Journal of Abnormal Psychology
2012, Vol. 121, No. 1, 119–128

0021-843X/11/$12.00 DOI: 10.1037/a0023938

Visuospatial Attention in Schizophrenia: Deficits in Broad Monitoring

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Although selective attention is thought to be impaired in people with schizophrenia (PSZ), prior research has found no deficit in the ability to select one location and withdraw attention from another. PSZ and healthy control subjects (HCS) performed a stimulus detection task in which one, two, or all four peripheral target locations were cued. When one or two locations were cued, both PSZ and HCS responded faster when the target appeared at a cued than uncued location. However, increases in the number of validly cued locations had much more deleterious effects on performance for PSZ than HCS, especially for targets of low contrast whose detection was more dependent on attention. PSZ also responded more slowly in trials with four cued locations relative to trials with one or two invalidly cued locations. Thus, visuospatial attention deficits in schizophrenia arise when broad monitoring is required rather than when attention must be focused narrowly.

Keywords: schizophrenia, visuospatial attention, attentional window, SARAT, default network

Neurocognitive deficits in schizophrenia best predict long-term functional disease outcome (Green, 1996; Green, Kern, & Heaton, 2004). These deficits are manifold but circumscribed, affecting numerous distinct mechanisms but sparing others (Gold, Hahn, Strauss, & Waltz, 2009). Efforts to develop effective pharmacotherapy for these symptoms rely on a mapping, characterization, and reduction of their complexity to a finite number of underlying problems (Marder & Fenton, 2004; Carter & Barch, 2007). A target of particular interest is selective attention, which has been frequently considered to be the root of impairments across a wide variety of cognitive tasks.

The visuospatial selective attention domain has been studied extensively in people with schizophrenia (PSZ), using variants of the Posner orienting paradigm (Posner, 1980), in which a cue directs attention either voluntarily or involuntarily to one of two possible target locations. Although many of these studies focused on examining possible lateralized abnormalities (Bustillo et al., 1997; Carter, Robertson, Chaderjian, Celaya, & Nordahl, 1992; Carter, Robertson, Chaderjian, O'Shora-Celaya, & Nordahl, 1994; Gold et al., 1992; Liotti, Dazzi, & Umilta, 1993; Maruff, Hay, Malone, & Currie, 1995; Posner, Early, Reiman, Pardo, & Dhawan, 1988; Sapir, Henik, Dobrusin, & Hochman, 2001; Strauss, Novakovic, Tien, Bylsma, & Pearlson, 1991; Wigal, Swanson, & Potkin, 1997), they also provide more general clues about visuospatial selective attention mechanisms in schizophrenia. Notably, collapsed across hemifields, the reaction time (RT) difference between trials with a valid cue (i.e., one that correctly predicts the location of an upcoming target) and an invalid cue (i.e., one that directs attention to a location where the target does not appear) is no smaller in PSZ than in healthy control subjects (HCS). This is true for all of the above studies, except Posner et al (1988). A deficit in the ability to select one location and withdraw attention from another would have manifested itself in slower RT in valid trials and faster RT in invalid trials and thus would have resulted in a smaller validity effect. PSZ displayed no such selection deficit, which is surprising given the hypothesized dysfunction of selective attention (Nuechterlein & Dawson, 1984; Luck & Gold, 2008). However, there is a suggestion of other, unexpected abnormalities.

A replicated finding is that the RT benefit of trials with a valid cue relative to a neutral condition that does not provide information about where to attend tends to be larger in PSZ than in HCS (Gold et al., 1992; Liotti, Dazzi, & Umilta, 1993; Bustillo et al., 1997; Sapir et al., 2001). A recent study confirmed this phenomenon in a direct assessment using optimized task conditions (Spencer et al., 2011). Thus, against all expectations, PSZ seemingly use the cue information more efficiently to orient attention in space. Alternatively, PSZ may be impaired on the neutral trials, with a reduced ability to spread attention widely and to maintain a broad focus of attention. The resulting disproportionate slowing in the neutral condition would result in the appearance of greater RT

This article was published Online First May 23, 2011.

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We thank Valerie M. Beck for help with setting up the eye-tracking procedure, Jacqueline Kiwanuka, Sharon August, and Leeka Hubzin for their assistance with the conduct of the study, Dr. Robert McMahon for statistical advice, and all volunteers participating in this study. This research was supported by a grant from the National Institute of Mental Health (grant number MH065034 to J.M.G. and S.J.L.).

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benefits of valid cues and reduced RT costs of invalid cues relative to the neutral condition. Indeed, the RT cost of invalid cues tends to be reduced in PSZ relative to HCS, although only in studies using a no-cue neutral condition in which the target is not preceded by any signal (Liotti, Dazzi, & Umilta, 1993; Nestor et al., 1992; Oie, Rund, & Sundet, 1998). When a double-cue neutral condition has been used, with peripheral cues at both locations, the RT cost on invalid relative to neutral trials tends to be larger in PSZ (Carter et al., 1992, 1994; Bustillo et al., 1997; Daban et al., 2004). A possible explanation, given that PSZ do not appear to derive greater alerting effects from cues (Daban et al., 2004; Gouzoulis-Mayfrank et al., 2007), is that the physical onset of the double-cue automatically directed the attentional focus to the two possible target locations in a bottom-up manner. Thus, PSZ may derive greater benefit than HCS from attention being spatially directed by peripheral cues to both locations before target onset, and conversely, the state of not having external signals guide attention to any specific locations may create disproportional impairment. That is, PSZ may have a deficit in attending broadly on the basis of endogenous attentional control mechanisms.

