

Relationships Between Divided Attention and Working Memory Impairment in People With Schizophrenia

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Recent studies suggest that people with schizophrenia (PSZ) have difficulty distributing their attention broadly. Other research suggests that PSZ have reduced working memory (WM) capacity. This study tested whether these findings reflect a common underlying deficit. We measured the ability to distribute attention by means of the Useful Field of View (UFOV) task, in which participants must distribute attention so that they can discriminate a foveal target and simultaneously localize a peripheral target. Participants included 50 PSZ and 52 healthy control subjects. We found that PSZ exhibited severe impairments in UFOV performance, that UFOV performance was highly correlated with WM capacity in PSZ ($r = -.61$), and that UFOV impairments could not be explained by either impaired low-level processing or a generalized deficit. These results suggest that a common mechanism explains deficits in the ability to distribute attention broadly, reduced WM capacity, and other aspects of impaired cognition in schizophrenia. We hypothesize that this mechanism may involve abnormal local circuit dynamics that cause a hyperfocusing of resources onto a small number of internal representations.

Key words: schizophrenia/spatial/working memory/attention/broad monitoring

Introduction

Recent studies from our group suggest that people with schizophrenia (PSZ) distribute their attention in an overly narrow fashion, impacting both visual attention and working memory (WM) capacity. The first evidence for this came from a spatial cuing experiment wherein participants were cued to 1, 2, or 4 peripheral locations, with the 4-cue condition being uninformative.¹ Fewer cued locations

allowed a more precise prediction of the target location and a narrower attentional focus, and both PSZ and healthy control subjects (HCS) were able to speed their response time, indicating both groups used the cue information to selectively orient spatial attention. However, PSZ exhibited a more pronounced step-wise slowing and decrement in target detection with spatial unpredictability than HCS. Moreover, PSZ performed worse in the 4-cue condition than on invalid trials. These results suggested that PSZ were able to narrow attention appropriately in response to predictive cues but were unable to maintain a broad focus when cues were nonpredictive. Consistent with these results, visual search experiments by Elahipanah and colleagues^{2,3} indicated that PSZ have a narrowed visual span.

We found converging evidence for impaired broad monitoring using a sustained attention paradigm in which targets could appear at any of 48 locations.⁴ PSZ showed a robust vigilance decrement with time on task, something that has rarely been observed with sequential target presentations at fixation (eg, most versions of the Continuous Performance Test). Thus, the requirement to monitor broadly may have made it more challenging for PSZ to maintain performance over time.

Further evidence for a tendency of PSZ to focus processing more narrowly than HCS was derived from a study of sequential WM encoding.⁵ When a tone cued the item most likely to be probed, both PSZ and HCS displayed significantly better memory for the cued item relative to uncued items. Surprisingly, when a second tone indicated that the second cued item rather than the first item was now the most likely to be probed, PSZ were more efficient than HCS at discarding the first cued item from memory, an effect not fueled by capacity limitations. This suggested a propensity of PSZ to narrowly focus their WM storage onto a single representation.

Perhaps the most convincing evidence for impairment resulting from excessive selection emerged from an event-related potential study examining contralateral delay activity (CDA).⁶ The CDA is a sustained negative potential observed during WM delay periods, with a larger amplitude contralateral to the relevant hemifield. In the task employed by Leonard and colleagues,⁶ participants were presented with 1, 3, or 5 items to remember, with the relevant items all appearing in 1 hemifield and an equal number of distractors in the other. In HCS, the amplitude of the CDA increases with the number of items held in WM until participants reach their capacity, at which point it asymptotes. Leonard and colleagues⁶ found that the CDA was significantly larger in PSZ than in HCS when 1 item was being remembered. That is, they devoted more selective processing resources to the attended object than controls. In contrast, PSZ had reduced CDAs at higher set sizes relative to HCS. Most critically, a larger CDA at set size 1 was correlated with worse performance at higher memory loads in PSZ. This suggested that the tendency to “hyperfocus” when faced with a single relevant item was associated with decreased memory storage at higher set sizes.

Thus, across experiments with different task demands, there is suggestive evidence that (1) PSZ have difficulty in broad monitoring but no problem narrowing the scope of attention, (2) PSZ tend to hyperfocus attention on single items in WM, and (3) attentional hyperfocusing may be related to WM capacity reduction. To further examine these relationships, we turned to the Useful Field of View (UFOV) test as a measure of the ability to distribute spatial attention.^{7,8}

On the basis of the studies discussed previously, we hypothesized that PSZ would show impairments in distributing attention broadly on the UFOV, and that performance on the UFOV would be predictive of WM capacity. Further, given the strong association of WM with measures of general intellectual ability, we predicted that the UFOV would show similar correlations.

