPD individuals, we predicted that the addition of interference would make their performance worse, but would not qualitatively change their performance pattern, since interference should simply augment an existing deficit in context processing. The third version of the AX-CPT, a “degraded” version, was designed to control for task difficulty. We predicted that the addition of degradation should make performance overall worse for both controls and individuals with SPD (i.e., making all error types worse), but should not qualitatively change the pattern of performance for either group. Additionally, we examined the relationship between performance on tasks specifically designed to assess context processing and performance on a task assessing working memory using an N-back working memory task, which may also depend on context processing. We predicted that individuals with SPD would also demonstrate impairment on this particular test of working memory. Finally, we predicted that, similar to what has been found in schizophrenia patients, context processing impairments would be related to clinical symptoms of disorganization in our SPD group.

Method

Participants

Participants were 63 individuals with Diagnostic and Statistical Manual of Mental Disorders (4th ed.; *DSM-IV*; American Psychiatric Association, 1994) SPD, 25 individuals with other, non-Cluster A, *DSM-IV* personality disorders (OPD; see Table 1) and 42 HCs. Participants ranged in age from 20 to 64. The individuals with SPD and OPD were ascertained either through recruitment by advertisements in local media outlets or flyers or from the outpatient clinics at the Bronx Veteran Affairs Medical Center. The HCs were recruited from the local community through advertisements or flyers. Participants were excluded for (a) meeting criteria for current (within 6 months of testing) substance abuse or dependence, (b) a positive urine toxicology screen, (c) a lifetime diagnosis of a psychotic disorder or bipolar I disorder, and (d) significant head trauma. Participants were assessed for Axis I psychopathology using the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1995), by a master's level or doctoral level interviewer who did not know the participants' cognitive task performance. In addition, participants were assessed for Axis II pathology using the Structured Interview for the *DSM-IV* Personality Disorders (SIDP; Pfohl, Blum, & Zimmerman, 1995). Consensus diagnoses were reached in a meeting of all raters with an expert diagnostician. OPD individuals were excluded from the current analyses if they met criteria for any Cluster A personality disorder (i.e., paranoid personality disorder, schizoid personality disorder) or if they met more than two criteria for SPD. HCs were excluded if they had either a self-reported personal or family history of schizophrenia, bipolar disorder or a psychiatric illness that required hospitalization or medical treatment, or a personal history of an Axis II disorder. In addition, overall psychopathology was assessed using the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962) and the Clinical Global Impression Scale (CGI; Guy, 1976). All participants signed informed consent forms in accordance with the Institutional Review Boards. None of the participants were taking psychotropic medication at the time of assessment.

Tasks and Apparatus

AX-CPT tasks—Participants performed three conditions of the AX-CPT: standard, interference, and degraded. In all three conditions, sequences of letters were visually presented one at a time in a continuous fashion on a computer display. Participants were instructed to make a positive finger press 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 *A*s) and nontarget probes (i.e., probes that were not *X*s), with the exception of the letters *K* and *Y*, which were excluded due to 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 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 4,900 ms. 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, total time on task). The task was presented in 4 blocks of 50 trials, all of which were either short (2 blocks) or long (2 blocks) delay trials, with the order of short and long delay blocks counterbalanced across participants.

Stimuli were presented centrally, for a duration of 300 ms, in 24-point uppercase Helvetica font. Participants 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 that recorded response choice and RT 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. Participants 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 for stimulus presentation and data collection (Cohen, MacWhinney, Flatt, & Provost, 1993).

The interference and degraded conditions of the AX-CPT were identical to the standard condition, as described above, except in the following respects. In the interference condition, distractor letters appearing in a different color (white) were presented in addition to the cue and probe letters. Participants were required to respond to the distractors to ensure encoding (by pressing the nontarget button), but were instructed to otherwise ignore them when monitoring for targets. During the short delay trials, the distractors occurred in the intertrial interval. On the long delay trials, the distractors occurred during the delay between the cue and the probe. During the delay period of every interference trial, three distractors were presented in sequence, each with a duration of 300 ms and an interstimulus interval of 1,000 ms. In the degraded condition, visual degradation was introduced by randomly removing (at each presentation) 85% of the pixels that make up each of the letters in the stimulus set. This level of degradation was determined through pilot study to produce approximately 75% accuracy in naming single letters.

N-back working memory task—The N-back is a commonly used measure of working memory (Braver et al., 1997; Casey et al., 1995; Cohen et al., 1996) that has been frequently shown to elicit performance deficits among individuals with schizophrenia and their unaffected relatives (Barch et al., 2002; Callicott et al., 2000, 2003; Egan et al., 2001; Menon et al., 2001; Perlstein, Carter, Noll, & Cohen, 2001). Materials for the N-back task were similar to those used by Braver et al. (1997). In the current study, participants observed letters presented on a computer screen one at a time. There were three conditions: (a) 0-back, (b) 1-back, and (c) 2-back. In the 0-back condition, participants responded to a single prespecified target letter (e.g., *X*). In the 1-back condition, the target was any letter identical to the one immediately preceding it (i.e., one trial back). In the 2-back condition, the target

was any letter identical to the one presented 2 trials back. Thus, working memory load is increased incrementally from the 0-back to the 2-back conditions.

Stimuli were presented as single letters appearing centrally in 24-point Helvetica font, white against a black background, sub-tending a visual angle of approximately 3°. All consonants of the alphabet were used as stimuli with the exception of *L* (because it is easily confused with the number 1) and *W* (because it is the only three syllable letter of the alphabet). Vowels were excluded. Further, the case of the presented lettered-stimuli changed randomly throughout the trials. Stimuli were presented in a pseudorandom sequence of consonants, randomly varying in case in order to prevent participants from relying on strategies of perceptual familiarity for responding. Stimuli were presented centrally on a controlled computer display for 500 ms. The interstimulus interval was 2,500 ms. Targets were presented on 33% of the trials. Conditions were presented in blocks of 25 trials, with three blocks at each load level (0-, 1-, 2-back) presented in a counterbalanced order.

Procedure

Participants were tested in a single testing session. Task order was counterbalanced across participants. Prior to performance of the first block of each task, standardized instructions describing the task appeared on the computer, and the experimenter answered any remaining questions regarding them. Participants were asked to respond as quickly as possible to each stimulus while maintaining accuracy. One full block of trials were then performed as practice prior to administration of the experimental trials for that condition. This ensured that participants understood the instructions and were performing the task appropriately.

Data Analysis

For the AX-CPT, error rates (misses and false alarms), signal detection indices (d'-context), and RTs for correct trials served as the dependent measures of interest. In particular, analyses focused on error rates for the two trials most related to effective context processing, BX and AY, for both the short and long delay intervals. For the N-back, accuracy scores were calculated for each participant across the three conditions (0-back, 1-back, 2-back). On the basis of an earlier factor analysis of schizotypal symptoms (Bergman et al., 1996), we calculated three cluster scores based on the number of *DSM-IV* criteria met for each patient —positive symptoms (Criteria 1, 2, 3, and 5), negative symptoms (Criteria 4, 6, and 7), and disorganized symptoms (Criteria 8 and 9)—and used these cluster scores for our correlational analyses.

