

Working Memory for Visual Features and Conjunctions in Schizophrenia

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The visual working memory (WM) storage capacity of patients with schizophrenia was investigated using a change detection paradigm. Participants were presented with 2, 3, 4, or 6 colored bars with testing of both single feature (color, orientation) and feature conjunction conditions. Patients performed significantly worse than controls at all set sizes but demonstrated normal feature binding. Unlike controls, patient WM capacity declined at set size 6 relative to set size 4. Impairments with subcapacity arrays suggest a deficit in task set maintenance: Greater impairment for supercapacity set sizes suggests a deficit in the ability to selectively encode information for WM storage. Thus, the WM impairment in schizophrenia appears to be a consequence of attentional deficits rather than a reduction in storage capacity.

Working Memory Capacity

Working memory impairments in patients with schizophrenia have been the focus of an intensive multidisciplinary research effort in the past decade. Behavioral studies have documented reliable deficits in a wide array of experimental paradigms that are thought to tap working memory (see Keefe, 2000, for a review). Furthermore, there have been several demonstrations that WM impairments in schizophrenia are related to patient performance on more cognitively complex measures of language comprehension, free recall, problem solving, and reasoning, suggesting that WM deficits may be implicated in the broad cognitive compromise that is characteristic of the illness (Condray, Steinhauer, van Kammen, & Kasperek, 1996; Gold, Carpenter, Randolph, Goldberg, & Weinberger, 1997; Perry et al., 2001; Stone, Gabrieli, Stebbins, & Sullivan, 1998). Functional neuroimaging studies have documented a range of prefrontal metabolic abnormalities in patients during the performance of several different WM tasks (Barch et al., 2001; Callicott et al., 2000; Carter et al., 1998; Manoach et al., 2000; Stevens, Goldman-Rakic, Gore, Fulbright, & Wexler, 1998). These abnormalities are most robust at higher working memory loads (e.g., 2-back vs. 1-back in *n*-back paradigms), suggesting that an understanding of the specific mechanisms that are implicated in WM deficits may provide an important window on the nature of cognitive dysfunction and the neurobiology of schizophrenia. The present study assesses the hypothesis that schizophrenia involves a reduction in WM capacity.

The term *capacity* has two distinctly different meanings in the context of WM systems. First, WM systems include a storage

component with a limited *storage capacity*, which determines how much information can be present in the system at one time. Additional processes may be recruited to facilitate information maintenance over time. The concept of storage capacity should not be confused with retention: Storage capacity refers to an amount of information, whereas retention refers to performance over time. Second, WM systems include a processing component (e.g., the *central executive* in the model of Baddeley, 1986); this component may be limited in its *processing capacity*, which refers to the amount of information that can be processed in a given period of time. These two types of capacity are analogous to a computer's memory capacity and processor speed, respectively, and they are conceptually independent: An individual may have a normal storage capacity and an impaired processing capacity, or vice versa. However, just as decreases in either the memory size or the processor speed of a computer will lead to decreased performance on most computing tasks, an impairment in working memory storage capacity may lead to performance deficits in behavioral tasks that are designed to tap processing capacity, and vice versa. For example, an individual with impaired processing capacity may exhibit deficits in digit-span tasks because impaired processing makes it difficult to encode and rapidly rehearse the digits in a manner that takes full advantage of the available storage capacity. Similarly, an individual with a reduced storage capacity may be unable to represent intermediate results in tasks such as mental arithmetic, leading to impaired performance. Deficits in either storage capacity or processing capacity would be expected to lead to impaired performance across a wide spectrum of everyday tasks, so both are important areas of inquiry. However, different neural systems may underlie the storage and processing components of WM; it is therefore important to determine whether schizophrenia involves deficits in the storage component, the processing component, or both. The present study was designed to determine the extent to which schizophrenia involves a specific deficit in storage capacity.

Reduced storage capacity should lead to a decrease in the number of items in a memory set that a subject can reproduce without error. In auditory digit-span tasks, for example, participants are presented with varying numbers of digits for subsequent

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recall, and an individual's span is defined as the longest string of digits that can be correctly reproduced. Although an impaired span may be the result of a decreased storage capacity, several other types of deficits can also explain an impaired span. First, verbal WM storage capacity appears to be limited to the amount of information that can be subvocally articulated in approximately 2 s (Baddeley, Thomson, & Buchanan, 1975; Schweickert & Boruff, 1986); individuals with reduced processing capacity/speed may simply be unable to articulate the digits as rapidly. Second, stimulus presentation and response production times increase when longer sequences are tested; failures at longer sequences may therefore result from impairments in retention rather than storage capacity. Finally, it is possible that deficits in selective attention could lead an individual to store and maintain irrelevant information in WM, leaving less capacity available for the digits. Thus, impairments in several specific processes could reduce estimates of storage capacity. These same issues also apply to ordered recall tasks, the *n*-back task, and variants of the sentence and computation span procedures that are common in the literature. Thus, although there is clear evidence that patients are impaired in such auditory-verbal tasks, it is not possible to conclude that the impairments are wholly or even partially due to a reduction in storage capacity per se.

Many recent studies of schizophrenia have explored visual WM rather than auditory-verbal WM, but these studies do not clearly address the nature of storage limitations. Much of the literature has focused on spatial WM, and impaired performance has been documented with tasks that require memory for one or two target locations (Carter et al., 1996; Park & Holzman, 1992; Tek et al., 2002). Impaired performance on such tasks would appear to implicate deficits in the precision of target encoding, enhanced susceptibility to interference, or failures of retention rather than storage capacity limitations because the actual storage demand (1 or 2 discrete items) imposed by such paradigms is minimal. Other studies have assessed storage capacity using variants of the Corsi blocks task, a visual analog to digit-span tasks in which the experimenter taps a set of blocks in a random sequence and the participant must reproduce the pattern of taps (Perry et al., 2001; Salame, Danion, Peretti, & Cuervo, 1998). However, reductions in span in this task are vulnerable to many of the same alternative explanations as are reductions in digit span.

The capacity issue has also been addressed in three studies using visual paradigms that appear to avoid these interpretive complications. These studies have used either grids or irregular spatial arrays with varying numbers of filled target items. Patients have either exhibited shorter spans or have deviated markedly from controls at larger set sizes (Dreher et al., 2001; Rizzo, Danion, Van Der Linden, Grange, & Rohmer, 1996; Salame et al., 1998). Although these results provide suggestive evidence of reduced storage capacity, they are complicated by encoding strategy effects. When a display contains a mix of filled and unfilled locations, particularly in a regular grid display, larger set sizes may have significant gestalt or configural properties, allowing participants to store an overall shape rather than the individual locations. Consequently, performance at higher set sizes may depend on a participant's ability to organize the information into a higher order chunk, and impaired performance may be the result of a deficit in the use of chunking strategies. This issue is particularly salient in light of evidence that schizophrenia patients have deficits in pro-

cessing configural properties of visual stimuli (Place & Gilmore, 1980; Silverstein et al., 1996). Thus, the empirical evidence for specific storage capacity limitations in schizophrenia, although suggestive, is not definitive.