Methods

Participants

Participants included 48 outpatients and 2 inpatients with a diagnosis of schizophrenia or schizoaffective disorder as confirmed by the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; SCID).⁹ PSZ recruitment took place at the Maryland Psychiatric Research Center and associated clinics in the greater Baltimore area. All PSZ were stably medicated with no changes to medication type or dosage for at least 4 weeks before testing (supplementary table 1). A total of 52 HCS participated as a comparison group (table 1). HCS recruitment was accomplished by random digit dialing and newspaper and web advertising. HCS were confirmed to have no psychiatric diagnoses by a SCID and Structured Interview for DSM-IV Personality,¹⁰ and reported that they were not taking psychiatric medications and had no personal or family history of psychosis. All participants were 18–55 years of age, clinically stable, and free of any medical conditions that could impact study results, including substance abuse in the last 6 months. Participants provided written informed consent for a protocol approved by the University of Maryland insti IRB.

Measures

UFOV Test. Participants were seated approximately 70 cm from a 17-in. cathode ray tube monitor in a dimly illuminated room. The UFOV has 3 subtests, presented in a fixed order. The first subtest, which we call “Masked Discrimination” (This task is typically called the “processing speed” task. However, it is simply a Masked Discrimination task, and exaggerated masking would lead to impaired performance in the absence of slowed processing speed.), began with a 2.46° × 2.46° white-outlined fixation box, displayed for 2000 ms at the center of the screen. A 1.64° × 1.23° silhouette of a car or

Table 1. Participant Characteristics and Cognitive Performance

	People With Schizophrenia	Healthy Control Subjects	Group Differences	
<i>N</i>	50	52		
Age	41.76 (10.13)	37.73 (11.10)	$t = -1.91, P = .06$	
Education	13.06 (2.27)	14.87 (1.97)	$t = 4.29, P = .000$	
Maternal education	13.46 (2.52)	14.12 (2.20)	$t = 1.40, P = .17$	
Paternal education	14.35 (3.75)	13.96 (2.90)	$t = -0.57, P = .57$	
Gender (% male)	70	63	$\chi^2 = 0.49, P = .53$	
Race (% Caucasian)	58	54	$\chi^2 = 0.53, P = .77$	
Cognitive performance				Cohen's <i>d</i>
Wechsler Abbreviated Scale of Intelligence	97.82 (11.98)	116.85 (9.03)	$t = 8.76, P = .000$	1.81
Wide Range Achievement Test	95.69 (12.11)	108.72 (12.44)	$t = 5.17, P = .000$	1.06
Wechsler Test of Adult Reading	98.57 (14.45)	112.11 (9.78)	$t = 5.38, P = .000$	1.12
MATRICES Consensus Cognitive Battery	29.90 (13.25)	52.46 (8.59)	$t = 9.98, P = .000$	2.07
K	2.37 (0.59)	2.87 (0.45)	$t = 4.64, P = .000$	0.95

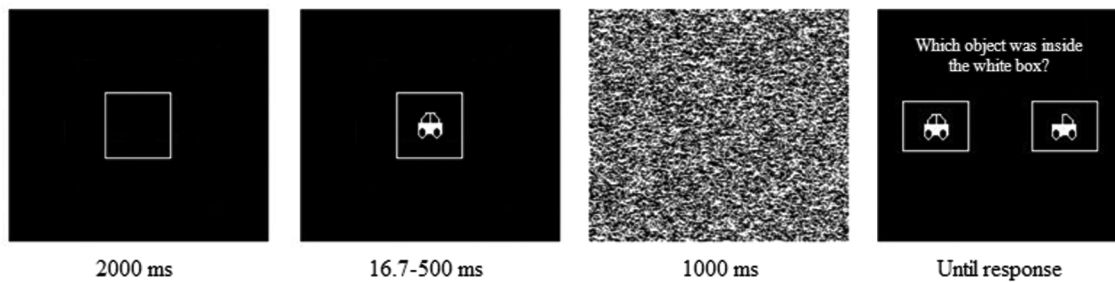
truck then appeared inside the fixation box (figure 1). After a display time that was individually adjusted as described on pages 3–4, a white-noise visual mask covered the entire screen for 1000 ms. Next, participants were presented with a response screen asking them to indicate whether a car or a truck had appeared in the fixation box. Participants were not instructed to fixate throughout the task.

The second subtest, “Divided Attention,” included the fixation box and the central car or truck as in the first subtest, plus the simultaneous presentation of an additional 1.64° × 1.23° silhouette of a car presented at 1 of 8 possible radial locations (0°, 45°, 90°, 135°, 180°, 225°, 270°, and 315°) centered around the fixation point at a distance of 9° visual angle. Both stimuli were then masked by the same white-noise display. To respond, participants first identified the central target by clicking on the image of the car or truck and then clicked on the location of the peripheral target. This task emphasized the ability to divide attention across a broad spatial range and multiple simultaneous targets.