Results

Demographic Information

Demographic and clinical characteristics of both groups are shown in Table 2. The groups differed significantly on age, with the HC group being younger, $F(2, 123) = 6.754$, $p < .01$, than both the SPD ($p < .01$) and OPD groups ($p < .01$), which did not differ from each other ($p < .70$). The groups also differed on education, $F(2, 114) = 6.551$, $p < .01$, with the HC group being significantly more educated than the SPD group ($p < .01$); the OPD group did not significantly differ on education from either the SPD ($p < .17$) or the HC ($p < .14$)

group. Paternal education followed the same pattern, $F(2, 105) = 4.430$, $p < .05$, as there was a significant difference between the HC group and the SPD group ($p < .01$); the OPD group did not differ from the SPD group ($p = .45$) or the HC group ($p = .13$). The groups did not differ in terms of maternal education, $F(2, 111) = 2.178$, $p = .12$. There were also significant group differences for Wechsler Adult Intelligence Scale—Revised (Wechsler, 1981) Vocabulary, $F(2, 111) = 3.414$, $p = .04$, and Block Design, $F(2, 111) = 6.6$, $p = .002$, as the SPD group performed significantly worse than both other groups on both measures. The OPD and HC groups did not significantly differ on either vocabulary ($p = .37$) or block design ($p = 1.0$) performance. Finally, the groups also differed in terms of the gender composition, as there were significantly more men in the SPD group, $\chi^2(2) = 17.2$, $p < .01$.

The OPD and SPD groups were also compared on symptom severity. The groups did not statistically differ in their Brief Psychiatric Rating Scale (Ventura, Lukoff, Nuechterlein, Liberman, & Green, 1993), $t(83) = 1.83$, $p = .07$, or Clinical Global Impressions Scale (Guy, 1976), $t(66) = 1.26$, $p = .21$, scores. In addition, more extensive demographic data were available for a subset of participants. This indicated that the SPD and OPD groups were also comparable in terms of the total number of hours worked per week, $t(32) = 0.003$, $p = .99$.

AX-CPT Results

Standard AX-CPT results—In the interest of space, and because our hypotheses focused on group differences, we focus on main effects of group or interactions with group in all of the analyses presented below. The error and RT data from the standard AXCPT were analyzed using three-factor analyses of variance (ANOVAs), with group (HC, OPD, SPD) as a between-subjects factor, and both delay (short, long) and condition (AX, AY, BX, BY) as within-subject factors. The ANOVA on the error data (see Table 3) revealed a significant main effect of group, $F(2, 127) = 90.944$, $p < .01$, a significant Group × Condition interaction, $F(6, 381) = 4.446$, $p < .01$, and a significant Group × Condition × Delay interaction, $F(6, 381) = 2.685$, $p < .015$. Planned contrasts indicated that, as predicted, the SPD individuals made significantly more BX errors than both the OPD ($p < .04$, short delay $d' = .44$, long delay $d' = .60$) and HC groups ($p < .04$, short delay $d' = .57$, long delay $d' = .61$), who were comparable with one another (see Table 4 for effect sizes). When the effect of delay was considered, the SPD individuals showed a decrease in AY errors from short to long, while the OPD and HC groups showed an increase, although only the HC demonstrated a significant change from short to long ($p < .05$). In addition, SPD individuals made significantly more BX than AY errors, $F(1, 127) = 18.28$, $p < .01$, while the other groups made more (or equal) AY than BX errors (though not significant).

For the RT data (see Table 3), the ANOVA revealed a significant Group × Condition interaction, $F(2, 118) = 19.54$, $p < .01$. Both the SPD and OPD individuals were slower than HC individuals for BX trials, but were faster for AY trials. In addition, AY RTs were significantly slower than BX RTs for HCs ($p < .001$), but not for the OPD or SPD groups. There was no significant main effect for group and no significant three-way interaction between group, condition, and delay.

The d'-context data were analyzed using a repeated-measures ANOVA, with delay (short, long) and group (HC, OPD, SPD) as factors. This analysis revealed a main effect of group,

$F(1, 125) = 9.87$, $p < .01$, and a Trend Level Group × Delay interaction, $F(1, 125) = 2.82$, $p = .06$. As shown in Table 3, SPD individuals showed overall lower d'-context compared with both HC and OPD groups ($p < .05$). In addition, SPD individuals showed a larger drop in d'-context from the short to long delay compared with HCs ($p < .05$), though not compared with OPDs. The HC and OPD groups did not differ significantly (ps < .2).

Degraded AX-CPT results—The error and RT data from the degraded AX-CPT were also analyzed using three-factor ANOVAs, with group (HC, OPD, SPD) as a between-subjects factor, and both delay (short, long) and condition (AX, AY, BX, BY) as within-subject factors. The error ANOVA (see Table 5) indicated a significant main effect of group, $F(2, 125) = 4.57$, $p < .01$. Interestingly, the Condition × Group interaction was not significant, $F(3, 123) = 0.92$, $p = .48$, but there was a significant three-way interaction between trial type, delay, and group, $F(6, 248) = 2.44$, $p < .04$. SPD participants made significantly more BX than AY errors at the long delay, $F(1, 125) = 16.9$, $p < .01$, and showed a trend for more BX than AY errors at the short delay, $F(1, 125) = 3.32$, $p = .07$. At the short delay, both the OPD and HC groups made numerically more BX than AY errors, but these differences were not significant (ps < .4). At the long delay, HC made more BX than AY errors and OPDs made more AY than BX errors, but these differences were again not significant (ps < .4). As shown in Table 5, for the HC and OPD groups, in the degraded AX-CPT, AY errors went up at the long delay, whereas for the SPD group, AY errors decreased significantly at the long delay, $F(1, 125) = 11.1$, $p < .01$.

For RTs (see Table 5), the ANOVA revealed no significant main effect of group, $F(2, 117) = 0.14$, $p = .87$. However, there was a significant Group × Condition interaction, $F(2, 117) = 15.17$, $p < .01$, but not a significant three-way interaction between group, condition, and delay. All three groups showed significantly slower AY than BX RTs (all ps < .05). However, as expected, the SPD participants were slower than the HCs in the BX condition ($p = .11$), and were faster in the AY condition ($p < .05$). The OPD individuals did not differ from either SPDs or HCs.

The d'-context data were analyzed using a repeated-measures ANOVA, with delay (short, long) and group (HC, OPD, SPD) as factors. This analysis revealed a main effect of group, $F(1, 125) = 4.45$, $p < .01$, but no significant Group × Delay interaction, $F(1, 125) = 1.5$, $p = .20$. As shown in Table 4, SPD individuals showed overall lower d'-context compared with both HCs ($p < .05$) and OPDs ($p = .06$). The HC and OPD groups did not differ significantly ($p = .60$).

Interference AX-CPT results—The error and RT data from the interference AX-CPT were analyzed using three-factor ANOVAs, with group (HC, OPD, SPD) as a between-subjects factor, and both delay (short, long) and condition (AX, AY, BX, BY) as within-subject factors. The error ANOVA (see Table 6) revealed a significant main effect of group, $F(2, 126) = 9.88$, $p < .01$, and a significant interaction between group and condition, $F(2, 126) = 4.32$, $p < .02$. Similar to the standard AX-CPT, the Group × Condition interaction reflected the fact that the SPD participants made significantly more BX errors than HC participants ($p < .01$, short delay $d' = .61$, long delay $d' = .42$), and exhibited a trend toward making more BX errors than OPD participants ($p = .07$, short delay $d' = .37$, long delay $d' = .$

29), but equal or fewer AY errors than both other groups. In addition, when collapsing across delay, SPD participants made significantly more BX than AY errors, $F(1, 126) = 5.09$, $p < .03$, whereas HC and OPD participants made more AY than BX errors (though not significant). There was no significant three-way interaction between group, delay, and condition.