Working Memory for Features and Conjunctions

The goal of the present study was to determine whether schizophrenia involves a reduction in the storage capacity of WM, using an experimental approach that minimizes the role of several potential confounds that might influence performance. Specifically, we used a visual change detection paradigm that has been studied extensively in normal individuals (e.g., Jiang, Olson, & Chun, 2000; Lee & Chun, 2001; Luck & Vogel, 1997; Simons & Levin, 1997; Vogel, Woodman, & Luck, 2001; Wheeler & Treisman, 2002). In this paradigm, illustrated in Figure 1, subjects first view a sample array containing varying numbers of simple, highly discriminable visual objects such as lines of varying orientation and/or color. Then, following a brief retention interval, a test array is presented and subjects are asked to indicate if the sample and test arrays were identical. The arrays are identical on 50% of trials, and one feature of one object changes on the other 50% of trials. In this paradigm, a participant with a storage capacity of *K* items should be perfectly accurate with arrays containing *K* or fewer items and should become progressively less accurate as the number of array items exceeds *K*. Pashler (1988) developed an equation for estimating *K* from this paradigm, and Cowan (2001) refined this formula to better account for the effects of guessing. Thus, this paradigm provides a direct means of estimating an individual's WM storage capacity.

Using variants of this paradigm, Luck, Vogel, and colleagues (Luck & Vogel, 1997; Vogel et al., 2001) found that healthy young adults have a visual WM capacity of only 3–4 objects (see also Cowan, 2001). This surprisingly small storage capacity did not appear to result from perceptual limitations because increasing the duration of the sample array by a factor of five did not enhance performance. In addition, capacity did not depend on the number of features that each object contained. That is, subjects could remember both the color and the orientation of a set of items just as accurately as they could remember only the color or only the orientation. Indeed, capacity was as great for objects defined by a conjunction of four features as it was for objects defined by a single feature. Similar results have been reported by other laboratories (Irwin & Andrews, 1996; Lee & Chun, 2001; Walker & Cuthbert, 1998; for limitations on this result, see Wheeler & Treisman, 2002, and Xu, 2002).

These findings suggest that integrated representations of objects are stored in visual WM rather than a set of independently represented features. The formation of such integrated representations first occurs at the level of perception; it is thought that spatially focused attention is required to achieve feature binding during perception (Treisman, 1996). In the absence of spatially focused attention, the large receptive fields of neurons in higher level areas of visual cortex lead to imprecision in coding the precise locations of object features, leading to errors in which features belonging to different objects are accidentally combined (Treisman, 1996). Perceptual-level binding does not appear to be impaired in schizophrenia: Previous studies of visual search for single-feature and conjunction targets have demonstrated that patients show normal

patterns of single-feature target “pop out” and largely normal reaction-time costs when searching for targets defined by feature conjunctions, and that patients were not more likely than controls to make perceptual errors due to miscombining or mislocalizing features (Carr, Dewis, & Lewin, 1998a, 1998b; Hess, Schu, Muller, & Schuttler, 1992; but see Mori et al., 1996).

However, a different mechanism may be used to encode and maintain bound representations in WM (Luck & Beach, 1998), and this mechanism may be impaired in schizophrenia. Specifically, computational simulations and electrophysiological recordings have suggested that integrated representations are maintained in WM by means of synchronized firing among the neurons that represent the individual features of each object (Lisman & Idiart, 1995; Luck & Vogel, 1998; Raffone & Wolters, 2001; Tallon-Baudry, Bertrand, & Fischer, 2001). Neural synchrony is often associated with gamma-band oscillations (Singer et al., 1997); several recent studies have demonstrated reduced gamma-band activity in schizophrenia (Clementz, Blumenfeld, & Cobb, 1997; Kwon et al., 1999). Therefore, it is possible that patients with schizophrenia may have an impairment in neural synchrony, which might lead in turn to a specific deficit in storing conjunctions in WM. One goal of the present study was to determine whether patients are more impaired for conjunctions than for simple features.

Experimental Design Considerations

As discussed above, it is difficult to measure an individual’s WM storage capacity independently of other factors, such as the individual’s processing capacity. Several aspects of the present experiment were designed to isolate WM storage capacity and to minimize other factors that might impair performance. First of all, Javitt, Liederman, Cienfuegos, and Shelley (1999) have shown that perceptual impairments may underlie deficits in several different WM tasks. To minimize the effects of perceptual impairments, the target stimuli in the present study were highly discriminable, and changes were very easy to detect at small set sizes. The shortest stimulus duration was 100 ms, which should be sufficient—given the simple and highly discriminable stimuli—to eliminate concerns over slowed perceptual processing (as documented in studies of backward masking; see Green & Nuechterlein, 1998, for review). In addition, we compared a 100-ms sample duration with a 500-ms sample duration to determine whether providing additional encoding time would eliminate any patient performance deficits. If patient performance is impaired due to a deficit in perception, then a fivefold increase in encoding time should lead to significant improvements in patient performance.

A second potential problem is that schizophrenia may involve deficits in the perception of configural information; to address this issue, we used sparse, random stimulus arrays that could not easily be encoded configurally. A third potential problem is that participants might use a strategy of encoding some of the items verbally and other items visually, leading to improved performance given independent, modality-specific storage systems. To minimize verbal encoding, the stimuli were presented briefly, and a given feature could occur more than once in a display (making it necessary to know the location of each feature, which is not easily encoded verbally). In addition, a previous study has shown that normal individuals do not engage in significant verbal recoding in

this paradigm, at least when the duration of the sample array is brief (Vogel et al., 2001).

A fourth potential problem is that impaired performance might reflect a deficit in maintenance rather than a reduction in storage capacity. To minimize the effects of maintenance deficits, we used a very short retention interval of only 2 s. Normal young adults show no significant loss of information in this paradigm with delays of up to 5 s (Vogel et al., 2001), so a 2-s delay should minimize any effects of maintenance deficits (especially compared with the 10- to 30-s intervals that have commonly been used in studies of schizophrenia; see, e.g., Keefe, Lees-Roitman, & Dupre, 1997; Park & Holzman, 1992). A fifth factor that can influence measures of WM capacity is the possibility that the act of reporting the contents of memory might interfere with the memory representation, especially in patients. The present paradigm avoids this problem (and most problems related to response selection and execution) by using a simple, unsped same–different response.