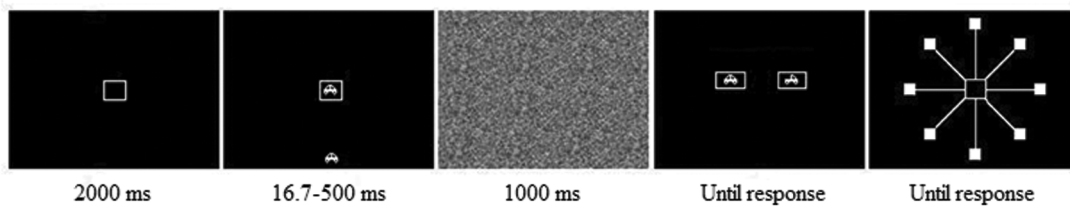
The third subtest, “Divided + Selective Attention” (This task is typically called the “selective attention” task. However, it combines the demands of the Divided Attention task with additional demands on selective attention, so we have used the name “Divided + Selective Attention.”), was identical to Divided Attention except that 47 distractor triangles (1.64° × 1.23°) were added to each display, uniformly filling the space between the possible target locations. This task required participants to both divide attention between the central and peripheral targets and to suppress the peripheral distractors. Although the distractors were uniform, top-down attentional control was necessary to localize the target¹¹ and to orient attention away from the central target.¹²

In each subtest, the dependent variable was the exposure time prior to the mask that was needed for the participant to achieve 75% performance accuracy. This exposure duration was called the “threshold” (lower thresholds indicate better performance). To determine the thresholds, the exposure time was adjusted by means of an adaptive staircase,^{13–15} which ranged from a maximum

Masked Discrimination



Divided Attention



Divided + Selective Attention

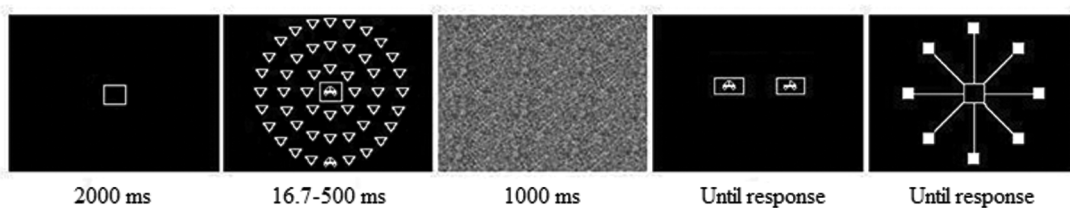


Fig. 1. Stimulus sequences from the Useful Field of View subtests. Images from the Masked Discrimination subtest have been enlarged to better demonstrate the details in the stimuli. Images for the other subtests are proportional to the actual display size.

exposure of 500 ms to a minimum of 16.7 ms (the refresh cycle of a 60-Hz cathode ray tube monitor). On the second and third subtests, participants had to accurately identify both the central and peripheral target for the trial to be considered accurate.

Change Localization. We used a change localization task to assess visual WM capacity. Our version of this task¹⁶ presents participants with an array of 4 colored squares, each with a visual angle of $0.7^\circ \times 0.7^\circ$. The stimuli are arranged around an invisible circle with a radius of 3° for 100 ms, with 1 square in each quadrant of the screen and at least 2.33° of visual angle separation from the next square. The 4 squares disappear from the screen, then reappear 900 ms later, with 1 square having changed color. Participants identify which square changed by clicking on it. This task provides an estimate of the participant's visual WM capacity, or K score, using the formula $K = \text{accuracy (ranging from 0.00 to 1.0)} \times \text{set size (for this task, 4)}$. We have previously shown that performance of PSZ is not limited by the brief stimulus durations in this task.¹⁷

Neuropsychological and Symptom Measures. Participants were administered the Wide Range Achievement Test reading subtest,¹⁸ Wechsler Test of Adult Reading,¹⁹ Wechsler Abbreviated Scale of Intelligence (WASI),²⁰ and the MATRICS Consensus Cognitive Battery (MCCB).^{21,22} Symptom severity was assessed with the Brief Psychiatric

Rating Scale²³ and the Scale for the Assessment of Negative Symptoms.²⁴

Data Analysis. Nonparametric tests were used in the basic between-group comparisons on the UFOV. We used Pearson correlations as well as a series of partial correlations to examine the relationships among UFOV, WM capacity, IQ, and MCCB performance.

Results

Demographic Variables

Participant demographic information is presented in table 1. HCS and PSZ were matched on all variables except personal education, which was lower for PSZ, $t(100) = 4.29, P < .01$, consistent with disease onset in early adulthood.

UFOV Performance

Figure 2 shows the mean display time thresholds for each group for the 3 UFOV subtests, and figure 3 presents histograms showing the threshold distributions. Many HCS achieved the lowest possible threshold in the Masked Discrimination and Divided Attention subtests, leading to highly skewed distributions, which Shapiro-Wilk tests confirmed were non-normal ($P < .0001$ for each). Therefore, we used Mann-Whitney *U* tests to compare HCS and PSZ. These differed significantly in all