The RT ANOVA (see Table 6) revealed no significant main effect of group, $F(2, 177) = 0.92$, $p = .40$, but a significant Condition × Group interaction, $F(2, 117) = 4.54$, $p < .01$. As can be seen in Table 6, all three groups had slower AY than BX RTs (all ps < .01). However, the Condition × Group interaction reflected the fact that the SPD individuals were slower than the HCs for BX trials ($p = .08$), though SPDs and OPDs did not differ on BX RTs. In addition, the RT difference between AY and BX trials was smaller for both SPD and OPD participants than for the HC group.

The d'-context data were analyzed using a repeated-measures ANOVA, with delay (short, long) and group (HC, OPD, SPD) as factors. This analysis revealed a main effect of group, $F(1, 125) = 414.9$, $p < .001$, but no significant Group × Delay interaction, $F(1, 125) = 1.4$, $p = .25$. As shown in Table 5, SPD individuals showed overall lower d'-context compared with both HC ($p < .01$) and OPD ($p < .01$). The HC and OPD groups did not differ significantly ($p = .13$).

Age, education, IQ, and gender contributions—As there were significant group differences between our groups for age, education, IQ, and gender, we sought to address the potential impact of these on the AX-CPT results through several analyses. To understand the effect of gender, we examined the impact of gender on the dependent variables of interest for our HC group. We found that very few of the variables significantly differed for men and women. In the standard condition, men made more BY errors following a short delay. For both the interference and degraded conditions, men made more BY and AY errors following a long delay. As AY errors are suggestive of intact context processing and group differences were not found for BY errors in our analyses, we concluded that gender was not accounting for the differences between our diagnostic groups.

To better understand the effects of age and education, we correlated these variables with our dependent variables of interest in our sample of HC. None of these variables were significantly correlated with age. Four variables in the standard condition (AX short, BY short, d'-context short, and d'-context long) and one variable in the degraded condition (BX long) were correlated with education. For three of these five variables, standard AX short, standard BY short, and degraded BX long, there were no differences between diagnostic groups in our analyses. For d'-context short and d'-context long, we regressed the scores of our HCs onto the education scores and then created standardized scores for our three groups. We found that the diagnostic group differences remained for the education-adjusted variables.

In addition, we selected a subsample of participants who were matched on both age and education and recomputed all of our analyses. All of the significant interactions remained

significant in this subsample, with the exception of the triple interaction of Group × Condition × Delay in the standard condition.

To better understand the impact of IQ, as measured by Wechsler Adult Intelligence Scale—Revised Vocabulary and Block Design performance, we correlated scores on vocabulary and block design with our dependent variables of interest (see Table 7). However, there were no significant correlations for either the HC or OPD groups. We did, however, enter these variables into all of our models as covariates. Again, we found that all of the significant interactions remained, with the exception of the triple interaction of Group × Condition × Delay in the standard condition. As almost all of our analyses remained significant for this subsample, we do not believe that differences in the context processing of our three groups was driven by differences in intelligence alone.

Cross-AX-CPT task comparisons—In order to understand group differences in performance across all three versions of our AX-CPT task, we calculated an overall d'-context score by averaging performance following long and short delay intervals for the standard, degraded, and interference conditions. We then computed a two-factor ANOVA, with group as a between-subjects factor, and the average d'-context score for each task version (standard, degraded, interference) as a within-subject factor. We found a significant main effect for group, $F(2, 122) = 11.68$, $p < .001$. As can be seen in Figure 1, individuals with SPD performed worse overall than both the HC (least significant difference [LSD] = .66, $p = .001$) and OPD (LSD = .48, $p = .006$) groups, who did not differ from one another (LSD = .18, $p = .31$) in their discrimination ability. In addition, we found a very large main effect for task version, $F(2, 244) = 144.47$, $p < .001$, as participants across the groups exhibited significantly worse performance when comparing the standard version with both the degraded (LSD = 1.21, $p < .001$) and the interference(LSD = .73, $p < .001$) versions of the task. In addition, across groups, participants performed significantly worse on the degraded version than they did on the interference version of the task (LSD = .48, $p < .001$). Furthermore, we found a significant interaction between group and task version $F(4, 244) = 2.74$, $p = .03$. To better understand the source of this interaction effect, we computed effect sizes (Cohen's d) comparing the SPD group with both of the other groups for the versions of the task. When the SPD and HC groups were compared, we found that the effect size for the interference task ($d = .95$) was larger than for both the standard task ($d = .81$) and the degraded task ($d = .53$). The same pattern held when the SPD and OPD groups were compared, as the effect size for the standard task ($d = .49$) and the degraded task ($d = .43$) were both smaller than the effect size for the interference task ($d = .60$). Thus, all groups demonstrated deterioration in their performance following the introduction of stimulus degradation and an interference stimulus.

However, the SPD group's performance was particularly worse than both of the other groups during the interference version of the task.

N-back Working Memory Test Results

The accuracy data from the N-back were analyzed using a two-factor ANOVA, with group as a between-subjects factor and condition (0-back, 1-back, 2-back) as a within-subject

factor. This ANOVA revealed main effects of group, $F(2, 121) = 10.86$, $p < .01$, and condition, $F(1, 121) = 92.94$, $p < .01$, as well as a significant Group × Condition interaction, $F(2, 121) = 8.72$, $p < .01$. As can been seen in Table 8, the SPD group did not differ from the OPD group at the 0-back condition ($p = .13$), but did show a trend for worse performance at the 1-back condition ($p = .06$). Additionally, the SPD group showed significantly impaired performance at the 2-back condition ($p < .01$) compared with the OPD group. Compared with the HC group, the SPDs performed significantly worse at both the 1-back ($p < .01$) and the 2-back ($p < .01$) conditions, but not the 0-back ($p = .32$) condition. The OPD and HC groups did not differ from each other on any variables (all ps > .70).

Cross-Task Correlations

In order to better understand the relationships between our tasks and specific symptoms of SPD, we correlated AX-CPT error rates for AY and BX trials, N-back accuracy scores, and scores on our symptom clusters for individuals in our SPD groups. Several cross-task correlations were significant (see Table 7), suggesting that there is a relationship between working memory, as assessed by the N-back, and context processing. In addition, for our SPD participants, scores on the disorganization symptom cluster were positively correlated with BX errors at both short ($r = .26$, $p = .04$) and long ($r = .32$, $p = .01$) delays during the standard AX-CPT task, whereas disorganization was negatively correlated with accuracy scores for both the 1-back ($r = –.26$, $p=.04$) and 2-back ($r = –.39$, $p=.002$) conditions of the N-back working memory test. These results suggest that greater disorganization was related to poorer performance on both context processing and working memory tasks. Positive and negative symptoms were not correlated with performance on either the AX-CPT or the N-back. Correlations with the positive symptom cluster ranged from .005 ($p = .97$) to .18 ($p = .17$), whereas for the negative symptom cluster they ranged from .007 ($p = .96$) to .12 ($p = .34$).

Discussion

The goal of the current study was to continue the evaluation of context processing deficits of individuals with SPD using a much larger sample, as well as a control group with substantial psychiatric symptoms. We predicted that individuals with SPD would demonstrate impairment in context processing in a similar manner to what we have observed in individuals with schizophrenia, and that this impairment would not be found in either the HC group or the psychiatric comparison group.