A sixth potential problem is that patients may have deficits in the operation of attentional processes that support different aspects of task performance. This problem is difficult to eliminate, but the present experimental design facilitates an examination of attentional impairments. There are at least two ways in which a deficit in attention might lead to impaired performance in this task. First, just as patients tend to miss occasional targets in the continuous performance task (Elvevag, Weinberger, Suter, & Goldberg, 2000; Nuechterlein, 1991), they may entirely fail to encode the sample array on a subset of trials in the present task. Such a deficit in the ability to sustain attention would logically lead to an equivalent proportional impairment regardless of condition or set size. Second, patients may have specific deficits in the use of attention to selectively encode relevant information. Such a deficit should be most evident at higher set sizes. When memory array sizes are below storage capacity, it is likely that bottom-up processes alone may be sufficient to guide encoding (see, e.g., Schmidt, Vogel, Woodman, & Luck, 2002). When confronted with a supercapacity array, however, it may be necessary to select only a subset of target items for storage, thereby avoiding an unsuccessful attempt to encode all items. Normal subjects demonstrate nearly identical WM capacity across different supercapacity array sizes (Vogel et al., 2001), indicating that the process of attentional selection is typically highly efficient. A deficit in this selective attention process in patients should specifically impair performance for supercapacity arrays, with minimal or no effect for arrays at or below capacity. Thus, deficits observed on array sizes that are within capacity would appear to implicate processes related to sustaining attention (or maintaining an accurate task representation), whereas deficits that are magnified by supercapacity arrays suggest an impairment in selective attention. Thus, it is possible to assess the presence of different types of attentional impairments by assessing performance with sample arrays that are below and above capacity.

It is possible to anticipate four possible patterns of results on the basis of previous findings in the schizophrenia literature. First, if patients have a deficit in the maintenance of neural synchrony—as might be expected given previous findings of reduced gamma-band activity—then severe impairments should be evident with feature-conjunction stimuli relative to single-feature stimuli across all set sizes. With a severe binding impairment, performance with a given number of conjunction targets should be approximately equal to that observed with twice as many feature targets. Second,

if patients have a simple reduction in storage capacity, a pattern of asymptotic deficits at higher set sizes in both single-feature and conjunction conditions would be expected. Note that a reduction in storage capacity should not affect performance at small set sizes, which are presumably within the capacity limits of even severely impaired patients. Third, in contrast, if patients have a deficit in the ability to sustain attention or maintain an accurate task representation, this would be expected to slightly degrade performance across all set sizes and stimulus conditions. There is no clear theoretical basis for predicting that such a sustained attention deficit would be magnified with increasing array sizes. Fourth, if patients have a deficit in the ability to use selective attention to guide WM encoding, this should impact performance specifically with supercapacity arrays, with a minimal, if any, effect on performance below capacity. Thus, a deficit in selective attention should be evident when the demand for selection is highest: when confronted with supercapacity arrays.

Method

Subjects

The patient participants ($N = 20$) were recruited from the Maryland Psychiatric Research Center outpatient research program and included 14 men and 6 women, 11 of whom were Caucasian, and 9 who were African American. They met criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (4th edition; *DSM-IV*; American Psychiatric Association, 1994) for schizophrenia or schizoaffective disorder. Diagnosis was established using a best-estimate approach in which information from the *Structured Clinical Interview for DSM-IV* (First, Spitzer, Gibbon, & Williams, 1997) was supplemented by information from family informants, previous psychiatrists, and medical records. Patients with organic brain disorders, mental retardation, history of significant head trauma, or a recent history of alcohol or substance abuse/dependence were excluded from participation. All patients had normal or corrected-to-normal visual acuity. All patients were judged to be clinically stable by their therapist and were assessed while receiving antipsychotic medication (11 received new generation antipsychotics, and 9 received conventional or "typical" antipsychotics).

A group of 18 healthy controls (7 men and 11 women: 11 Caucasians and 7 African Americans) participated in the study. These participants were recruited from the community using newspaper and radio advertisements. Control participants were free from a current or past *DSM-IV* Axis I or Axis II disorder and did not have a family history of psychotic illness as determined by *SCID-IV* (Pfohl, Blum, & Zimmerman, 1997) and Family Research Diagnostic Criteria interviews (Andreasen, Rice, Endicott, Reich, & Coryell, 1986). Subjects with organic brain disorders, mental retardation, history of significant head trauma, or a recent history of alcohol or substance abuse/dependence were excluded from participation. All controls had normal or corrected-to-normal visual acuity.

The two groups were similar in age: patients, $M = 36.2$ ($SD = 7.2$); controls, $M = 38.9$ ($SD = 10.8$), but differed significantly in years of education: patients, $M = 12.5$ ($SD = 2.1$); controls, $M = 14.4$ ($SD = 2.0$), $t(36) = 2.9$, $p < .01$. The groups also differed on the Reading subtest of the Wide Range Achievement Test—3 (WRAT-3; Wilkinson, 1993): patients, $M = 93.0$ ($SD = 13.8$); controls, $M = 102.3$ ($SD = 9.9$), $t(36) = 2.4$, $p < .05$. The WRAT-3 performance of the control group closely approximated the standardization sample mean of 100, and the average score for the patient group was clearly in the normal range.

All subjects were provided a complete description of the proposed study and gave written informed consent prior to study participation. Patients were required to demonstrate an understanding of study demands, risks, and their right to withdraw in response to probe questions prior to signing

consent documents. The Institutional Review Board of the University of Maryland School of Medicine approved the study protocol and consent procedures. The healthy control participants were compensated for study participation.

Materials and Procedure

The change detection task was modeled after the task originally used by Luck and Vogel (1997), illustrated in Figure 1. Each trial consisted of the presentation of a sample array of colored bars followed by a 2,000-ms delay, then by a test array (4,000 ms). The sample and test arrays were either identical or differed by one feature, each occurring on 50% of the trials. Subjects responded by pressing a button to indicate if the test array was the same or different from the previous sample array. Subjects were instructed to emphasize accuracy, and no mention was made of a need to respond quickly. Testing was completed in a 1-hr session, with a practice block preceding testing and a mandatory break after 30 min of testing. All subjects demonstrated an ability to distinguish the colors and orientations used in the experiment by making correct responses on at least 75% of the practice trials, which included items from each set size and encoding condition.