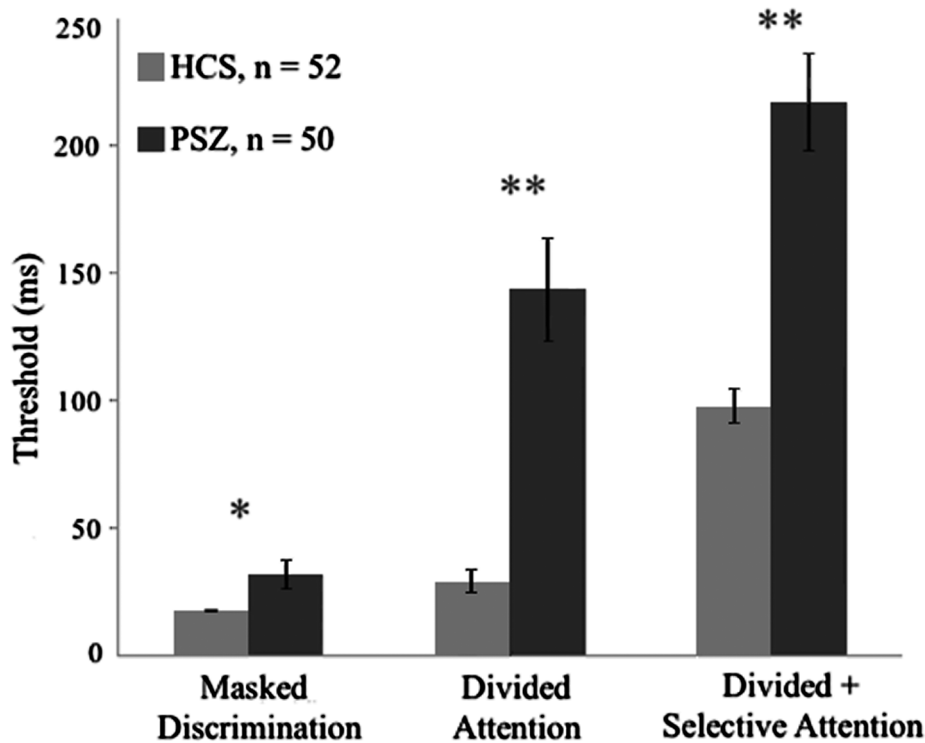


Fig. 2. Mean threshold (\pm standard error of the mean) on each Useful Field of View subtask in people with schizophrenia (PSZ) and healthy control subjects (HCS).