The current study directly tested the hypothesis that PSZ have difficulty distributing attention broadly under endogenous control. We used a visuospatial attention paradigm in which a central cue predicted the location of a peripheral target stimulus. One, two, or all four possible target locations could be cued simultaneously, manipulating the degree to which narrow focusing versus broad spatial monitoring was required. The target usually appeared at a cued location (valid trials), but occasionally at an uncued location (invalid trials), thus allowing us to simultaneously assess the ability to spread attention across varying numbers of locations and the ability to focus attention (by comparing performance for valid vs. invalid trials). We predicted that stimulus detection deficits in PSZ relative to HCS would be particularly pronounced when all four locations were cued, necessitating a broad and diffuse attentional focus. If deficits are specific to broad monitoring rather than disengaging and shifting attention, performance of PSZ on these trials should be impaired relative to both valid and invalid predictive cue trials, but the difference between valid and invalid trials should be equivalent in PSZ and HCS. We also manipulated target contrast, predicting that the differences between PSZ and HCS would be larger for low-contrast targets because high-contrast targets evoke automatic detection mechanisms that are less influenced by the top-down distribution of spatial attention (Hawkins, Shafto, & Richardson, 1988). Because we manipulated target contrast, and PSZ sometimes exhibit contrast sensitivity impairments (reviewed by Javitt, 2009), we included a perceptual control task to ensure that the observed differences between PSZ and HCS were not a result of low-level sensory mechanisms.

Method

Participants

Twenty-nine clinically stable, medicated outpatients meeting Diagnostic and Statistical Manual of Mental Disorders-IV (*DSM–IV*; American Psychiatric Association, 1994) criteria for schizophrenia ($n = 13$ paranoid, 7 undifferentiated, 2 residual, 1 disorganized) or schizoaffective disorder ($n = 6$), and 26 matched HCS participated. Demographic and clinical information is summarized in Table 1. Diagnosis was established using a best estimate approach in which information from a Structured Clinical Interview for *DSM–IV* (SCID) was combined with a review of medical records at a consensus diagnosis meeting chaired by one of the authors (J.M.G.). All patients were receiving antipsychotic medication at time of testing; four were treated with first-generation antipsychotics, 23 with second-generation antipsychotics, and two with both. Fifteen patients additionally received mood stabilizing medication, five a benzodiazepine, and three benztropine, an antiparkinsonian medication. A set of analyses of medication effects will be presented at the end of the Results section. Only patients whose medication had not changed in the preceding four weeks were enrolled. Control participants were recruited from the com-

Table 1
Group Demographics (Mean ± SD)

	Patients	Controls
Age	41.4 ± 9.8 (range 22–53)	41.9 ± 9.0 (range 23–54)
Male:Female	16:13	13:13
AA:C:A:AI[a]	10:16:2:1	12:14:0:0
Education (years)	13.1 ± 2.3	14.7 ± 1.8*
Parental education[b]	14.3 ± 3.3[c]	13.5 ± 1.9
WASI	101.5 ± 13.5	112.0 ± 11.9*
MATRICS total score	33.8 ± 15.3	48.9 ± 10.5**
WRAT 4 standard score	98.9 ± 14.2	99.4 ± 12.7
WTAR standard score	101.9 ± 16.8	103.5 ± 12.2
BPRS	36.2 ± 7.4 (range 24–53)	
SANS	33.8 ± 12.9 (range 4–57)	
LOFS	20.5 ± 6.3 (range 10–34)	
Calgary Depression Scale	2.8 ± 2.6 (range 0–9)	

Note. BPRS = Brief Psychiatric Rating Scale (Overall & Gorman, 1962); SANS = Scale for the Assessment of Negative Symptoms (Andreasen, 1984); LOFS = Level Of Functioning Scale (Hawk, Carpenter, & Strauss, 1975); Calgary Depression Scale (Addington, Addington, Maticka-Tyndale, & Joyce, 1992).
[a] AA = African American; C = Caucasian; A = Asian; AI = American Indian. [b] Average over mother's and father's years of education. [c] Data unavailable for two subjects.
* $p < .01$. ** $p < .001$; significant difference between PSZ and HCS in independent samples *t* test.

munity via random digit dialing and word of mouth and were not taking any psychotropic medication. None of the control participants had a current Axis I or II diagnosis, as established by a SCID, and no self-reported family history of psychosis. Two HCS had a history of Major Depression, now in full remission. Groups did not differ in age [$t(53) = 0.2$, $p > .8$], parental education [$t(51) = 1.13$, $p > .2$], sex (χ^2 $p > .7$), or ethnicity (χ^2 $p > .3$). However, PSZ had fewer years of education than HCS [$t(53) = 2.88$, $p < .01$]. All participants provided informed consent for a protocol approved by the University of Maryland School of Medicine Institutional Review Board. Before participants signed the consent form, the investigator reviewed its content with them and answered any questions. For PSZ, basic understanding of study demands and risks was formally evaluated in the presence of a third-party witness.

Neuropsychological Testing

Participants completed the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999), the Wide Range Achievement Test (WRAT 4; Wilkinson, & Robertson, 2006), the Wechsler Test of Adult Reading (WTAR; Wechsler, 2001), and the MATRICS battery (Nuechterlein, & Green, 2006). These tests were usually given on a separate day from the main experiment. PSZ scored lower than HCS on the WASI ($p = .004$) and MATRICS battery ($p < .001$), but there were no group differences on the WRAT 4 ($p > .8$) or WTAR ($p > .6$), suggesting similar premorbid functioning (see Table 1).

Equipment

Tasks were completed in a dimly illuminated room on a 17" CRT monitor with a 60-Hz refresh rate. Eye-tracking was performed throughout both tasks to monitor central fixation, using an EyeLink 1000 eye-tracking system (SR Research Ltd., Mississauga, Ontario) operating at 2000 Hz. The eye-tracker consisted of an infrared light source and video camera, providing an image of the participant's right eye. Participants rested their heads on a chin and forehead rest at 70 cm viewing distance from the monitor.