All stimulus displays were presented within a $10.23^\circ \times 7.20^\circ$ region on a video monitor with a white background at a viewing distance of 70 cm. Each colored bar was randomly presented in this area, with a minimum separation of 3° . The bars were $1.58^\circ \times 0.156^\circ$, and the color of each bar was selected at random (with replacement) from a set of four highly discriminable colors: red, blue, green, and black. The orientation of each bar was selected randomly (with replacement) from a set of four orientations: 0° , 45° , 90° , and 135° from vertical. Color and orientation were selected independently for each bar.

Each participant was tested with set sizes of 2, 3, 4, and 6 bars. Each set size was tested with a separate block of 96 trials, with the 4 blocks presented in a random order across subjects. Each block contained 32 trials in each of the three conditions—color, orientation, and conjunction—presented in that order. In the color condition, the color of one item could be different on the test trials from the sample array: in the orientation condition, the orientation of one item could be altered from sample to test array. In the conjunction condition, either one color or one orientation could be changed. Within each of these three conditions, 50% of the sample arrays were randomly presented for 100 ms, and 50% were randomly presented for 500 ms. The experiment included a total of 384 trials and comprised a 2 (group) between-subjects and 4 (set size) \times 3 (condition) \times 2 (exposure duration) within-subjects mixed model design.

Statistical Methods

The major analyses were performed using two different measures of performance. First, we computed A' , a nonparametric measure of sensitivity (comparable to the parametric measure d') that is widely used in signal detection experiments (Stanislaw & Todorow, 1999). A' has a maximum value of 1.0, and a value of .50 represents chance performance. A' scores were calculated for each participant at each level of condition, stimulus duration, and set size, using the formulas: $A' = .50 + (H - F)/(1 + H - F)/4H(1 - F)$, when hit rate (H) \geq false alarm rate (F); and $A' = .50 - (F - H)/(1 + F - H)/4F(1 - H)$, when $F > H$ (Stanislaw & Todorow, 1999, Equation 2). The hit rate was computed as the proportion of correct responses in pairs of trials where the two test stimuli were the same. The false alarm rate was computed as the proportion of incorrect responses in pairs of trials where the two test stimuli were different. A' is not defined when either H or F has a value of 0 or 1, so values of 0 were replaced with $.50/n$, and values of 1 were replaced with $(n - .50)/n$, where n was the number of trials contributing to the score.

The second measure of performance was K , which is an estimate of the average number of items that the observer held in memory on a typical

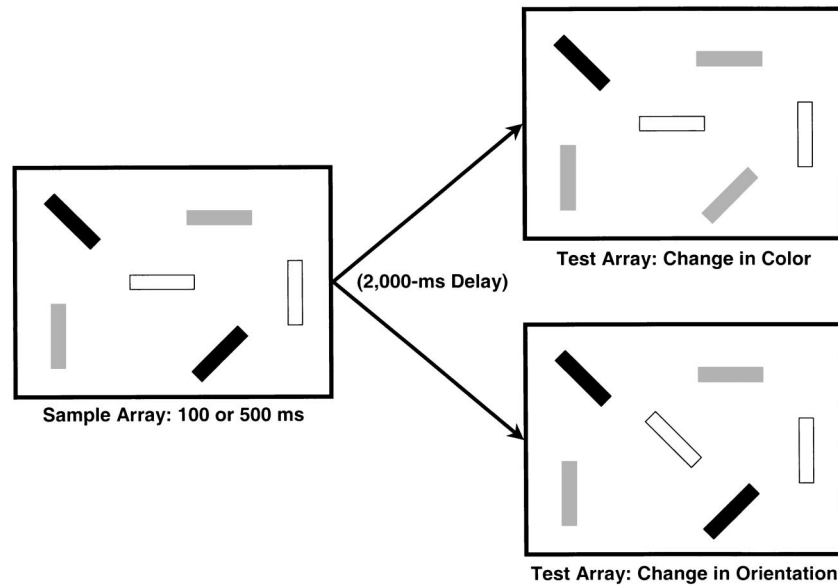


Figure 1. Sample stimuli and trial sequence. The sample array was presented for 100 or 500 ms. Following a 2,000-ms delay, a test array was presented. On 50% of trials, one element was different on the test array, as seen with a single color change in the top test array or a single orientation change in the bottom array. Actual test stimuli were red, blue, green, and black.

trial. A formula for estimating K was first described by Pashler (1988), and an improved formula was proposed by Cowan (2001). The logic behind these formulas is simple: If an observer remembers K items from a display containing n items, then the observer should be able to detect a change in one of the n items on K/n trials. Observers will also guess; the two formulas for estimating K differ in their assumptions about guessing. Cowan's (2001) formula is clearly more appropriate, so this is the formula we used. It is defined as $K = S \times (H - F)$, where S is the set size. Values of H and F that were equal to 0 and/or 1 were adjusted in the same manner as in the A' calculations. K can be no greater than the set size of the array; it can therefore be used as an estimate of storage capacity only when the set size exceeds the observer's capacity. Therefore, these scores were calculated only for set sizes of 4 or 6.

The data were analyzed using the generalized estimating equations (GEE) method (Liang & Zeger, 1986) for repeated measures, which is a more general, flexible version of a repeated measures analysis of variance (ANOVA). An initial model included all possible interactions of diagnosis, encoding condition, stimulus duration, and set size. This was simplified using a backward stepwise procedure to eliminate one term at a time, starting with the highest order interactions and dropping terms for which $p > .05$ unless they were involved in higher order interactions containing that term. All main effects were retained in the final model. When significant ($p < .05$) interactions were found, post hoc tests were used to further explore differences among factors involved in the interactions, conducted as follows. Mean values at each combination of all levels of the two factors were calculated from equally weighted averages of the means for each cell over all other factors not involved in the interaction. These estimated means were used to explore how differences between levels of one factor varied across levels of the second factor involved in an interaction. For example, the estimated mean difference between healthy controls and patients was calculated at each level of set size. The squared difference divided by a robust estimate of the variance of the difference was used to calculate a one degree of freedom chi-square test for the statistical significance of the difference. Similarly, mean differences were calculated within each group between successive levels of set size, and appropriate contrasts on these estimates were used to compare the effects of specific

changes in set size in controls and patients. All post hoc tests following a significant test of interaction were conducted using an unadjusted $p < .05$ as a criterion for significant differences. Separate analyses were conducted for A' and K , the only difference being that only two levels of set size (4 and 6) were used for the K analyses.

Results

A' Analyses

Model selection steps for the A' analysis are summarized in Table 1. Neither the four-way (Diagnosis \times Condition \times Set Size \times Stimulus Duration) nor any of the three-way interactions were statistically significant (all $ps > .05$). The final model included the main effects of condition, set size, and diagnosis as well as the statistically significant two-way interactions between diagnosis and set size and between condition and set size. The main effect of duration was not significant, nor was it involved in any significant higher order interactions.