On our modified AX-CPT, context processing is best understood by the pattern of errors, rather than by the overall number of errors, that an individual makes. Specifically, a greater number of AY errors or a slowing of RTs for AY trials suggests intact context processing. In addition, individuals with intact context processing tend to make a smaller number of BX errors, or to demonstrate a decrease in RTs for BX trials. Impaired context processing, on the other hand, is indicated by the reverse pattern: greater numbers of BX and smaller numbers of AY errors. In addition, the introduction of an interference between the cue and probe generally elicits poorer performance in individuals with impaired context processing

following this pattern of fewer AY and more BX errors, whereas the introduction of stimulus degradation generally elicits more errors overall across all groups.

On the standard version of the AX-CPT, individuals with SPD clearly demonstrated impaired context processing. They made more BX than AY errors, suggesting poor maintenance of context, and demonstrated a decrease in AY errors from short delay to long delay, suggesting an inability to maintain contextual information over time. The SPD group also made significantly more BX errors overall than the other two groups. Individuals in our OPD and HC groups, on the other hand, did not exhibit this pattern. The HC group made more AY than BX errors and demonstrated an increase in AY errors from short to long. Although the OPD group exhibited similar numbers of AY and BX errors, they made substantially fewer BX errors than the SPD group and demonstrated an increase in AY errors from short delay to long delay, suggesting that although their performance was not perfect, they were able to use contextual information to influence their responses. When RTs were considered, the SPD group again displayed evidence of impaired context processing, as their RTs were faster than the HC group for AY trials but were slower for BX trials. However, the RTs of the SPD group were comparable with those of individuals in the OPD group, who also exhibited faster RTs on AY trials and slower RTs on BX trials than those in the HC group. When we considered discrimination, individuals in the SPD group demonstrated significantly poorer scores compared with both other groups and showed a larger drop in d'-context from short to long delay than the HC group.

For the interference task, which was designed to elicit poorer performance in all participants, but particularly for individuals with impaired context processing, our SPD group again demonstrated poor performance relative to the other groups. SPDs made more BX errors than both other groups, and made equal or fewer AY errors. Again, although the OPD and HC groups made more AY than BX errors, the SPD group made more BX than AY errors. Additionally, the SPDs demonstrated slower RTs than the HCs for BX trials and faster RTs than the HCs for AY trials, although the SPD and OPD groups performed similarly. As expected, all groups demonstrated more BX errors following a long delay (the condition in which interfering items were presented between the cue and the probe), although this pattern was even more striking for the SPD group, suggesting that they had even more difficulty maintaining the noncue in working memory than the other groups. Finally, individuals with SPD demonstrated significantly worse discrimination that both other groups.

For the degraded condition, we predicted that the introduction of the degradation would lead to a general decrease in performance for all groups. Consistent with this hypothesis, all groups made overall more errors in the degraded task than in standard AX-CPT, and this was true for all error types, including BY errors. For controls, the increase in errors was somewhat larger for BX than AY trials, which we had not predicted. However, the increase was even larger for BY trials, particularly at the long delay, which is consistent with the hypothesis that degradation induces a nonspecific decrement in performance. RTs were also somewhat slower overall for the degraded compared with standard AX-CPT, and this did not seem to differ by trial type, again consistent with a general decrement in performance. Although the introduction of degradation induced general performance decrements for all groups, the SPD participants still displayed a pattern of performance indicative of a deficit in

context processing. Specifically, the SPD group made more BX errors than the other groups at both the long and short delay, and fewer AY errors than both other groups at the long delay. For RTs, SPDs demonstrated faster RTs for AY trials and slower RTs for BX trials than the HCs, although not for the OPDs. When discrimination was considered, SPDs again did worse than both other groups.

When comparing across versions of the AX-CPT, we found that all three groups demonstrated poorer discrimination after the introduction of stimulus degradation. The introduction of an interference stimulus between cue and probe also resulted in lower discrimination for all three groups, although this effect was the most pronounced for the SPD participants. The interference version of the task is more difficult than the standard version, and differences between versions of the task may be due to greater discriminating power of the interference version. However, the degraded version of the task is also harder than the standard version, and post hoc comparisons suggested that it was also more difficult than the interference version for participants in all three groups. In addition, as shown in Figure 1, variance is at least as great in the degraded as the interference condition. Further, in pilot work we found that the reliability of the degraded and the interference tasks were similar in HCs. Taken together, such results suggest that discriminating power was at least as great in the degraded task as the interference tasks. Thus, we believe that the poor performance of the SPD group, compared with both the OPD and HC groups, during the interference task as compared with the degraded task reflects a core deficit in context processing that makes the introduction of an interference stimulus particularly challenging.

Additionally, we found that individuals with SPD demonstrated impaired performance on the N-back working memory test relative to both other groups. This impairment was greater at the most demanding condition, the 2-back interval, suggesting that these impairments in working memory worsen as demand increases. When we analyzed cross-task correlations, we found that for the SPD group, error rates for most of the AX-CPT variables were significantly correlated with accuracy data for all three conditions of the N-back. Furthermore, we found that within our SPD group, disorganized symptoms were related to performance on both the AX-CPT and the N-back, such that those individuals who demonstrated more disorganization performed worse on both the context processing and working memory tasks.

Thus, these results suggest that the deficits in context processing in errors exhibited during both the AX-CPT and the N-back are in fact present in individuals with SPD, compared with both healthy and psychiatrically comparable controls. As predicted, our HC group made very few errors overall, although they did tend to make AY errors reflecting healthy processing of context. The performance of our OPD group was not perfect, as would be expected with any psychiatric control group. For example, when we examined RTs, the performance of individuals with other personality disorders was more similar to the SPDs than to the HCs, though not in all conditions. This suggests that individuals with other personality disorders may exhibit some difficulty with context processing that requires the use of compensatory strategies, which take longer but appear effective, as their error rates are more similar to the HC. Additionally, analyses of effect sizes of the errors made by our three groups suggest a much larger effect for our SPD group than both other groups. Thus,

not only did the SPD participants make significantly more BX errors and fewer AY errors, the differences in these error rates were meaningful.

As predicted, the introduction of the interference led to increased BX errors at the long delay in all groups, though the effect of interference was greatest in the SPD group. We did find the predicted overall decrease in performance across groups with the introduction of the stimulus degradation, though the SPD group still showed evidence of impaired context processing, and not simply a generalized increase in errors, even in the degraded condition. In addition, symptoms of disorganization appeared to relate to impairments in context processing and working memory, a pattern that replicates similar findings in individuals with schizophrenia (MacDonald, Carter, et al., 2005).

One interesting finding of the current study was the important contribution of delay. Previous studies on context processing in schizophrenia have demonstrated that schizophrenia patients tend to make fewer AY and more BX errors at long delay compared with short delay, indicating that context processing deteriorated even further when individuals were asked to maintain the context representation for a longer duration. In our prior study of context processing in individuals with SPD (Barch et al., 2004), we only found a significant delay effect in the degraded condition, and it was not in the predicted direction. However, in the current study, we found significant Condition × Group × Delay interactions for both the standard and the degraded conditions in the predicted direction. Specifically, individuals with SPD demonstrated a decrease in AY errors from short to long delay, whereas the other two groups demonstrated an increase. These results may have differed from our previous study because of the increase in sample size (leading to more power to detect an interaction with delay) and the addition of a psychiatric control group that showed error patterns more similar to controls (again potentially enhancing power to detect interactions with delay).