Stimuli and Tasks

Spatial Attentional Resource Allocation Task (SARAT). The SARAT has been previously described and validated as a tool for manipulating the size of the attentional focus in space (Hahn, Ross, & Stein, 2006). The task was slightly modified from the original version. Participants were required to keep their eyes fixated on a central circle containing a fixation cross (trials with eye movements were eliminated) and to detect a target signal at any of four peripheral locations marked by placeholders (see Figure 1). With eyes directed at the center of the fixation cross, the center of the target location placeholders was positioned at an eccentricity of 12.5°. The diameter of the central circle was 3.0°, and that of each of the placeholders was 1.5°. The central circle and placeholders, black against white, formed a background that remained on display throughout the task. A target consisted of one of the placeholders filling with a checkerboard of gray and white squares of 3 × 3 pixels each, yielding a spatial frequency of ~5.4 cycles/° (note that these squares are too small to be discernible in Figure 1). The luminance of the white checks equaled that of the white background. Two target contrasts were tested. The contrast of the gray checks was 80% for the high-contrast targets and 20% for the low-contrast targets. Contrast was calculated as (white luminance − luminance of gray checks)/white luminance (as measured with a J17 LumaColor photometer, Tektronix, Beaverton, OR). Upon detecting a target, participants pressed a button with their dominant index finger as quickly as possible.

A trial began once continuous central fixation was maintained for 500 ms. A cue then appeared in the central circle. Target presentation followed after a variable stimulus-onset-asynchrony of 400, 700, 1000, or 1300 ms. The target was visible for 500 ms, and the cue remained on display until 500 ms after target offset. The cue consisted of one, two, or four quarters of the fixation circle turning black, indicating that the subsequent target was likely to appear in one of the corresponding quadrants of the display. When two quadrants were cued, they were always adjoining (both top, both bottom, both left, or both right).

The number of cued locations is related to the predictability of the target location. Fewer cued locations provide more precise information about the target location, allowing for a narrower and more intense attentional focus at the cued location(s) (Hahn, Ross, & Stein, 2006). Conversely, increasing the number of cued locations increases spatial uncertainty and the need to monitor broadly. The cue provided invalid information on 20% of the trials in which one or two locations were cued.

The cue was not followed by a target on 9.7% of trials, presented unpredictably, to discourage anticipatory responding to the cue. These cue-only trials were identical to the other trials, except that no target was presented during the 500-ms target interval. False alarms during these trials averaged 1.3 ± 3.8% in HCS and 7.8 ± 25% in PSZ [$t(53) = 1.28$, NS]. All trials were followed by a 1500-ms intertrial interval, during which only the task background was presented. In total, there were 336 valid trials (56 × 1/2/4 cued locations × high/low target contrast), 56 invalid trials (14 × 1/2 cued locations × high/low contrast), and 42 cue-only trials (14 × 1/2/4 cued locations), tested over 14 blocks interspersed by rest periods. All trial types were randomized over every two consecutive blocks. The task took approximately one hour to complete.

Perceptual control task. To test whether possible group differences in perceptual sensitivity could explain the results we obtained, we invited all participants of the main experiment back to perform a contrast sensitivity measure that used the SARAT stimuli but had minimal attentional requirements and manipulated target contrast across a wide range of values (see Figure 1). A standard two-interval forced choice procedure was used to avoid response bias effects (Macmillan, & Creelman, 1991), and the method of constant stimuli was used rather than an adaptive staircase to avoid confounding sensitivity with lapses of attention. Originally, contrast levels of 4, 8, 16, 32, and 64% were tested. After five HCS and nine PSZ had completed the task, a 12% contrast condition was added for the remaining participants. Each trial began with a 500-ms fixation period, followed by the onset of a one-location cue. One thousand milliseconds after cue onset, a brief, clearly audibly tone was presented, and 1500 ms after the first tone, a different, easily differentiable tone was presented. A 500-ms target stimulus came on at the cued location simultaneously with one of