Table 2 presents the A' results for patients and controls for each encoding condition and set size, collapsed across sample duration. Figure 2 shows the effects of set size on A' for patients and controls, collapsed across encoding condition and sample duration; Figure 3 shows the effects of encoding condition on A' for patients and controls, collapsed across set size and sample duration. As seen in Figure 2, healthy controls were more accurate than patients at all set sizes (minimum $p = .006$). Averaged across condition, duration, and set size, the estimated mean (\pm standard error) A' scores were .82 ($SE = .01$) in patients with schizophrenia and .89 ($SE = .01$) in healthy controls, $\chi^2(1, N = 38) = 26.44, p < .001$. This significant main effect of diagnosis was not modified by any two- or three-way interaction effects involving stimulus exposure duration or encoding condition. Thus, there is no evidence that

Table 1
Model Selection Using Repeated Measures (GEE) Analysis of A' Scores

Backward selection step	Terms deleted	χ^2	df	p
1	Condition \times Set Size \times Duration \times Diagnosis	5.16	6	.52
2	Set Size \times Duration \times Diagnosis	1.38	3	.71
3	Condition \times Duration \times Diagnosis	0.99	2	.61
4	Condition \times Set Size \times Diagnosis	8.34	6	.21
5	Condition \times Set Size \times Duration	11.77	6	.07
6	Duration \times Diagnosis	0.02	1	.88
7	Set Size \times Duration	3.44	3	.33
8	Condition \times Diagnosis	3.50	2	.17
9	Condition \times Duration	4.42	2	.11

Final model	Terms retained	χ^2	df	p
	Condition	18.24	2	<.001
	Set size	34.14	3	<.001
	Duration	1.59	1	.21
	Diagnosis	15.52	1	<.001
	Set Size \times Diagnosis	16.86	3	<.001
	Condition \times Set Size	15.91	6	.01

Note. $N = 38$ for all analyses. GEE = generalized estimating equations.

patients differed from controls in the ability to bind features with conjunction stimuli. Similarly, there is no evidence that the patients' performance was limited by perceptual encoding speed under these conditions: overall A' scores for controls at the 100- and 500-ms exposures were .89 ($SE = .01$) and .90 ($SE = .01$), respectively ($ps = .20$). In the patient group, A' scores for the 100- and 500-ms exposures were .83 ($SE = .01$) and .83 ($SE = .01$) respectively ($ps = .48$).

As seen in Figure 2, there was a clear decrease in A' with each increase in set size. These decreases were statistically significant

Table 2
 A' Scores by Set Size, Condition, and Diagnosis

Set size and condition	Healthy participants		Patients with schizophrenia	
	M	SE	M	SE
2				
Conjunction	.946	.005	.900	.023
Orientation	.944	.006	.917	.009
Color	.957	.002	.937	.006
3				
Conjunction	.896	.013	.850	.016
Orientation	.918	.009	.873	.018
Color	.942	.006	.917	.012
4				
Conjunction	.888	.011	.842	.013
Orientation	.892	.013	.793	.023
Color	.923	.010	.899	.015
6				
Conjunction	.764	.027	.658	.033
Orientation	.807	.018	.623	.032
Color	.850	.014	.725	.028

Note. Least square means and standard errors, collapsed across duration, are presented from the model. $A' = \text{duration} + \text{condition} + \text{diagnosis} + \text{set size} + (\text{Set Size} \times \text{Condition}) + (\text{Set Size} \times \text{Diagnosis}) + (\text{Condition} \times \text{Diagnosis}) + (\text{Set Size} \times \text{Condition} \times \text{Diagnosis})$.

for each comparison (i.e., set sizes 2 vs. 3, 3 vs. 4, and 4 vs. 6, averaging across durations and encoding conditions) for both patients and controls (all $ps < .02$). However, the magnitude of A' decrease differed as a function of diagnosis: We observed a significant Group \times Set Size interaction effect ($p = .001$). Although patients showed a slightly larger decline in A' than controls for each increase in set size, the difference in the size of the set size effect between groups was significant only in the change from set sizes 4 to 6 ($p < .001$).

None of the other significant main or interaction effects involved diagnosis; therefore, these effects are only briefly summarized. We observed a significant main effect of encoding condition, which resulted from the color condition being significantly easier (overall $A' = .89$; see Figure 3) than either the orientation

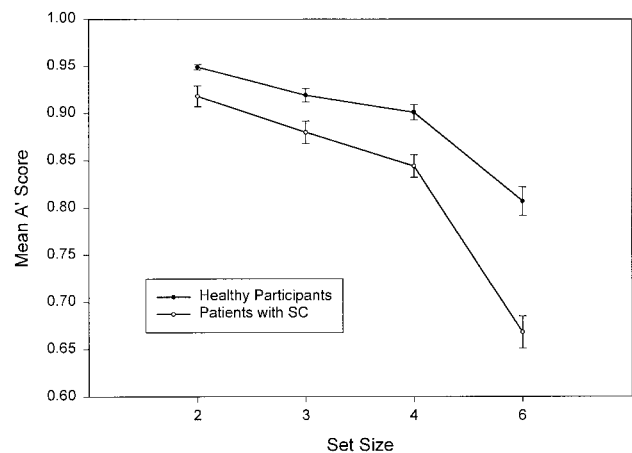


Figure 2. Mean A' at set sizes 2, 3, 4, and 6 by diagnosis. The mean A' ($\pm SE$) values were calculated after collapsing across encoding conditions and exposure durations. Patients performed significantly worse than healthy participants at each set size (all $ps < .01$). SC = schizophrenia.

($A' = .85$) or the conjunction condition ($A' = .84$). In both groups, performance in the color condition was significantly higher than the orientation or conjunction condition, which did not differ from each other (see Figure 3). Note that patient performance was slightly higher in the conjunction condition than in the orientation condition (A' scores of .81 and .80, respectively), suggesting that our failure to document a Group \times Encoding Condition interaction was not likely to be the result of a lack of statistical power. The overall condition effect was slightly smaller at smaller set sizes, presumably due to a ceiling effect, which led to a significant interaction between set size and encoding condition (see Tables 1 and 2).

K Analyses

The analyses of the K scores was highly consistent with the A' analyses. None of the three- or four-way interactions was statistically significant, so these interactions were dropped from the final model. After these terms were dropped, the final model included two significant two-way interactions (Diagnosis \times Set Size and Condition \times Duration) and significant main effects for condition, set size, and diagnosis. The main effects for encoding condition (color easier than orientation or conjunction) and the Condition \times Duration interaction (color condition easier at longer exposure) precisely parallel those obtained using A' scores and are not discussed further.