One limitation of the current study is that we had significant group differences on our intelligence measures. Like individuals with schizophrenia, SPD participants in the current study were found to perform worse on measures of verbal and performance intelligence, an effect that is almost certainly related to their schizophrenia spectrum pathology. This makes it impossible for us to conclude with certainty that the SPD group's poor performance was not due to a generalized deficit in intellectual functioning. However, it is important to note that individuals with SPD did not make large numbers of BY errors, reflecting completely inaccurate responses, nor did they make large numbers of AX errors, reflecting a failure to accurately identify correct cue-probe pairs. Instead, they made many BX errors and very few AY errors, suggesting that they were able to understand the task and accurately respond to the correct probe; however, they were not able to effectively integrate the contextual information provided by the cue stimulus. This makes a generalized deficit explanation extremely unlikely. Further, it may be the case that the deficits in vocabulary acquisition and even block design scores may be the result of deficits in context processing or working memory (e.g., there is clear evidence that vocabulary acquisition is dependent on working memory; see deJong & Olson, 2004; Gathercole, Tiffany, Briscoe, Thorn, & ALSPAC Team, 2005). Thus, controlling or equating for IQ may eliminate precisely the variance in which we are interested.

These results of the current study again point to the importance of deficits in the maintenance of context information, as well as deficits in the initial representation of context, in schizophrenia spectrum disorders. They suggest that context processing deficits are in fact present in individuals with SPD, which leads to two important conclusions. First, context processing abnormalities appear to be a part of schizotaxic pathology, even in the absence of psychosis, suggesting that these abnormalities are a promising cognitive endophenotype for spectrum disorders. They also highlight an area of impairment that is crucial to understanding the pathology of SPD in its own right, as these impairments may relate to the chronic and debilitating social inappropriateness and interpersonal isolation of the disorder.

Acknowledgments

This research was supported by National Institute of Mental Health Grants MH 56140 (to Larry J. Siever) and MH 63116 (to Philip D. Harvey) and by the VA VISN-3 Mental Illness Research, Education and Clinical Center.

References

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed.. Author; Washington, DC: 1994.

Barch DM, Braver TS, Cohen JD, Servan-Screiber D. Context processing deficits in schizophrenia. Archives of General Psychiatry. 1998; 55:187–188.

Barch DM, Carter CS. Amphetamine improves cognitive function in medicated individuals with schizophrenia and in healthy volunteers. Schizophrenia Research. 2005; 77:43–58. [PubMed: 16005384]

Barch DM, Carter CS, Braver TS, McDonald A, Sabb FW, Noll DC, et al. Selective deficits in prefrontal cortex regions in medication naive schizophrenia patients. Archives of General Psychiatry. 2001; 50:280–288. [PubMed: 11231835]

Barch DM, Carter CS, MacDonald AW III, Braver TS, Cohen JD. Context-processing deficits in schizophrenia: Diagnostic specificity, 4-week course, and relationships to clinical symptoms. Journal of Abnormal Psychology. 2003; 112:132–143. [PubMed: 12653421]

Barch DM, Csernansky J, Conturo T, Snyder AZ, Ollinger J. Working and long-term memory deficits in schizophrenia. Is there a common underlying prefrontal mechanism? Journal of Abnormal Psychology. 2002; 111:478–494. [PubMed: 12150424]

Barch DM, Mitropoulou V, Harvey PD, New AS, Silverman JS, Siever LJ. Context-processing deficits in schizotypal personality disorder. Journal of Abnormal Psychology. 2004; 113:556–568. [PubMed: 15535788]

Beech A, Baylic GC, Smithson P, Claridge G. Individual differences in schizotypy as reflected in measures of cognitive inhibition. British Journal of Clinical Psychology. 1989; 28:117–129. [PubMed: 2743052]

Bergman AJ, Harvey PD, Mitropoulou V, Aronson A, Marder D, Silverman J, et al. The factor structure of schizotypal symptoms in a clinical population. Schizophrenia Bulletin. 1996; 22:501–509. [PubMed: 8873300]

Bergman AJ, Harvey PD, Roitman SL, Mohs RC, Marder D, Silverman JM, et al. Verbal learning and memory in schizotypal personality disorder. Schizophrenia Bulletin. 1998; 24:635–641. [PubMed: 9853795]

Bowie CR, Harvey PD. Cognition in schizophrenia: Impairments, determinants, and functional importance. Psychiatric Clinics of North America. 2005; 28:613–633. [PubMed: 16122570]

Brambilla P, MacDonald AW III, Sassi RB, Johnson MK, Mallinger AG, Carter CS, Soares JC. Context processing in bipolar disorder patients. Bipolar Disorders. 2007; 9:230–237. [PubMed: 17430297]

Braver TS, Barch DM, Cohen JD. Cognition and control in schizophrenia: A computational model of dopamine and prefrontal function. Biological Psychiatry. 1999; 46:312–328. [PubMed: 10435197]

Braver TS, Cohen JD. Dopamine, cognitive control, and schizophrenia: The gating model. Progress in Brain Research. 1999; 121:327–349. [PubMed: 10551035]

Braver TS, Cohen JD, Nystrom LE, Jonides J, Smith EE, Noll DC. A parametric study of prefrontal cortex involvement in human working memory. NeuroImage. 1997; 5:49–62. [PubMed: 9038284]

Burton SC. Strategies for improving adherence to second-generation antipsychotics in patients with schizophrenia by increasing ease of use. Journal of Psychiatric Practice. 2005; 11:369–378. [PubMed: 16304505]

Cadenhead KS, Perry W, Shafer K, Braff DL. Cognitive functions in schizotypal personality disorder. Schizophrenia Research. 1999; 37:123–132. [PubMed: 10374648]

Callicott JH, Bertolino A, Mattay VS, Langheim FJ, Duyn J, Coppola R, et al. Physiological dysfunction of the dorsolateral prefrontal cortex in schizophrenia revisited. Cerebral Cortex. 2000; 10:1078–1092. [PubMed: 11053229]

Callicott JH, Egan MF, Mattay VS, Bertolino A, Bone AD, Verchinski B, et al. Abnormal fMRI response of the dorsolateral prefrontal cortex in cognitive intact siblings of patients with schizophrenia. American Journal of Psychiatry. 2003; 160:709–719. [PubMed: 12668360]

Casey BJ, Cohen JD, Jezzard P, Turner R, Noll DC, Trainor R, et al. Activation of prefrontal cortex in children during a non-spatial working memory task with functional MRI. NeuroImage. 1995; 2:221–229. [PubMed: 9343606]

Chen EY, Hui CL, Dunn EL, Lien YJ, Chang YH, Hwu HG. A prospective 3-year longitudinal study of cognitive predictors of relapse in first-episode schizophrenia patients. Schizophrenia Research. 1998; 77:99–104. [PubMed: 16005389]

Cohen JD, Barch DM, Carter C, Servan-Schreiber D. Context-processing deficits in schizophrenia: Converging evidence from three theoretically motivated cognitive tasks. Journal of Abnormal Psychology. 1999; 108:120–133. [PubMed: 10066998]

Cohen JD, Braver TS, Perlstein WM, Jonides J, Smith EE, Noll DC. Temporal dynamics of cortical activity in verbal working memory. NeuroImage. 1996; 3:S537.