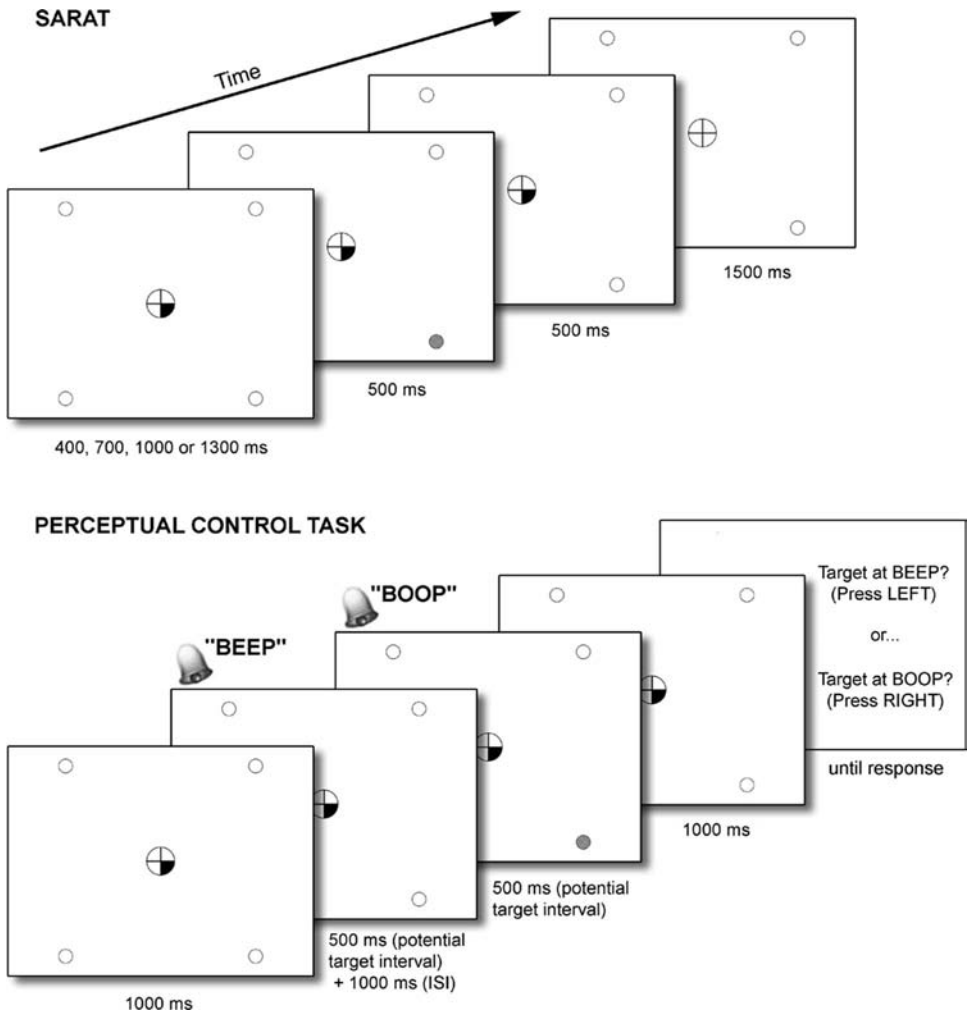

Figure 1. Trial examples of the Spatial Attentional Resource Allocation Task (SARAT, top) and the perceptual control task (bottom). For the SARAT, one, two, or all four locations were cued and subjects responded upon detecting the target. For the perceptual control task, one location was always cued. Target onset coincided with either one of two briefly presented tones ("BEEP" or "BOOP"), and subjects made a forced choice as to with which tone the target coincided.

the two tones. Thus, participants could always predict exactly and with absolute certainty where (at the cued location) and when (coinciding with one of the two tones) the target would appear. The central cue disappeared 1500 ms after the onset of the second tone. At the end of each trial, participants made an unspeeded forced choice indicating whether the target coincided with the first or second tone. Thirty trials of each contrast were tested, divided over five runs, each followed by a rest period. Contrast levels were randomized within each run. The task took approximately 30 min to complete. Twenty-five PSZ and 22 HCS completed it; the others were lost to follow-up. Two PSZ performed at chance across all contrast levels and clearly had difficulty following instructions. Their data were excluded from analysis of the control task, resulting in $n = 23$ PSZ. Their SARAT data were not excluded because there was no indication that they had problems understanding this simple stimulus detection task.

Data Analysis

SARAT. Trials with RT below 200 ms or above 2500 ms were considered outliers and excluded from analyses (0.13% and 0.14% of trials in PSZ and HCS, respectively). RTs were expressed as means and omission errors as the percentage of trials in which a target was presented but no response was made, as in previous studies using this paradigm (Hahn, Ross, & Stein, 2006; Hahn, Ross, & Stein, 2007; Hahn et al., 2007). Trials with fixations (defined as stationary eye-position for $\geq$10 ms) outside a central circle measuring 4° of visual angle in diameter that occurred between cue onset and target offset were excluded from analyses of RT and omission errors. The percentage of trials with such eye movements was also analyzed. Eye-tracking data were lost for one HCS, for whom all trials were included in analyses of RT and omission errors. Exclusion of this HCS did not change any of the results.

Performance indices were analyzed by three-factor mixed-model ANOVAs with Group (PSZ, HCS) as a between-subjects factor and the number of cued locations (NumCuedLoc; 1,2,4) and Target contrast (high, low) as within-subject factors. One ANOVA included valid trials with one or two cued locations and nonpredictive trials (four cued locations), and a separate ANOVA included invalid trials with one or two cued locations and nonpredictive trials. To compare the validity effect (invalid vs. valid trial performance) between groups, RT and omission errors in trials with one or two cued locations were also submitted to a four-factor ANOVA that included both valid and invalid trials (Group × Validity × NumCuedLoc × Target contrast).

Perceptual control task. The percentage of trials in which the participant correctly reported which interval contained the target was analyzed using a two-factor ANOVA with Group as a between-subjects factor and Target contrast as a within-subject factor. Trials with fixations outside the central fixation area between cue onset and the end of the second potential target interval were excluded from analysis.

Results

SARAT

Reaction Time

Valid versus nonpredictive trials. First we consider trials with one, two, or four cued locations on which the target appeared at a cued location. PSZ responded more slowly than HCS overall (see Figure 2), leading to a significant main effect of Group [$F(1, 53) = 6.79$, $p < .02$] in three-factor ANOVA. Both groups responded more slowly to low- than high-contrast targets, as supported by a main effect of Target contrast [$F(1, 53) = 101.1$, $p < .001$]. RTs increased monotonically with greater spatial uncertainty, leading to a main effect of NumCuedLoc [$F(2, 106) = 108.4$, $p < .001$]. This uncertainty-dependent slowing was of approximately twice the magnitude in PSZ as that in HCS, as supported by a significant interaction of Group with NumCuedLoc [$F(2, 106) = 11.2$, $p < .001$]. The average RT slowing from trials with one to trials with four cued locations was 49 ms in HCS versus 94 ms in PSZ [$t(53) = 3.79$, $p < .01$; Cohen's $d = 0.63$].

The three-way interaction (Group × NumCuedLoc × Target contrast) was also significant [$F(2, 106) = 4.05$, $p = .02$]. This reflected a larger effect of the number of cued locations for low-contrast than high-contrast targets in PSZ but not in HCS. Supporting this, separate two-factor ANOVAs in PSZ and HCS yielded a significant interaction of NumCuedLoc with Target contrast only in PSZ [$F(2, 56) = 4.23$, $p = .02$]. In PSZ, the RT slowing from trials with one to trials with four cued locations averaged 81.4 ms for high-contrast targets and 106 ms for low-contrast targets [$t(28) = 2.18$, $p < .038$; paired samples t test]. In HCS, this RT slowing was almost identical between the target contrasts (49.9 vs. 48.1 ms; NS).[1] The slowing did not correlate with scores on any of the neuropsychological measures in either PSZ or HCS for either target intensity.