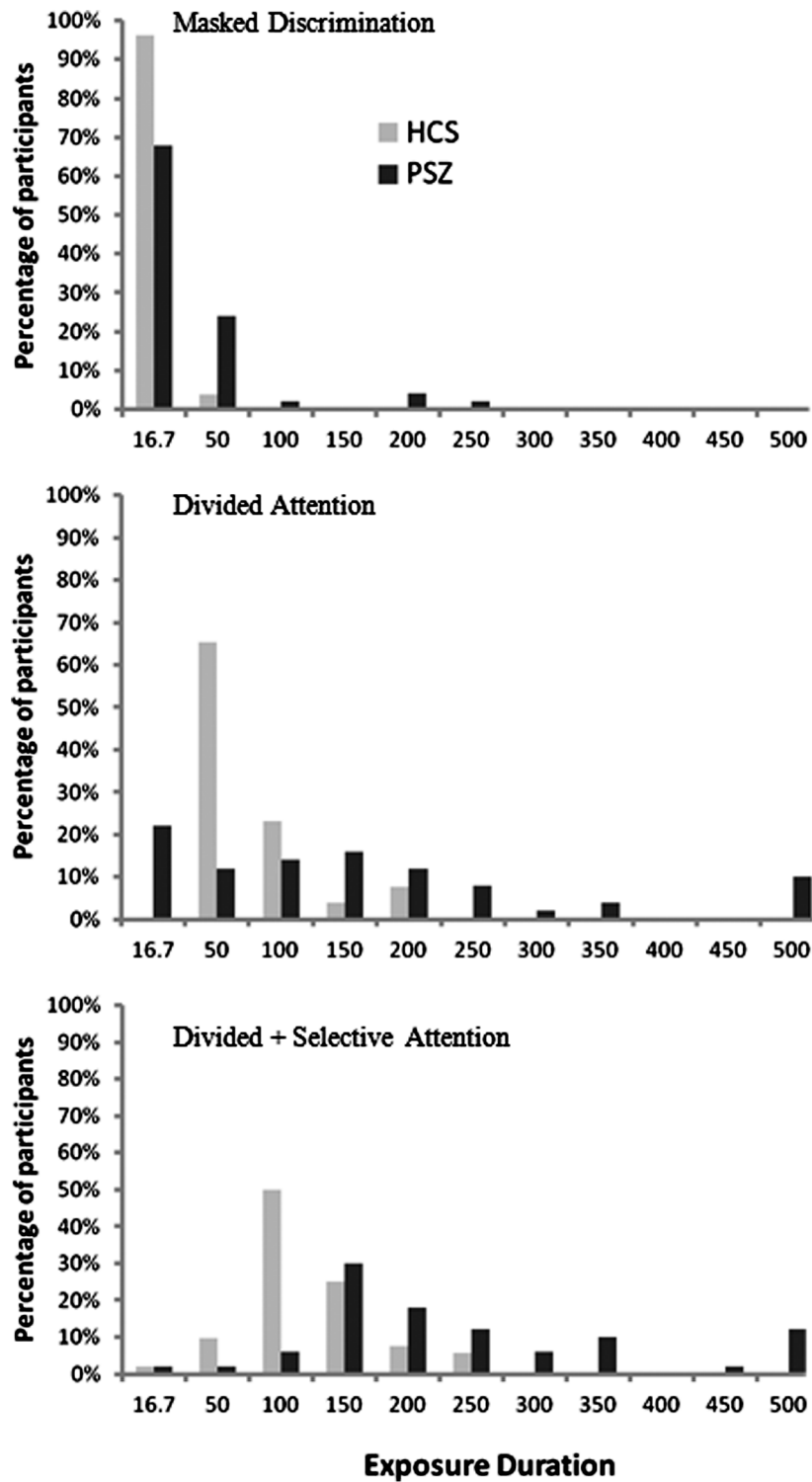


Fig. 3. Distributions of thresholds in the 3 Useful Field of View subtests.

3 subtests: Masked Discrimination $U = 3.78, P < .001$; Divided Attention $U = 5.71, P < .001$; and Divided + Selective Attention $U = 5.52, P < .001$. The effect sizes (Cohen's d) associated with these differences were 0.70 for Masked Discrimination, 1.33 for Divided Attention, and 1.31 for Divided + Selective Attention.

To examine the pattern of performance across subtests, we first computed the difference between performance of the Divided Attention and Masked Discrimination subtests for each subject and compared these difference scores between groups. We found a significant difference, $U = 5.38, P < .001$, indicating that PSZ were significantly

more impaired than HCS by the addition of a peripheral task. We then computed the difference between the Divided Attention and Divided + Selective Attention subtests. This difference score was not significantly different between groups, $U = -1.09$, $P = .28$. Thus, although the Divided + Selective Attention task was more difficult than the Divided Attention task, the resulting performance impairment by the added selective attention component was no larger in PSZ than in HCS. These results are consistent with prior research showing that PSZ are not disproportionately impaired in many selective attention experiments.^{25,26}

WM Capacity and Correlations

As in prior studies,^{6,16,17} WM capacity (quantified as K) was reduced in PSZ (mean K = 2.37) compared with HCS (mean K = 2.87). This effect was large ($d = 0.95$) and significant, $t(100) = 4.64$, $P < .001$.

Performance on the Divided Attention UFOV subtest was strongly correlated with K in PSZ (Pearson's $r = -.61$, $P < .001$), but not in HCS ($r = .05$, $P > .7$), and this difference in correlation was significant ($z = 3.72$, $P < .001$). Eliminating the 5 PSZ who performed at the 500-ms threshold limit did not impact this correlation ($r = -.62$, $P < .001$). In the Divided + Selective Attention subtest, where there was greater variance among HCS, performance was again robustly correlated with K in PSZ ($r = -.49$, $P < .001$), but not in HCS ($r = .09$, $P > .5$; difference between correlations $z = -3.07$, $P < .01$). The same pattern and magnitude of correlations were observed when Spearman correlations were computed (supplementary table 2). Pearson rather than Spearman correlations are reported here for comparison with the partial correlations presented later. To isolate the role of Divided Attention in PSZ, we also examined correlations with K using the difference score between the Masked Discrimination and the Divided Attention subtest: this correlation remained significant in PSZ, $r = -.61$, $P < .001$, suggesting that lower level sensory factors do not explain the relationship with WM (supplementary figure 1). We also examined correlations with K using the difference score between the Divided Attention and the Divided + Selective Attention subtest (supplementary figure 2). This correlation was not significant in PSZ, $r = .24$, $P = .11$, suggesting that it is the requirement to divide attention that was critical for the correlation of the Divided + Selective Attention subtest with WM (difference between correlations $z = -4.62$, $P < .01$).

In PSZ, Divided Attention performance robustly correlated with WASI IQ ($r = -.49$, $P < .001$) and with the MCCB composite score ($r = -.40$, $P = .004$). This raises the possibility that all impairments in our PSZ sample reflect a single source of variance (a generalized cognitive impairment). However, the correlations between Divided Attention and K were still strong in PSZ in partial correlations controlling for the MCCB composite score or

WASI IQ score ($r = -.49$ and $-.46$, respectively, $P < .001$ for both). This correlation also remained strong in PSZ after covarying performance in Masked Discrimination ($r = -.56$, $P < .001$). Thus, the correlation between Divided Attention and WM capacity cannot be explained by either impaired general neuropsychological performance (as indexed by MCCB or by IQ) or impaired low-level processing (as indexed by Masked Discrimination).