Mean K scores are shown in Figure 4 for set sizes 4 and 6, collapsed across encoding condition and duration. The control group had a mean K of 2.85 ($SE = 0.20$) at set size 4 and a nearly identical mean K of 2.89 ($SE = 0.11$) at set size 6. For the patient group, however, the mean K was 2.30 ($SE = 0.13$) at set size 4, but it dropped precipitously to 1.44 ($SE = 0.16$) at set size 6. The overall difference in K led to a significant main effect of diagnosis, $\chi^2(1, N = 38) = 15.46, p < .001$, and the drop in K at set size 6 in the patient group led to a significant Diagnosis \times Set Size

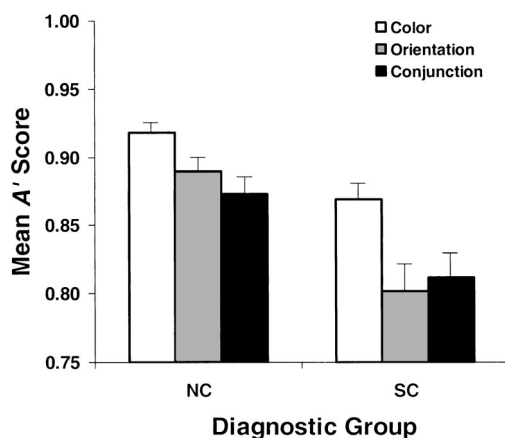


Figure 3. Mean A' values for single-feature and conjunction targets. The mean A' ($\pm SE$) values were calculated after collapsing across set sizes and exposure durations. Performance in the color condition was significantly higher than in either the orientation or conjunction condition for both groups (all $ps < .005$). The orientation and conjunction conditions did not differ significantly in either group. NC = nonschizophrenia controls; SC = schizophrenia.

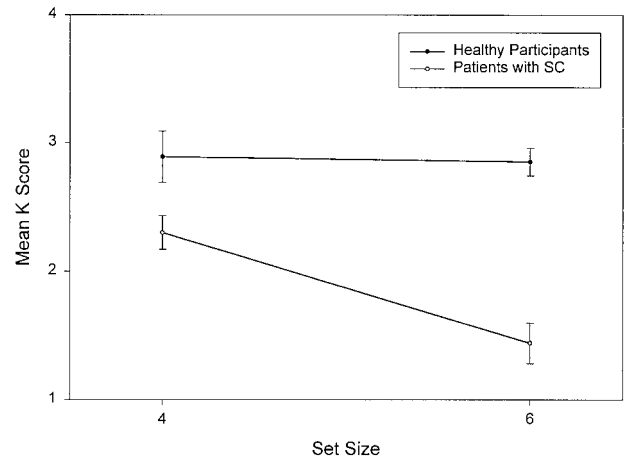


Figure 4. Mean K ($\pm SE$) scores by diagnosis. The mean K scores were calculated after collapsing across encoding conditions and exposure durations. The decline in patient performance at set size 6 resulted in a significant Group \times Set Size interaction effect ($p < .001$). SC = schizophrenia.

interaction, $\chi^2(1, N = 38) = 11.50, p < .001$. These results indicate that patients generally store less information in WM than controls and that patients actually store less information from large arrays than from intermediate arrays, whereas control performance does not decline.

Discussion

The results of this study demonstrate that patients with SC have marked WM deficits, including clear deficits in the amount of information they store in WM, which is consistent with previous reports in the literature. However, we believe that these results extend the current literature in demonstrating important areas of fully intact functioning in schizophrenia as well as suggesting a critical role for abnormalities in the function of attention in the selection of representations that are encoded into WM, issues we now discuss in turn.

In both patients and controls, the information stored in WM appeared to consist of integrated representations of objects rather than independent features, consistent with previous research in normal individuals (Lee & Chun, 2001; Luck & Vogel, 1997; Vogel et al., 2001; but see Wheeler & Treisman 2002, and Xu, 2002). This conclusion is directly supported by the fact that similar performance was observed in the conjunction and single-feature (orientation) conditions in both subject groups. If features were coded independently, A' scores in the conjunction condition should be dramatically worse than in the single-feature condition: The conjunction stimuli would demand twice as much storage capacity at each set size as the single-feature conditions. The fact that single-feature and conjunction conditions yield similar A' values within each size clearly argues against the notion of independent feature rather than integrated storage (see Table 2). Comparisons across set sizes and conditions are also instructive. If features were coded independently, performance with conjunction stimuli at set sizes 2 and 3 should closely resemble single-feature performance at set sizes 4 and 6, respectively. A brief inspection of Table 2 is

sufficient to demonstrate that conjunction performance at smaller set sizes far exceeds single-feature performance at the larger set sizes, again demonstrating that WM representations appear to be integrated.

The fact that performance was significantly better in the single-feature color condition than in the orientation or conjunction condition does not undermine the claim but merely indicates that color was easier to remember than orientation. This is clear evidence that patients are able to bind features into integrated object representations for WM storage, at least with the type of two-feature conjunction stimuli used in the present study. Therefore, the WM capacity limitation in schizophrenia does not appear to result from a failure in feature binding that could dramatically reduce storage capacity. This extends previous work demonstrating the intact operation of attention in feature binding in the context of visual search paradigms among patients with schizophrenia (Carr, Dewis & Lewin, 1998a, 1998b). This evidence of intact binding should serve to constrain the interpretation of gamma-band abnormalities in schizophrenia: Patients are fully able to form integrated, bound representations in the service of visual perception and maintain these representations in WM for at least the 2-s delay interval tested here.

The patient WM deficit documented here does not appear to result from perceptual limitations. If performance had been limited by perceptual factors, then increasing the amount of time available for perception should have led to improved performance in the schizophrenia group. However, a fivefold increase in the duration of the sample array did not substantially improve their performance. Moreover, studies of normal individuals have found that conjunction stimuli are more perceptually demanding than feature stimuli (e.g., Treisman & Gelade, 1980); but the SC group did not show a greater impairment in the conjunction condition than in the two single-feature conditions. Thus, WM capacity limitations in this type of change detection paradigm do not appear to be related to limitations in the quality or precision of the perceptual representation of the sample display.

The patient group diverged from the controls in two qualitatively different ways. First, patients performed worse than controls at all set sizes, including those below their capacity. Second, the patients demonstrated an exaggerated performance decrement at the highest set sizes, as evidenced by the significant Group \times Set Size interaction effect seen with both the A' and K measures. Specifically, patient capacity, as assessed by the K scores, decreased significantly from set size 4 to set size 6, whereas K was stable across set sizes in controls.