Invalid versus nonpredictive trials. Next, we compared invalid cue trials with one and two cued locations and nonpredictive cue trials (four cued locations). In HCS, RTs were approximately the same for all cue types. Remarkably, PSZ were actually slower on the nonpredictive than invalid cue trials. These differences yielded a significant interaction of NumCuedLoc (1,2,4) with Group [$F(2, 106) = 5.44$, $p = .006$] in three-factor ANOVA. One-factor ANOVAs confirmed an effect of NumCuedLoc in PSZ [$F(2, 56) = 8.87$, $p < .001$] but not in HCS [$F(2, 50) < 1$]. Interactions involving Target contrast were not significant. Thus, PSZ but not HCS were impaired when the cue directed them to spread attention across four locations compared to when the cue directed them to focus on an incorrect location.

Valid versus invalid trials. Both groups displayed slower RT on invalid than valid cue trials with one or two cued locations, and this was confirmed by a significant main effect of Validity [$F(1, 53) = 25.7$, $p < .001$] in a four-factor ANOVA [Group × Validity × NumCuedLoc (1,2) × Target contrast]. There were no significant interactions involving Validity and Group [Validity × Group: $F(1, 53) = 1.1$, $p > .3$], and the Validity effect was significant ($p < .001$) in both HCS (41 ms, averaged over NumCuedLoc and Target contrast) and PSZ (45 ms). The effect size of the group difference in the validity effect was Cohen's $d = 0.13$, confirming the absence of a group effect. Thus, as in many prior studies, we found no evidence of an impairment in the ability of PSZ to focus attention onto one or two cued locations.

% Omission Errors[2]

Valid versus nonpredictive trials. Both PSZ and HCS made more omission errors with low- than high-contrast targets (see Figure 3). However, PSZ but not HCS showed an increase in omission errors with greater spatial unpredictability for the low-contrast targets. This was supported by a significant three-way interaction [$F(2, 106) = 4.55$, $p = .013$]. Separate two-factor ANOVAs in PSZ and HCS confirmed a significant NumCuedLoc × Target contrast interaction in PSZ [$F(2, 56) = 5.82$, $p = .005$], which was absent in HCS [$F(2, 50) < 1$]. In follow-up one-factor ANOVAs, the effect of NumCuedLoc in PSZ was significant for low-contrast [$F(2, 56) = 3.84$, $p = .027$] but not high-contrast targets [$F(2, 56) < 1$]. The increase in omission errors from trials with one to trials with four cued locations for low-contrast targets was of moderate effect size ($d = 0.58$). It did not correlate with scores on any of the neuropsychological measures.

[1] Disproportionate slowing with more cued locations in PSZ was not restricted to trials with four cued locations but became gradually more pronounced with an increasing number of cued locations. This is substantiated by the finding that, for low-contrast targets, PSZ and HCS differed significantly in their degree of slowing not only for the RT difference between trials with one and four cued locations [$t(53) = 3.70$, $p < .001$, independent samples t test], but also for the RT difference between trials with one and two cued locations [$t(53) = 2.73$, $p < .01$].

[2] This measure failed Shapiro-Wilk tests for normality in both HCS ($p < .001$) and PSZ ($p = .05$). Data transformation could not remedy this. Skewness (HCS: 2.49, PSZ: 0.71) was mainly attributable to a few large outlier values. Skewness was substantially reduced (HCS: 0.569, PSZ: 0.585) by excluding the two HCS and the three PSZ with the largest percentage of omission errors, which also aided normality (Shapiro-Wilk test: $p = .12$ in HCS, $p = .15$ in PSZ). After excluding these subjects from the ANOVA of valid trials, the critical three-way interaction was still significant ($p < .045$).

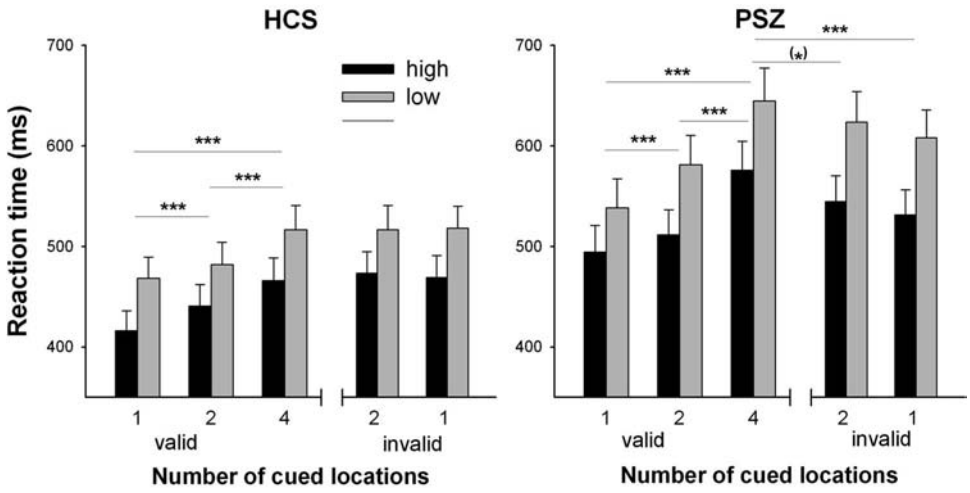

Figure 2. Reaction times of healthy control subjects (HCS) and people with schizophrenia (PSZ) in the SARAT. The graph compares the averages (±SEM) of trials with high-contrast targets ("high") and low-contrast targets ("low"), and trials with one, two, or four validly cued target locations and one or two invalidly cued locations. (*) $p = .07$, *** $p < .001$ in Tukey's tests comparing RT, averaged over high- and low-contrast targets, between trials with one, two, or four validly cued locations, and between trials with one and two invalidly cued locations and trials with four cued locations.

Invalid versus nonpredictive trials. PSZ made a disproportionately large number of omission errors on both nonpredictive and invalid cue trials with low-contrast targets. This was substantiated by a Group × Target contrast interaction [$F(1, 53) = 7.37$, $p < .01$] in a three-factor ANOVA. Follow-up t tests confirmed a significant group difference for low-contrast [$t(53) = 2.93$, $p = .005$] but not high-contrast targets [$t(53) = 1.43$, NS]. In PSZ, the omission rate was numerically greater on nonpredictive trials than on invalid trials with a single cued location, whereas HCS showed the opposite pattern. However, effects involving NumCuedLoc were not significant.