Next, we sought to determine to what degree Divided Attention and K each explain individual differences in broader measures of cognitive ability. We conducted a series of partial correlations in PSZ to determine the variance in WASI IQ that is uniquely explained by Divided Attention and K individually. A parallel set of analyses was performed with the MCCB total score. These analyses were conducted only for PSZ. The results for WASI IQ are illustrated in figure 4. These analyses indicated that K explained unique variance in both WASI ($r = .44$, $P = .002$) and MCCB ($r = .54$, $P < .001$) after controlling for Divided Attention performance. In contrast, Divided Attention explained no significant unique variance in either WASI ($r = -.22$, $P = .14$) or MCCB ($r = -.04$, $P = .80$) after controlling for K. The same pattern of results was seen with the MCCB total score as with IQ. Given the high correlation between Divided Attention and K, it appears that a significant portion of the variance in broad cognitive ability in PSZ (as measured with WASI or MCCB) can be explained by the overlapping variance between K and Divided Attention, and a separate portion of this variance is uniquely explained by aspects of K unrelated to Divided Attention performance.

As seen in table 1, WASI IQ scores in the HCS group were above the nominal population mean of 100, raising the possibility that between-group differences might be attributed to differences in general intellectual ability. However, the IQ difference between groups was fully consistent with the differences seen in a previous meta-analysis.²⁷ To examine IQ effects, we pair matched PSZ and HCS who differed by ≤ 1 point in IQ. This resulted in groups of 19 PSZ and 19 HCS with mean IQs of 109.37 and 109.21, respectively. These matched groups differed on the Divided Attention and Divided + Selective Attention subtests (P 's = .04 and .01, respectively), demonstrating that impairment in PSZ on these subtests cannot be explained by differences in IQ. Notably, the matched groups did not differ on K, consistent with our correlational evidence indicating that much of the variance in IQ can be explained by variance in K. It is also consistent with the correlations shown in figure 4 indicating that only a portion of the variance in K is related to Divided Attention.

Clinical Symptoms and Medication

We examined correlations between performance on the UFOV Divided Attention subtest and clinical symptom ratings from the Brief Psychiatric Rating Scale and Scale

in some visuospatial cuing experiments^{1,26} when required to spread attention broadly. Third, PSZ may tend to favor the region around fixation, giving this region greater weight than peripheral locations. Consistent with this explanation are experiments suggesting a narrowed visual span in PSZ,^{2,3} and experiments showing that PSZ make an unusually large number of hypometric saccades when attempting to fixate a peripheral target during a prosaccade task.^{35,36} The latter can be explained by a greater weighting of the fixation point relative to the peripheral target. Inconsistent with this last explanation is the finding that PSZ are unimpaired or even more efficient than HCS in covertly (ie, while continuing to fixate the center) shifting attention to a predicted peripheral target location.^{1,26} These 3 possibilities are not mutually exclusive.

Previous studies have suggested that PSZ have a deficit in distributing attention broadly but do not have a deficit in filtering irrelevant information.^{1,37} This study brings these 2 lines of evidence together, showing both a dramatic deficit in distributing attention broadly and a lack of an additional filtering deficit.

Performance in the Divided Attention task correlated robustly with K and broader measures of cognition that do not ostensibly involve visuospatial attention. These correlations were observed only in PSZ. Note, our failure to observe correlations between UFOV and K in controls does not contradict evidence that the operation of selective attention during WM encoding is an important source of normal individual differences^{38,39}; the UFOV assesses a different aspect of attention. Thus, the UFOV deficit appears to index impairment in a specific process that impacts multiple cognitive operations, solely in PSZ. This is consistent with our previous event-related potential results⁶ indicating that the factors that produce reduced WM capacity in PSZ are not the same as the factors that explain individual differences in capacity among healthy individuals.^{38,39} The WM and UFOV impairments in PSZ do not reflect a sensory impairment or a generalized deficit because the correlation between Divided Attention and K remained strong when Masked Discrimination performance, IQ, or the MCCB composite score were partialled out. The impairment on the UFOV cannot be explained by the unusually high IQ of the HCS because the difference in IQ between PSZ and HCS was quite typical²⁷ and because the deficit remained in IQ-matched subsamples.

It is not intuitively obvious why the ability to divide or spread spatial attention should display such a close relationship to broader neuropsychological measures. We speculate that impairments in the Divided Attention subtest, and in part also reduced WM capacity, reflect an underlying abnormality in the dynamics of local cortical circuits in PSZ. Briefly, we propose that an imbalance between excitatory and inhibitory function tends to cause exaggerated local inhibition and an increase in winner-take-all processing.^{6,40-42} This winner-take-all processing mode is suggested to cause a “hyperfocusing” of

resources onto a small number of locations or objects, whether they are currently visible (as in the Divided Attention subtest) or being held in memory (as in our WM task). When applied to external representations, the tendency to hyperfocus may lead to deficits in dividing attention among multiple targets or spreading attention among multiple locations or a broad area in space. When hyperfocusing is applied to internal representations, this would lead to a reduction in the number of items, rules, or response alternatives that can be simultaneously active, which could compromise more complex cognitive operations.