This pattern of between-groups differences is difficult to explain with a simple reduction in storage capacity alone. First, a simple reduction in storage capacity should not produce a deficit when the set size is below the patient group's capacity; yet, the patients performed worse than the controls at set size 2, a load that must be considered within capacity for the patients (as evidenced by K scores > 2 at set size 4). Second, a simple capacity reduction should lead to a lower asymptotic K value in the patients; it cannot explain the substantial decline that was observed as the set size increased from 4 to 6 items.

The fact that patient K scores declined at set size 6 relative to that observed at set size 4 suggests that patient capacity fluctuates in relationship to the need to selectively encode items into WM. That is, when presented with an array size that clearly exceeds

WM capacity, participants can encode only some of the items from the array. When confronted with this demand for attentional selection, patient performance declines relative to that observed with a display size that presents less of a demand for selective encoding. Thus, it appears that patient WM capacity may be dynamically related to the function of selective attention. This type of attentional impairment can create the appearance of a simple capacity reduction: Selective attention is most critical in the face of high loads, and a deficit in attention results in failures to utilize potential storage capacity efficiently. This emphasis on the role of selective attention is consistent in spirit with the model proposed by Engle, Kane, and Tuholski (1999), who emphasized the critical role of "controlled attention" in mediating individual differences in WM capacity and the role of WM in more complex forms of cognition such as reasoning.

Previous evidence supports the hypothesis that patients demonstrate an impairment in the use of selective attention to guide WM encoding using an auditory WM paradigm. Weiss, Vrtunski, and Simpson (1988) first determined the individual maximal auditory span for a group of patients and controls and then examined recall when subjects were presented with superspan lists (lengths of up to 13 items were tested). Superspan recall was considered as a percentage of maximal span, thereby adjusting for between-groups differences in simple span. Unlike psychiatric controls, patients demonstrated a dramatic reduction in performance when faced with the challenge of superspan lists, frequently recalling 3 or fewer items from lists of 7 or more items even though their mean recall was 6.36 items. Thus, when faced with the need to selectively encode a subset of the information, patient capacity declined dramatically, a result remarkably similar to what we observed using a very different type of paradigm.

To summarize, the overall pattern of results from the present experiment suggests that two different factors are responsible for the impaired performance of the patients relative to the controls. First, just as patients miss occasional targets in the continuous performance test, they may occasionally fail to encode the sample array in the present task, leading to errors at all set sizes. This may be conceived of as a deficit in sustained attention or in the ability to maintain a high-quality representation of the task context (see, e.g., Cohen & Servan-Schreiber, 1992). Second, patients appear to have a deficit in selective attention, making it difficult for them to selectively store a subset of the array when the array exceeds capacity. These sustained attention and selective attention deficits both lead to a reduction in the amount of task-relevant information that the patients can store in WM. However, the present study provides no evidence for a simple reduction in WM storage capacity per se. That is, patients may have the same potential WM storage capacity as the controls but are unable to use this capacity effectively.

Highlighting the possible role of attention in the WM impairment of schizophrenia does not rule out the existence of deficits in perceptual encoding or operations involved in WM maintenance or manipulation. It is clear that different experimental paradigms pose challenges to different aspects of the distributed system that mediates WM and are therefore likely to elicit somewhat different and, at times, contradictory results. We do believe, however, that simple explanations of schizophrenia WM impairment in terms of maintenance-related processes alone, or some type of storage capacity limitation alone, simply do not fit many of the accumu-

lated findings in the literature. Our data suggest that a full account of WM limitations in schizophrenia needs to consider the role of attentional processes that are involved in the selective encoding of perceptual representations into WM.

Studies of patients with schizophrenia inevitably are complicated by the role of medication effects. The current patients were not randomly assigned to treatment and were receiving clinically determined medication type and dose. This precludes any meaningful analysis of potential medication effects because patient clinical variables were confounded with medication type and dose. However, it seems highly unlikely that negative medication effects played a major role in the current results, given that WM deficits have been documented in medication-naïve samples (Barch et al., 2001). Furthermore, there is very little evidence that antipsychotic medications have such negative effects in patients with schizophrenia; they may indeed have some positive effects, including in short-term memory paradigms that impose a demand to selectively encode target versus distractor items (Blyler & Gold, 2000; Green et al., 1997; Keefe, Silva, Perkins, & Lieberman, 1999). Generalizing about medication effects is potentially misleading, as there is preliminary evidence that the new generation of medications may have a variety of subtle beneficial and negative cognitive effects (Meltzer & McGurk, 1999). The fact that a variety of WM deficits have been documented in the unmedicated, nonpsychotic relatives of patients (Conklin, Curtis, Katsanis, & Iacono, 2000; Park, Holzman, & Goldman-Rakic, 1995) argues strongly against the possibility that negative treatment effects explain deficits observed in patient groups. Therefore, WM impairment in patients cannot be easily attributed to treatment or symptom effects and instead appears to be a marker of the disease. However, it is clearly possible that findings in the literature based on different WM tasks and study groups may not generalize to the current task, and in the absence of empirical data addressing this issue, the possibility that our results reflect a negative (or positive) medication effect cannot be definitively ruled out.

In studies of schizophrenia patients, researchers are also inevitably confronted with the problem of the generalized deficit (see recent discussions by Chapman & Chapman, 2001; Strauss, 2001). That is, patients perform more poorly than controls on most demanding cognitive tasks, and failure to match tasks on discriminating power can yield artifactual evidence of specific rather than general cognitive deficits. It is difficult to meaningfully account for the present data in terms of a generalized deficit without implicating WM. First, we observed differences at all set sizes, including small set sizes where control performance approached ceiling, an area of presumed diminished psychometric sensitivity. Thus, there is clear evidence of impaired WM performance. Second, although the interaction between diagnosis and set size for the A' measure can be explained on the grounds of greater sensitivity at larger set sizes, the decline in K at set size 6 cannot be explained in this manner. That is, although K remained constant from set size 4 to set size 6 among controls, it dropped substantially for the patient group. Thus, the decline in K at set size 6 cannot be attributed to an increase in task difficulty and psychometric sensitivity. Instead, it is apparently an abnormality in selective attention that makes the task difficult, but only for the patient group.

This evidence of a specific impairment in the operation of selective attention in WM may have broad implications: Some type of limited-capacity storage/processing system is considered a

critical part of any cognitive architecture, and impairment in this component would have widespread ramifications. Therefore, a single and specific impairment in this system could produce impairment on a wide variety of cognitive tasks. That is, the deficit may be confined to a specific process and yet may be generalized in impact across tasks, given the central role of the WM system in many aspects of cognition.