Valid versus invalid trials. Both groups made more omission errors on invalid than valid trials, as confirmed by a significant main effect of Validity [$F(1, 53) = 5.38$, $p < .05$] in a four-factor ANOVA. There were no significant interactions involving Validity and Group (Validity × Group: $p = .15$). This provides additional evidence that PSZ are unimpaired at directing attention toward some locations and withdrawing attention from others.

Percentage of Trials With Eye Movements

Valid versus nonpredictive trials. Trials with eye movements away from the fixation point occurring between cue onset and target offset were more numerous in PSZ than HCS (see Figure 4). Their number increased with spatial unpredictability in PSZ but not in HCS, especially for low-contrast targets. This led to a significant three-way interaction [$F(2, 104) = 6.93$, $p = .001$]. In

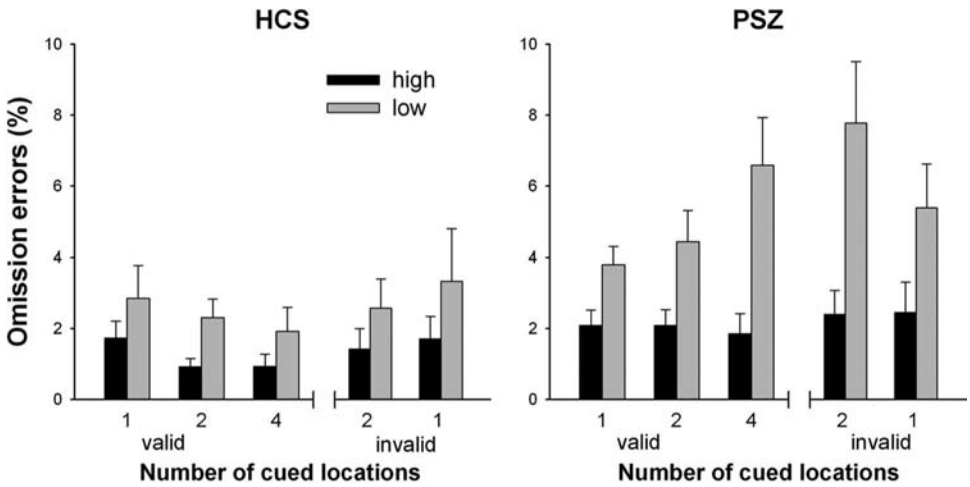

Figure 3. The percentage of omission errors (averages ± SEM) of HCS and PSZ in the SARAT.

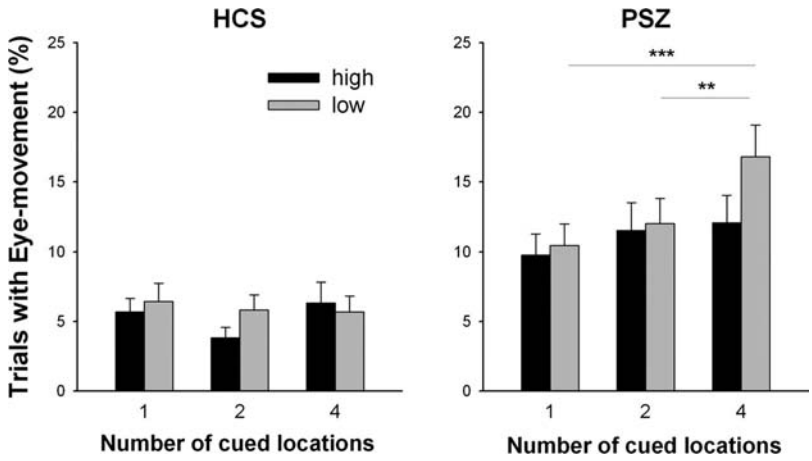

Figure 4. The percentage of trials with eye movement in HCS and PSZ in the SARAT. The graph compares trials with high-contrast targets ("high") and low-contrast targets ("low") with one, two, or four validly cued locations. ** $p < .01$, *** $p < .001$ in Tukey's test comparing only trials with low-contrast targets because a cue effect was identified only for these trials.

HCS, the percentage of trials with eye movements did not depend on NumCuedLoc or Target contrast, as confirmed by an absence of main effects or interaction in a two-factor ANOVA. In PSZ, however, the NumCuedLoc × Target contrast interaction was significant [$F(2, 56) = 5.54$, $p = .006$]. In one-factor ANOVAs, the effect of NumCuedLoc was significant in PSZ for low-contrast targets [$F(2, 56) = 15.3$, $p < .001$] but not high-contrast targets [$F(2, 56) = 1.80$, NS], indicating that spatial uncertainty increased eye movements particularly for trials with low-contrast targets (Cohen's $d = 0.64$ for the difference between one and four cued locations for low-intensity targets).

Invalid versus nonpredictive trials, and valid versus invalid trials. Invalid trials were not analyzed because the percentage of trials with eye movements was typically zero as a result of the low total number of invalid cue trials.

Effects of Medication Status

Additional three-factor ANOVAs were performed comparing PSZ who received a given medication with all other PSZ. Each ANOVA included NumCuedLoc (1, 2, 4) and Target contrast as within-subject factors, but one ANOVA included Benzodiazepine, one Mood stabilizer, one Clozapine, and one Typical antipsychotic (always present vs. absent) as a between-subjects factor. As above, separate ANOVAs were performed for valid and nonpredictive and for invalid and nonpredictive trials. The only significant interaction involving Benzopdiazepine was with NumCuedLoc on valid trial RT [$F(2, 54) = 3.44$, $p < .05$]. This effect was driven by two of the five patients treated with benzodiazepines displaying RT differences of >170 ms between trials with one and four cued locations. When repeating the original Group × NumCuedLoc × Target contrast ANOVA without these five patients, the same main effects and interactions were observed. The only significant interaction involving Mood stabilizer was with Target contrast on the percentage of trials with eye movements [$F(1, 27) = 4.36$, $p < .05$]. In PSZ not receiving mood stabilizers, trials with eye movements were more numerous for low- than high-contrast targets [13% versus 10%; $t(13) = 3.15$, $p < .01$], but in PSZ treated with mood stabilizers trials with eye movements were almost identical between high- and low-contrast targets (13% in each case). When repeating the original Group × NumCuedLoc × Target contrast ANOVA without these 15 patients, the same results were obtained. There were no interactions involving Clozapine or Typical antipsychotic.