The above-mentioned hypothesis could explain the present finding of impaired performance and shared variance among the Divided Attention subtest, WM capacity estimates, and the WASI and MCCB measures of broad cognitive function. It is also consistent with the results of other tasks in which hyperfocusing on 1 source of information may be deleterious to performance for other sources of information, such as the attentional blink paradigm³¹⁻³⁴ and some spatial cuing paradigms.^{1,26} Additional tasks have found PSZ to be actually more efficient than HCS at limiting their memory content to task-relevant material and eliminating no-longer relevant information from storage,⁵ as well as having stronger delay-period activity than HCS when maintaining a single object and filtering out another, even in subsets of PSZ and HCS with equivalent overall WM capacity.⁶ Thus, multiple sources of evidence are converging on the theory that many aspects of impaired cognitive performance in PSZ may reflect an underlying hyperfocusing mechanism.

In the partial correlations, we saw that controlling for K strongly attenuated the relationship between the Divided Attention subtest and the WASI or MCCB. Thus, the variance in Divided Attention performance that overlaps with K is critical in explaining WASI and MCCB scores. However, there was also shared variance between K and the broader measures of cognition that was independent of Divided Attention. In other words, the Divided Attention subtest can explain some portions of the shared variance between K and broad functioning while sharing little to no relation with other aspects of K also related to broad functioning in PSZ. However, a large portion of variance in K that is associated with broader functioning can be explained by processes shared with Divided Attention.

Limitations

Our conclusions need to be balanced by acknowledging some limitations. The present results cannot tell us whether the deficits observed in the Divided Attention subtest reflect a problem in dividing attention between multiple objects/locations or in distributing attention broadly in space. New experimental paradigms will be needed to distinguish between these possibilities. Further, some of

our suggestions are based on correlational evidence and comparisons with past experiments. Such evidence cannot prove causality, and we are currently pursuing experimental approaches to directly test the hypothesis that hyperfocusing impacts both attention and WM in schizophrenia.

Supplementary Material

Supplementary material is available at <http://schizophreniabulletin.oxfordjournals.org>.

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References

- Hahn B, Robinson BM, Harvey AN, et al. Visuospatial attention in schizophrenia: deficits in broad monitoring. *J Abnorm Psychol.* 2012;121:119–128.
- Elahipanah A, Christensen BK, Reingold EM. Visual search performance among persons with schizophrenia as a function of target eccentricity. *Neuropsychology.* 2010;24:192–198.
- Elahipanah A, Christensen BK, Reingold EM. Controlling the spotlight of attention: visual span size and flexibility in schizophrenia. *Neuropsychologia.* 2011;49:3370–3376.
- Hahn B, Robinson BM, Kaiser ST, et al. Kraepelin and Bleuler had it right: people with schizophrenia have deficits sustaining attention over time. *J Abnorm Psychol.* 2012;121:641–648.
- Hahn B, Hollingworth A, Robinson BM, et al. Control of working memory content in schizophrenia. *Schizophr Res.* 2012;134:70–75.
- Leonard CJ, Kaiser ST, Robinson BM, et al. Toward the neural mechanisms of reduced working memory capacity in schizophrenia. *Cereb Cortex.* 2013;23:1582–1592.
- Ball K, Owsley C. The useful field of view test: a new technique for evaluating age-related declines in visual function. *J Am Optom Assoc.* 1993;64:71–79.
- Ball K, Roenker DL. *UFOV Useful Field of View Manual.* San Antonio, TX: The Psychological Corporation; 1998.
- First MB, Spitzer RL, Miriam G, Williams JBW. *Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Patient Edition With Psychotic Screen.* New York, NY: New York State Psychiatric Institute, Biometrics Research; 2002.
- Pfohl B, Blum N, Zimmerman M. *Structured Interview for DSM-IV Personality Disorders (SIDP-IV).* Iowa City, IA: University of Iowa; 1995.
- Hyun JS, Woodman GF, Luck SJ. The role of attention in the binding of surface features to locations. *Vis cogn.* 2009;17:10–24.
- Carrasco M, Evert DL, Chang I, Katz SM. The eccentricity effect: target eccentricity affects performance on conjunction searches. *Percept Psychophys.* 1995;57:1241–1261.
- Edwards JD, Ross LA, Wadley VG, et al. The useful field of view test: normative data for older adults. *Arch Clin Neuropsychol.* 2006;21:275–286.
- Edwards J, Wadley V, Myers Re, Roenker DL, Cissell G, Ball K. Transfer of a speed of processing intervention to near and far cognitive functions. *Gerontology.* 2002;48:329–340.
- Edwards JD, Vance DE, Wadley VG, Cissell GM, Roenker DL, Ball KK. Reliability and validity of useful field of view test scores as administered by personal computer. *J Clin Exp Neuropsychol.* 2005;27:529–543.
- Johnson MK, McMahon RP, Robinson BM, et al. The relationship between working memory capacity and broad measures of cognitive ability in healthy adults and people with schizophrenia. *Neuropsychology.* 2013;27:220–229.
- Gold JM, Wilk CM, McMahon RP, Buchanan RW, Luck SJ. Working memory for visual features and conjunctions in schizophrenia. *J Abnorm Psychol.* 2003;112:61–71.
- Wilkinson GS, Robertson GJ. *Wide Range Achievement Test 4 Professional Manual.* Lutz, FL: Psychological Assessment Resources; 2006.
- Wechsler D. *Wechsler Test of Adult Reading (WTAR).* San Antonio, TX: The Psychological Corporation; 2001.
- Wechsler D. *Wechsler Abbreviated Scale of Intelligence.* San Antonio, TX: The Psychological Corporation; 1999.
- Nuechterlein KH, Green MF, Kern RS, et al. The MATRICS Consensus Cognitive Battery, part 1: test selection, reliability, and validity. *Am J Psychiatry.* 2008;165:203–213.
- Kern RS, Nuechterlein KH, Green MF, et al. The MATRICS Consensus Cognitive Battery, part 2: co-norming and standardization. *Am J Psychiatry.* 2008;165:214–220.
- Overall J, Gorham D. The brief psychiatric rating scale. *Psychol Rep.* 1962;10:799–812.
- Andreasen N. The Scale for the Assessment of Negative Symptoms (SANS): conceptual and theoretical foundations. *Br J Psychiatry.* 1989;155:49–58.
- Gold JM, Hahn B, Strauss GP, Waltz JA. Turning it upside down: areas of preserved cognitive function in schizophrenia. *Neuropsychol Rev.* 2009;19:294–311.
- Spencer KM, Nestor PG, Valdman O, Niznikiewicz MA, Shenton ME, McCarley RW. Enhanced facilitation of spatial attention in schizophrenia. *Neuropsychology.* 2011;25:76–85.
- Dickinson D, Ramsey ME, Gold JM. Overlooking the obvious: a meta-analytic comparison of digit symbol coding tasks and other cognitive measures in schizophrenia. *Arch Gen Psychiatry.* 2007;64:532–542.
- Andreasen NC, Pressler M, Nopoulos P, Miller D, Ho BC. Antipsychotic dose equivalents and dose-years: a standardized method for comparing exposure to different drugs. *Biol Psychiatry.* 2010;67:255–262.
- Green MF, Nuechterlein KH, Mintz J. Backward masking in schizophrenia and mania. I. Specifying a mechanism. *Arch Gen Psychiatry.* 1994;51:939–944.
- Butler PD, DeSanti LA, Maddox J, et al. Visual backward-masking deficits in schizophrenia: relationship to visual pathway function and symptomatology. *Schizophr Res.* 2003;59:199–209.
- Cheung V, Chen EH, Chen RL, Woo MF, Yee BK. A comparison between schizophrenia patients and healthy controls on the expression of attentional blink in a rapid serial visual presentation (RSVP) paradigm. *Schizophr Bull.* 2002;28:443–458.
- Wynn JK, Breitmeyer B, Nuechterlein KH, Green MF. Exploring the short term visual store in schizophrenia using the attentional blink. *J Psychiatr Res.* 2006;40:599–605.