Cohen JD, MacWhinney B, Flatt MR, Provost J. Psy-Scope: A new graphic interactive environment for designing psychology experiments. Behavioral Research Methods, Instruments & Computers. 1993; 25:257–271.

Cohen JD, Servan-Schreiber D. Context, cortex and dopa-mine: A connectionist approach to behavior and biology in schizophrenia. Psychological Review. 1992; 99:45–77. [PubMed: 1546118]

Davidson M, Harvey PD, Powchik P, Parella M, White L, Knobler HY, et al. Severity of symptoms in geriatric chronic schizophrenic patients. American Journal of Psychiatry. 1995; 152:197–207. [PubMed: 7840352]

deJong PF, Olson RK. Early predictors of letter knowledge. Journal of Experimental Child Psychology. 2004; 88:254–273. [PubMed: 15203300]

Diforio D, Walker E, Kestler LP. Executive function in adolescents with schizotypal personality disorder. Schizophrenia Research. 2000; 42:125–134. [PubMed: 10742650]

Egan MF, Goldberg TE, Kolachana BS, Callicott JH, Mazzantu CM, Straub RE, et al. Effect of COMT Val108/158 Met genotype on frontal lobe function and risk for schizophrenia. Proceedings of the National Academy of Sciences, USA. 2001; 98:6917–6922.

Farmer CM, O'Donnell BF, Niznikiewicz MA, Voglmaier MM, McCarley RW, Shenton ME. Visual perception and working memory in schizotypal personality disorder. American Journal of Psychiatry. 2000; 157:781–786. [PubMed: 10784472]

First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. Structured Clinical Interview for DSM-IV Axis I Disorders—Patient Edition (SCID-I/P, Version 2.0). New York State Psychiatric Institute; New York: 1995.

Friedman JI, Temporini H, Davis KL. Pharmacological strategies for augmenting cognitive performance in schizophrenia. Biological Psychiatry. 1999; 45:1–16. [PubMed: 9894570]

Gathercole SE, Tiffany C, Briscoe J, Thorn A, ALSPAC Team. Developmental consequences of poor phonological short-term memory function in childhood: A longitudinal study. Journal of Child Psychology & Psychiatry. 2005; 46:598–611. [PubMed: 15877766]

Green MF. Cognitive impairment and functional outcome in schizophrenia and bipolar disorder. Journal of Clinical Psychiatry. 2006; 67:3–8. [PubMed: 16965182]

Green MF, Waldron JH, Coltheart M. Emotional context processing is impaired in schizophrenia. Cognitive Neuropsychiatry. 2007; 12:259–280. [PubMed: 17453905]

Guy, W. ECDEU assessment manual for psychopharmacology: Revised. National Institute of Mental Health; Rockville, MD: 1976. Clinical Global Impressions.; p. 218-222.

Harvey PD, Reichenberg A, Romero M, Granholm E, Siever LJ. Dual-task information processing in schizotypal personality disorder: Evidence of impaired processing capacity. Neuropsychology. 2006; 20:453–460. [PubMed: 16846263]

Heaton RK, Gladsjo JA, Palmer BW, Kuck J, Heaton RK, Jeste DV. Stability and course of neuropsychological deficits in schizophrenia. Archives of General Psychiatry. 2001; 58:24–32. [PubMed: 11146755]

Heinrichs RW. The primacy of cognition in schizophrenia. American Psychologist. 2005; 60:229–242. [PubMed: 15796677]

Kirrane RM, Mitropoulou V, Nunn M, New AS, Harvey PD, Schopick F, et al. Effects of amphetamine on visuospatial working memory performance in schizophrenia spectrum personality disorder. Neuropsychopharmacology. 2000; 22:14–18. [PubMed: 10633486]

Koenigsberg HW, Buchsbaum MS, Buchsbaum BR, Schneiderman JS, Tang CY, New A, et al. Functional MRI of visuospatial working memory in schizotypal personality disorder: A region-of-interest analysis. Psychological Medicine. 2005; 35:1019–1030. [PubMed: 16045068]

Lenzenweger MF. Schizotaxia, schizotypy, and schizophrenia: Paul Meehl's blueprint for the experimental psychopathology and genetics of schizophrenia. Journal of Abnormal Psychology. 2006; 115:195–200. [PubMed: 16737381]

MacDonald AW III, Carter CS, Kerns JG, Ursu S, Barch DM, Holmes AJ, et al. Specificity of prefrontal dysfunction and context processing deficits to schizophrenia in never-medicated patients with first-episode psychosis. American Journal of Psychiatry. 2005; 162:475–484. [PubMed: 15741464]

MacDonald AW III, Pogue-Geile MF, Johnson MK, Carter CS. A specific deficit in context processing in the unaffected siblings of patients with schizophrenia. Archives of General Psychiatry. 2003; 60:57–65. [PubMed: 12511173]

Menon V, Anagnoson RT, Mathalon DH, Glover GH, Pfefferbaum A. Functional neuroanatomy of auditory working memory in schizophrenia: Relation to positive and negative symptoms. NeuroImage. 2001; 13:433–446. [PubMed: 11170809]

Mishara AL, Goldberg TE. A meta-analysis and critical review of the effects of conventional neuroleptic treatment on cognition in schizophrenia: Opening a closed book. Biological Psychiatry. 2004; 55:1013–1022. [PubMed: 15121486]

Mitropoulou V, Harvey PD, Zegarelli G, New AS, Silverman JM, Siever LJ. Neuropsychological performance in schizotypal personality disorder: Importance of working memory. American Journal of Psychiatry. 2005; 162:1896–1903. [PubMed: 16199836]

Moriarty PJ, Harvey PD, Granholm E, Mitropoulou V, Siever LJ. Information processing capacity in schizotypal personality disorder: Evidence from a study of dual-task information processing. Journal of Clinical and Experimental Neuropsychology. 2003; 25:335–347. [PubMed: 12916647]

Moritz S, Mass R. Reduced cognitive inhibition in schizotypy. British Journal of Clinical Psychology. 1997; 36:365–376. [PubMed: 9309352]

Niznikiewicz MA, Shenton ME, Voglmaier M, Nestor PG, Dickey CC, Rhoads R, et al. Semantic dysfunction in woman with schizotypal personality disorder. American Journal of Psychiatry. 2002; 159:1767–1774. [PubMed: 12359685]

Niznikiewicz MA, Voglmaier M, Shenton ME, Seidman LJ, Dickey CC, Rhoads R, et al. Electrophysiological correlates of language processing in schizotypal personality disorder. American Journal of Psychiatry. 1999; 156:1052–1058. [PubMed: 10401451]

Nuechterlein, KH. Vigilance in schizophrenia and related disorders.. In: Steinhauer, SR.; Gruzelier, JH.; Zubin, J., editors. Handbook of schizophrenia: Vol. 5. Neuropsychology, psychophysiology, and information processing. Elsevier; Amsterdam: 1991. p. 397-433.

Overall JE, Gorham DR. The Brief Psychiatric Rating Scale. Psychological Reports. 1962; 10:799–812.