Finally, for PSZ, we performed Pearson correlations of haloperidol equivalents (Andreasen, Pressler, Nopoulos, Miller, & Ho, 2010) with the difference between valid trials with one and four cued locations in the three dependent variables (RT, omission errors, trials with eye-movements). There were no significant correlations for trials with low-contrast or high-contrast targets.

Perceptual Control Task

The purpose of this task was to test whether the difficulty of PSZ in detecting the low-contrast targets under conditions of spatial uncertainty and the specific psychophysical parameters of the SARAT could have been an artifact of poorer perceptual sensitivity rather than a consequence of an impaired ability to distribute attention broadly. Reduced contrast sensitivity in PSZ to our task stimuli would be indicated by impaired discrimination performance relative to HCS, especially for trials with low contrast levels. As can be seen from Figure 5, performance accuracy was almost identical between PSZ and HCS, and this was supported by the absence of a Group main effect [$F(1, 43) < 1$] or a Group × Target contrast interaction [$F(4, 172) < 1$] in a two-factor ANOVA. When including the 12% data point and limiting ANOVA to participants for whom this data point was available ($n = 14$ PSZ, $n = 17$ HCS), the same results were obtained [Group: $F(1, 29) < 1$; Group × Target contrast: $F(5, 145) < 1$]. Accuracy did not differ between groups at any contrast level in independent-samples t tests ($p > .3$ in each case). Thus, we conclude that although impaired contrast sensitivity has been observed in PSZ under different stimulus and task conditions (reviewed by Javitt, 2009), our sample of PSZ had no perceptual deficit relative to HCS in detecting the low-contrast stimuli under the present conditions. It is

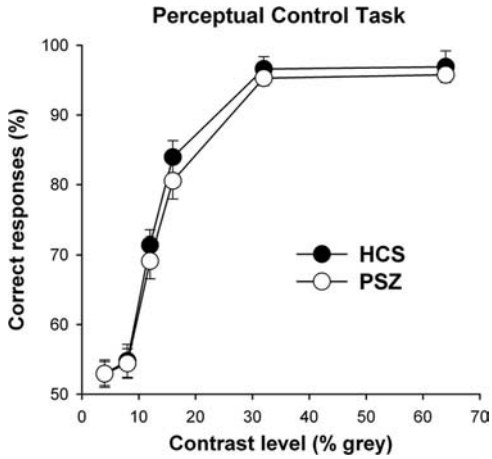

Figure 5. Response accuracy (averages ± SEM) in the perceptual control task for HCS (n = 22) and PSZ (n = 23). The 12% contrast level data points include only 17 HCS and 14 PSZ.

extremely unlikely that any difference in perceptual sensitivity that could not be detected when tested directly was responsible for the performance patterns in the SARAT.

Discussion

The present findings resolve the apparent discrepancy between the widespread belief that schizophrenia involves impaired attentional selection and the repeated finding that PSZ exhibit normal and even superior visuospatial attentional cuing effects (Gold et al., 2009; Spencer et al., 2011). Specifically, the present study confirms the observation that PSZ are unimpaired at focusing attention on one location and withdrawing attention from others, but it demonstrates a substantial impairment in the ability of PSZ to distribute attention broadly.

On valid trials, both groups displayed slower responding when the target location became more uncertain, but this effect was substantially more pronounced for PSZ than HCS. If this was attributable to better attentional selection with more precise cueing, larger performance costs would have been observed on invalid cue trials. Instead, the performance difference between valid and invalid trials when one or two locations were cued did not differ between PSZ and HCS, as observed in previous studies (reviewed by Gold et al., 2009). Furthermore, PSZ but not HCS actually responded more slowly on nonpredictive than invalid trials. Thus, attending broadly is actually more deleterious to performance in PSZ than focusing attention away from the location of the upcoming target. The results indicate that the observed performance pattern arose because PSZ were disproportionately impaired in nonpredictive cue trials that required monitoring all four possible target locations.

Impairment with more spatial uncertainty was particularly pronounced in trials with low-contrast targets in PSZ. First, RT slowing with spatial uncertainty was greater with low- than high-contrast targets in PSZ but not HCS. Second, omission errors in trials with low-contrast targets increased with greater spatial uncertainty in PSZ but not HCS. Third, on trials with low-contrast targets, PSZ made more eye movements away from central fixation when the cue was nonpredictive. Thus, spatial unpredictability combined with low physical target salience created the largest performance impairment in PSZ. However, our perceptual control experiment indicated that, under the current task conditions, PSZ and HCS did not differ in contrast sensitivity to the targets per se; that is, PSZ had no problems detecting the peripheral low-contrast stimuli when attentional demands were minimized. The disparity between this finding and studies that did identify contrast sensitivity reductions in PSZ may be attributable to differences in stimulus properties (ours are likely to be processed by the magno- and parvocellular visual pathways, while processing deficit may be specifically magnocellular; Javitt, 2009), in patient populations (reduced contrast sensitivity may be associated with negative symptoms; Slaghuis, 2004; Keri, Kiss, Kelemen, Benedek, & Janka, 2005), in medication status (the current sample received mostly atypical neuroleptics, while reduced contrast sensitivity may be specific to PSZ medicated with typical antipsychotics; Chen et al., 2003), or in the measurement method (by using the method of constant stimuli rather than an adaptive staircase we rule out differences resulting from nonspecific factors such as lapses of attention). Importantly, without drawing any more general conclusions about perceptual abnormalities in schizophrenia, we can say that the current stimulus detection deficit observed when multiple locations were cued was clearly attentional and not sensory in nature.