33. Mathis KI, Wynn JK, Breitmeyer B, Nuechterlein KH, Green MF. The attentional blink in schizophrenia: isolating the perception/attention interface. *J Psychiatr Res.* 2011;45:1346–1351.
34. Mathis KI, Wynn JK, Jahshan C, Hellemann G, Darque A, Green MF. An electrophysiological investigation of attentional blink in schizophrenia: separating perceptual and attentional processes. *Int J Psychophysiol.* 2012;86:108–113.
35. Everling S, Krappmann P, Preuss S, Brand A, Flohr H. Hypometric primary saccades of schizophrenics in a delayed-response task. *Exp Brain Res.* 1996;111:289–295.
36. Leonard CJ, Robinson BM, Kaiser ST, et al. Testing sensory and cognitive explanations of the antisaccade deficit in schizophrenia. *J Abnorm Psychol.* 2013;122:1111–1120.
37. Gold JM, Fuller RL, Robinson BM, McMahon RP, Braun EL, Luck SJ. Intact attentional control of working memory encoding in schizophrenia. *J Abnorm Psychol.* 2006;115:658–673.
38. Vogel EK, McCollough AW, Machizawa MG. Neural measures reveal individual differences in controlling access to working memory. *Nature.* 2005;438:500–503.
39. Fukuda K, Vogel EK. Human variation in overriding attentional capture. *J Neurosci.* 2009;29:8726–8733.
40. Durstewitz D, Seamans JK. The dual-state theory of prefrontal cortex dopamine function with relevance to catechol-o-methyltransferase genotypes and schizophrenia. *Biol Psychiatry.* 2008;64:739–749.
41. Lisman JE, Coyle JT, Green RW, et al. Circuit-based framework for understanding neurotransmitter and risk gene interactions in schizophrenia. *Trends Neurosci.* 2008;31:234–242.
42. Rolls ET, Loh M, Deco G, Winterer G. Computational models of schizophrenia and dopamine modulation in the prefrontal cortex. *Nat Rev Neurosci.* 2008;9:696–709.