Park S, Holzman PS, Lenzenweger MF. Individual differences in spatial working memory in relation to schizotypy. Journal of Abnormal Psychology. 1995; 104:355–363. [PubMed: 7790637]

Perlstein WH, Carter CS, Noll DC, Cohen JD. Relation of prefrontal cortex dysfunction to working memory and symptoms in schizophrenia. American Journal of Psychiatry. 2001; 158:1105–1113. [PubMed: 11431233]

Pfohl, B.; Blum, N.; Zimmerman, M. Structured Interview for DSM-IV Personality (SIDP-IV). University of Iowa, College of Medicine; Iowa City: 1995.

Prouteau A, Verdoux H, Briand C, Lesage A, Lalonde P, Nichole L, et al. Cognitive predictors of psychosocial functioning outcome in schizophrenia: A follow-up study of subjects participating in a rehabilitation program. Schizophrenia Research. 2005; 77:343–353. [PubMed: 16085207]

Roitman SE, Cornblatt BA, Bergman A, Obuchowski M, Mitropoulou V, Keefe RS, et al. Attentional functioning in schizotypal personality disorder. American Journal of Psychiatry. 1997; 154:655–660. [PubMed: 9137121]

Roitman SE, Mitropoulou V, Keefe RS, Silverman JM, Serby M, Harvey PD, et al. Visuospatial working memory in schizotypal personality disorder patients. Schizophrenia Research. 2000; 41:447–455. [PubMed: 10728721]

Saykin AJ, Shtasel DL, Gur RE, Kester DB, Mozley LH, Stafiniak P, et al. Neuropsychological deficits in neuroleptic naive patients with first-episode schizophrenia. Archives of General Psychiatry. 1994; 51:124–131. [PubMed: 7905258]

Siegel BV, Trestman RL, O'Faithbheartaigh S, Mitropoulou V, Amin F, Kirrane RM, et al. D-amphetamine challenge effects on Wisconsin Card Sort Test performance in schizotypal personality disorder. Schizophrenia Research. 1996; 20:29–32. [PubMed: 8794491]

Siever LJ. Biological markers in schizotypal personality disorder. Schizophrenia Bulletin. 1995; 11:564–574. [PubMed: 4081650]

Siever LJ, Davis KL. A psychobiological perspective on the personality disorders. American Journal of Psychiatry. 1991; 148:1647–1658. [PubMed: 1957926]

Silver H, Feldman P, Bilker W, Gur RC. Working memory deficit as a core neuropsychological dysfunction in schizophrenia. American Journal of Psychiatry. 2003; 160:1809–1816. [PubMed: 14514495]

Ventura J, Lukoff D, Nuechterlein KH, Liberman RP, Green MF. Manual for the Expanded Brief Psychiatric Rating Scale. International Journal of Methods in Psychiatric Research. 1993; 3:227–244.

Voglmaier M, Seidman LJ, Salisbury D, McCarley RW. Neuropsychological dysfunction in schizotypal personality disorder: A profile analysis. Biological Psychiatry. 1997; 41:530–540. [PubMed: 9046985]

Wechsler, D. The Wechsler Adult Intelligence Scale—Revised manual. Psychological Corporation; San Antonio, TX: 1981.

Weinberger DR, Berman KF, Suddath R, Torrey EF. Evidence of dysfunction of a prefrontal-limbic network in schizophrenia: A magnetic resonance imagining and regional cerebral blood flow study of discordant monozygotic twins. American Journal of Psychiatry. 1992; 149:890–897. [PubMed: 1609867]

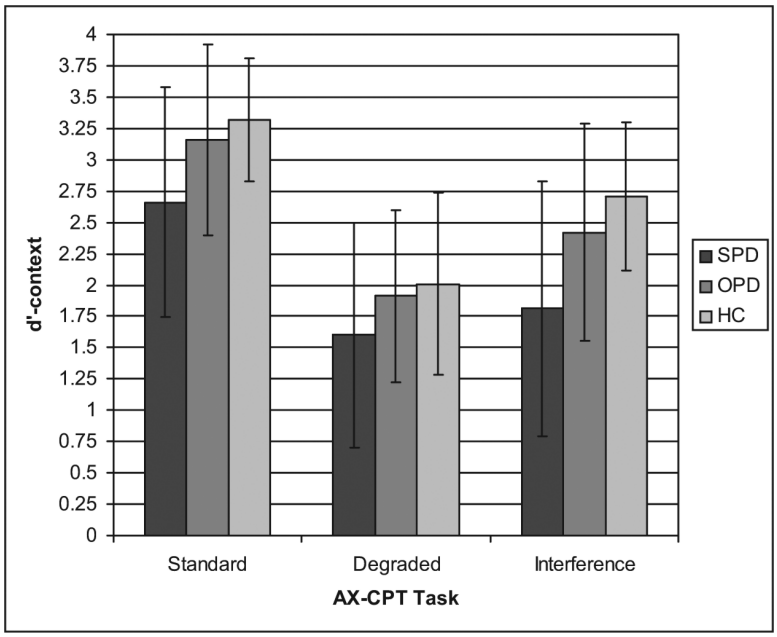

Figure 1.

d′-context scores by group for the three versions of the AX Continuous Performance Test (AX-CPT). SPD = schizotypal personality disorder; OPD = other (non-Cluster A) personality disorders; HC = healthy controls.

Table 1

Frequency of DSM-IV Diagnostic Classifications for the OPD Group and Comorbid Diagnoses for the SPD Group

	Group	
***DSM-IV* personality disorder**	**Other PD**	**Schizotypal PD**
Paranoid	0 (0%)	35 (56%)
Schizoid	0 (0%)	4 (6%)
Schizotypal	0 (0%)	63 (100%)
Antisocial	1 (4%)	6 (10%)
Borderline	5 (20%)	17 (27%)
Histrionic	0 (0%)	1 (2%)
Narcissistic	4 (16%)	7 (11%)
Avoidant	6 (24%)	27 (43%)
Dependent	0 (0%)	5 (8%)
Obsessive-Compulsive	12 (48%)	18 (29%)
PD not otherwise specified	4 (16%)	0 (0%)
Modal number of total PDs	2	1

Note. Percentages total more than 100 because several participants met criteria for more than one personality disorder (PD). OPD = other (non-Cluster A) personality disorders; SPD = schizotypal personality disorder; *DSM-IV* = *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.).

Table 2

Sample Characteristics

	Group					
	Healthy controls		Other PD		Schizotypal PD	
Characteristic	***M***	***SD***	***M***	***SD***	***M***	***SD***
Age (in years)	30.1	9.7	38.5	13.5	37.4	10.4
Sex (% male)	47.6		48.0		82.5	
Education (in years)	16.7	2.4	15.6	3.1	14.7	2.5
Maternal education	15.1	3.1	14.2	2.2	13.7	3.3
Paternal education	15.4	3.2	13.7	3.5	12.9	4.3
Vocabulary scores	11.8	2.7	11.2	2.5	10.4	2.5
Block Design scores	11.7	3.3	11.7	3.1	9.7	2.7
Total hours worked per week	41.9	8.4	17.7	30.1	17.7	21.4
BPRS total score			27.5	6.2	32.6	12.8
CGI total score			3.1	0.6	3.3	0.6

Note. BPRS = Brief Psychiatric Rating Scale; CGI = Clinical Global Impressions Scale.