Why would a broad monitoring deficit be moderated by target contrast? The greater sensitivity of the low-contrast targets to the deleterious effects of a broad attentional state may be conceptualized under limited resource capacity models of attention (e.g., Kahneman, 1973). Broad monitoring of all four locations may be more effortful for PSZ than HCS and exceed the available processing resources, thus leading to a suboptimal attentional state. Bottom-up orienting may have aided detection of high-contrast targets even when PSZ were in a suboptimal attentional state (Hawkins, Shafto, & Richardson, 1988). Detection of low-contrast targets, however, is more dependent on spatial attention. When spatial uncertainty is high, the size of the attentional window must be expanded to effectively monitor all possible target locations (Eriksen & Yeh, 1985; Belopolsky, Zwaan, Theeuwes, & Kramer, 2007), and this spreading of the benefits of attentional processing across the visual field enables the detection of even low-contrast targets. Thus, the patient deficit may be described as suboptimal maintenance of a wide attentional window. Indeed, it has recently been suggested that PSZ have a narrowed "attentional spotlight" and insufficient attentional resources to maintain a wide visual span (Elahipanah, Christensen, & Reingold, 2010).

Psychophysical models suggest that inattention slows the rate of perceptual information acquisition (Luck & Vecera, 2002; Palmer, 1998) and thus increases the time required to reach detection threshold. For low-contrast stimuli, the detection threshold may never be reached on some trials, leading to omission errors. Thus, a failure of adopting a broader attentional window with more spatial uncertainty can explain both the increased RTs for low- and high-contrast targets and the increased rate of omission errors for low-contrast targets in PSZ. This failure, quantified as the difference in RT or omission errors between trials with one and four cued locations in PSZ, did not correlate with any of the neuropsychological indices collected, including IQ, MATRICS domains, WTAR, and WRAT. Thus, at this point in time, there is no obvious

clue about the degree to which a broad monitoring deficit may relate to other aspects of cognitive dysfunction described in PSZ. Interpreted broadly and generalized beyond the visuospatial domain, a narrowed attentional focus, or inability to spread attention broadly, may limit the ability to process multiple inputs or perceive multiple concomitant possibilities. We speculate that this type of processing limitation may translate into a reduced ability to consider multiple alternatives and may underlie the reduced cognitive flexibility described in PSZ (e.g., Elliot, McKenna, Robbins & Sahakian, 1995). Clearly, the current findings do not suffice to support such generalization, but they may lay the foundation for future work.

The eye movement data suggest that PSZ may have tried to overcome their difficulty in efficiently spreading attention across the visual field by resorting to a serial focusing of the target locations once they failed to detect a target. PSZ made more fixations outside the central fixation area on nonpredictive cue trials, in particular those with low-contrast targets. These eye movements must have been exploratory rather than being triggered by the physical target onset because exogenous triggering would have yielded more eye movements for high-contrast targets. Instead, PSZ appeared to initiate a large portion of eye movements upon guessing that they missed the target, or in an effort to verify the occurrence of a low-contrast target. Thus, RT, omission errors, and central fixation performance provided converging evidence for a deficit in the ability of PSZ to monitor broadly and maintain a wide attentional window. Based on the pattern of results obtained previously using the Posner paradigm (see Introduction), we suggest that this impairment is a result of dysfunction in top-down attentional control rather than an inability to distribute attention widely as a result of bottom-up orienting. Future studies may test this possibility by using peripheral cues.

An fMRI study of the SARAT in healthy adults found that activity in the rostral anterior cingulate and posterior cingulate cortex was predictive of trial-by-trial RT, but only on nonpredictive cue trials that require broad monitoring (Hahn, Ross, & Stein, 2007). These areas are central hubs of the default network of resting brain function, which other studies have shown to be dysfunctional in PSZ (Pomarol-Clotet et al., 2008; Whitfield-Gabrieli et al., 2009). The combination of these previous results suggests that the broad monitoring deficit of PSZ may be caused by dysfunction of the default network. Although the default network is usually associated with inward focusing and task-independent thought processes (Raichle et al., 2001), some studies suggested that this network is also involved in maintaining a broad attentional state of diffuse "watchfulness" toward the external environment (Gilbert, Dumontheil, Simons, Frith, & Burgess, 2007; Gilbert, Simons, Frith, & Burgess, 2006; Hahn, Ross, & Stein, 2007). This "sentinel function"(Buckner, Andrews-Hanna, & Schacter, 2008) is thought of as a safety mode entered when perceptual processing resources are not directed to specific external stimuli. We suggest that a "sentinel dysfunction" may underlie the impaired performance of PSZ when broad attention is required.

There are several potential limitations that should be considered. At this point in time, it is unclear to what degree the identified deficit in spreading attention widely generalizes beyond the spatial domain. Additionally, it is unknown whether it reflects the number of discrete possible target locations or simply the size of the area over which attention has to be spread. Thus, future experiments should address, for example, whether the same results are obtained if there is greater positional uncertainty about target locations. Another limitation is that the current PSZ sample represents a largely stable, medicated outpatient population, and future studies will need to establish to what degree the present findings generalize across disease stages or states. Furthermore, future studies should expand on the present findings by differentiating between different symptom profiles and by more expansive analyses of potential medication effects than possible within the current sample. The current study provides the first clear evidence that visuospatial attention in schizophrenia is marked by a deficit in maintaining a wide attentional window rather than in focusing narrowly. This suboptimal attentional state appears to increase the threshold of physical target salience necessary to trigger target detection, and it may be reflective of an impaired sentinel function of the default network.

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Received November 23, 2010
Revision received March 21, 2011
Accepted March 28, 2011 ■