Table 3

Standard AX Continuous Performance Test: Errors and Reaction Times

	Group											
	Healthy controls				Other personality disorder				Schizotypal personality disorder			
	Errors		RT		Errors		RT		Errors		RT	
Trial type	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Short delay												
AX	2.1	3.5	463.4	113.0	4.1	10.6	435.9	84.4	3.7	6.3	447.4	107.5
AY	4.8	12.1	589.4	117.6	4.5	7.8	543.5	83.6	6.6	12.8	562.4	98.7
BX	2.2	4.9	432.1	148.6	4.5	8.4	555.2	230.2	15.5	28.5	530.9	174.9
BY	1.3	3.6	381.3	85.3	1.3	4.8	418.1	102.5	2.2	4.6	439.1	98.9
d'-context	3.5	0.4			3.4	0.7			3.1	0.9		
Long delay												
AX	6.4	10.7	506.9	120.9	14.2	24.9	462.0	68.8	18.0	24.2	506.7	107.9
AY	8.7	12.8	644.7	120.0	6.1	7.7	584.9	82.9	4.9	9.8	597.0	103.4
BX	5.4	9.0	450.5	151.8	4.3	10.5	508.4	192.8	18.5	28.2	561.3	202.3
BY	7.7	3.7	419.4	94.3	0.0	0.0	437.7	93.7	1.3	4.9	487.9	97.4
d'-context	3.1	0.7			2.8	1.2			2.2	1.2		

Note. Error data are proportions of errors, and reaction time (RT) data are milliseconds

J Abnorm Psychol. Author manuscript; available in PMC 2014 September 27.

Table 4

Effect Sizes of Group Differences for Three Versions of the AX Continuous Performance Test

	Error type			
Trial type	**BX short**	**BX long**	**AY short**	**AY long**
Standard				
SPD vs. OPD	.44	.60	.17	.11
SPD vs. HC	.57	.61	.15	.45
OPD vs. HC	.02	.11	0	.27
Degraded				
SPD vs. OPD	.33	.26	.25	.35
SPD vs. HC	.52	.36	.39	.12
OPD vs. HC	.20	.19	.14	.24
Interference				
SPD vs. OPD	.37	.29	.25	.27
SPD vs. HC	.61	.42	.05	.07
OPD vs. HC	.45	.17	.07	.20

Note. Data are effect sizes [(mean of Group 1 – mean of Group 2)/SD_{pooled}]. SPD = schizotypal personality disorder; OPD = other (non-Cluster A) personality disorders; HC = healthy controls.

Table 5

Degraded AX Continuous Performance Test: Errors and Reaction Times

	Group											
	Healthy controls				Other personality disorder				Schizotypal personality disorder			
	Errors		RT		Errors		RT		Errors		RT	
Trial type	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Short delay												
AX	17.1	14.9	501.8	133.3	16.1	11.6	457.4	73.9	17.0	15.0	462.2	105.0
AY	11.6	11.1	622.0	112.7	13.6	17.9	629.3	110.2	19.1	21.1	603.0	102.9
BX	13.7	10.1	470.2	154.5	17.2	20.0	528.6	177.1	24.8	25.3	523.3	158.5
BY	2.2	4.3	438.1	99.9	4.4	10.1	458.2	121.2	7.1	15.3	486.9	103.3
d'-context	2.2	0.7			2.14	0.7			1.8	0.9		
Long delay												
AX	24.4	23.8	541.8	114.9	30.5	24.5	501.5	90.1	34.4	28.2	530.2	113.5
AY	13.5	18.0	687.0	130.5	17.3	17.7	673.6	120.5	11.1	16.2	616.4	123.9
BX	16.4	15.1	464.9	129.1	13.4	16.6	517.2	176.7	23.9	24.5	504.7	135.4
BY	19.5	5.1	463.4	105.7	2.3	6.0	532.2	129.2	3.5	12.4	504.8	112.2
d'-context	1.8	0.9			1.7	1.0			1.2	1.1		

Note. Error data are proportions of errors, and reaction time (RT) data are milliseconds.

Table 6

Interference AX Continuous Performance Test: Errors and Reaction Times

	Group											
	Healthy controls				Other personality disorder				Schizotypal personality disorder			
	Errors		RT		Errors		RT		Errors		RT	
Trial type	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Short delay												
AX	4.7	6.7	340.0	68.4	12.3	22.4	336.8	66.6	15.7	19.7	325.6	73.5
AY	14.3	16.9	487.8	105.5	13.5	12.0	493.0	96.8	14.9	16.9	484.8	164.5
BX	2.2	5.7	311.0	73.5	5.8	11.0	363.9	168.3	14.9	25.0	365.1	185.1
BY	0.2	1.5	301.8	58.9	0.0	0.0	346.7	103.3	0.1	1.4	360.7	148.7
d'-context	3.3	0.5			3.8	2.7			2.3	1.1		
Long delay												
AX	17.4	14.6	384.4	114.4	18.4	15.8	408.3	122.4	33.9	26.9	419.2	125.4
AY	11.5	14.2	468.6	120.0	13.9	15.4	458.5	86.9	9.9	15.2	432.4	132.3
BX	13.8	17.4	325.0	120.8	17.2	17.8	382.1	176.9	24.1	26.5	356.6	160.8
BY	2.3	5.6	323.4	80.3	3.2	10.6	364.7	114.1	2.2	6.1	378.7	166.5
d'-context	2.2	0.9			3.6	2.0			1.3	1.2		

Note. Error data are proportions of errors, and reaction time (RT) data are milliseconds.

J Abnorm Psychol. Author manuscript; available in PMC 2014 September 27.

Table 7

Cross-Task Correlations: Schizotypal Personality Disorder Group

Trial and delay	0-back	1-back	2-back	Vocabulary	Block Design
Standard AX-CPT version					
AY short	–.20	–.09	–.30*	–.39**	–.21
AY long	–.42**	–.26*	–.31*	–.15	–.07
BX short	–.25	–.30*	–.28*	–.16	–.30*
BX long	–.16	–.30*	–.29*	–.16	–.33*
d'-context short	.42**	.37**	.28*	.03	.25
d'-context long	.31*	.36**	.44**	.21	.31*
Interference AX-CPT version					
AY short	–.06	.17	.18	–.06	.17
AY long	–.07	.05	.05	.01	.10
BX short	–.27*	–.22	–.28*	–.27*	–.34**
BX long	–.26*	.05	–.41**	–.28*	–.36*
d'-context short	.36**	.46**	.57**	.30*	.34**
d'-context long	.27*	.52**	.56**	.28*	.29*
Degraded AX-CPT version					
AY short	–.07	–.25	–.25	–.10	.04
AY long	.14	.05	.05	.15	.02
BX short	–.39**	–.43**	–.43	–.18	–.34**
BX long	–.41**	.21	–.25	.12	–.14
d'-context short	.33*	.44**	.49**	.24	.34**
d'-context long	.41**	.34**	.50**	.17	.25

* $p < .05$.

** $p < .01$.

Table 8

N-Back Working Memory Test: Correct Responses and Reaction Times

	Group											
	Healthy controls				Other personality disorder				Schizotypal personality disorder			
	Correct response		RTs		Correct response		RTs		Correct response		RTs	
Condition	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
0-back	95.9	6.8	577.5	131.7	96.9	4.0	587.7	108.6	94.4	7.7	552.8	121.2
1-back	95.0	4.4	617.7	133.5	93.7	5.6	651.6	137.9	90.3	7.7	615.3	158.8
2-back	91.5	6.7	728.7	215.8	87.9	8.0	786.5	226.3	82.3	9.3	677.4	220.3

Note. Response data are proportion of correct responses, and reaction time (RT) data are milliseconds.