It is important to highlight the fact that these data also suggest an area of intact function in schizophrenia. Patients appear to bind independent features into integrated object representations in a similar fashion as healthy controls. It is thought that this process requires the operation of controlled attentional processing. Although attention is widely considered to be an area of important impairment in schizophrenia, it appears that the function of attention in feature binding is spared, consistent with previous studies of visual search (Carr, Dewis, & Lewin, 1998a, 1998b). In contrast, we hypothesize that the deficit in overall WM performance may be attributable to a different attention-dependent process, the process of selection for WM encoding and storage. This pattern of impaired/preserved functions of attention illustrates the multifaceted nature of the construct and illustrates the need to define attention in terms of specific processes operating within specific cognitive systems (Luck & Vecera, 2002). The fact that different aspects of attention may be dissociable argues strongly against the notion of a unified attentional system and instead suggests that different forms of attentional processing are implemented in different neural systems. Demonstrating areas of intact function, such as feature binding, is as important as documenting deficits in the cognitive/anatomic mapping of the functioning of attention in schizophrenia.

References

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington DC: American Psychiatric Association.
- Andreasen, N. C., Rice, J., Endicott, J., Reich, T., & Coryell, W. (1986). The family history approach to diagnosis: How useful is it? *Archives of General Psychiatry*, *43*, 421–429.
- Baddeley, A. D. (1986). *Working memory*. New York: Oxford University Press.
- Baddeley, A. D., Thomson, N., & Buchanan, M. (1975). Word length and the structure of short-term memory. *Journal of Verbal Learning and Verbal Behavior*, *14*, 575–589.
- Barch, D. M., Carter, C. S., Braver, T. S., Sabb, F. W., MacDonald, A., III, Noll, D. C., & Cohen, J. D. (2001). Selective deficits in prefrontal cortex function in medication-naïve patients with schizophrenia. *Archives of General Psychiatry*, *58*, 280–288.
- Blyler, C. R., & Gold, J. M. (2000). Cognitive effects of typical antipsychotic treatment: Another look. In T. Sharma & P. D. Harvey (Eds.), *Cognition in schizophrenia: Impairments, importance, and treatment strategies* (pp. 241–265). Oxford, England: Oxford University Press.
- Callicott, J. H., Bertolino, A., Mattay, V. S., Langheim, F. J. P., Duyn, J., Coppola, R., et al. (2000). Physiological dysfunction of the dorsolateral prefrontal cortex in schizophrenia revisited. *Cerebral Cortex*, *10*, 1078–1092.
- Carr, V. J., Dewis, S. A. M., & Lewin, T. J. (1998a). Illusory conjunctions and perceptual grouping in a visual search task in schizophrenia. *Psychiatry Research*, *80*, 69–81.
- Carr, V. J., Dewis, S. A. M., & Lewin, T. J. (1998b). Preattentive visual search and perceptual grouping in schizophrenia. *Psychiatry Research*, *79*, 151–162.

- Carter, C. S., Perlstein, W., Ganguli, R., Brar, J., Mintun, M., & Cohen, J. D. (1998). Functional hypofrontality and working memory dysfunction in schizophrenia. *American Journal of Psychiatry*, *155*, 1285–1287.
- Carter, C., Robertson, L., Nordahl, T., Chaderjian, M., Kraft, L., & O'Shara-Celaya, L. (1996). Spatial working memory deficits and their relationship to negative symptoms in unmedicated schizophrenia patients. *Biological Psychiatry*, *40*, 930–932.
- Chapman, L. J., & Chapman J. P. (2001). Commentary on two articles concerning generalized and specific cognitive deficits. *Journal of Abnormal Psychology*, *110*, 31–39.
- Clementz, B. A., Blumenfeld, L. D., & Cobb, S. (1997). The gammaband response may account for poor P50 suppression in schizophrenia. *Neuroreport*, *22*, 3889–3893.
- Cohen, J. D., & Servan-Schreiber, D. (1992). Context, cortex, and dopamine: A connectionist approach to behavior and biology in schizophrenia. *Psychological Review*, *99*, 45–77.
- Condray, R., Steinhauer, S. R., van Kammen, D. P., & Kasperek, A. (1996). Working memory capacity predicts language comprehension in schizophrenic patients. *Schizophrenia Research*, *20*, 1–13.
- Conklin, H. M., Curtis, C. E., Katsanis, J., & Iacono, W. G. (2000). Verbal working memory impairment in schizophrenia patients and their first-degree relatives: Evidence from the digit span task. *American Journal of Psychiatry*, *157*, 275–277.
- Cowan, N. (2001). The magical number 4 in short-term memory: A reconsideration of mental storage capacity. *Behavioral and Brain Sciences*, *24*, 87–185.
- Dreher, J.-C., Banquet, J.-P., Allilaire, J.-F., Paillere-Martinot, M.-L., Dubois, B., & Burnod, Y. (2001). Temporal order and spatial memory in schizophrenia: A parametric study. *Schizophrenia Research*, *51*, 137–147.
- Elvevag, B., Weinberger, D. R., Suter, J. C., & Goldberg, T. E. (2000). Continuous Performance Test and schizophrenia: A test of stimulus-response compatibility, working memory, response readiness, or none of the above? *American Journal of Psychiatry*, *157*, 772–780.
- Engle, R. W., Kane, M. J., & Tuholski S. W. (1999). Individual differences in working memory capacity and what they tell us about controlled attention, general fluid intelligence, and functions of the prefrontal cortex. In A. Miyake & P. Shah (Eds.), *Models of working memory* (pp. 102–134). Cambridge, England: Cambridge University Press.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. (1997). *Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-IV)*. Washington, DC: American Psychiatric Publishing.
- Gold, J. M., Carpenter, C., Randolph, C., Goldberg, T. E., & Weinberger, D. R. (1997). Auditory working memory and Wisconsin Card Sorting Test performance in schizophrenia. *Archives of General Psychiatry*, *54*, 159–165.
- Green, M. F., Marshall, B. D., Jr., Wirshing, W. C., Ames, D., Marder, S. R., McGurk, S., et al. (1997). Does risperidone improve verbal working memory in treatment-resistant schizophrenia? *American Journal of Psychiatry*, *154*, 799–804.
- Green, M. F., & Nuechterlein, K. H. (1998). Backward-masking performance in schizophrenia. In M. F. Lenzenweger & R. H. Dworkin (Eds.), *Origins and development of schizophrenia: Advances in experimental psychopathology* (pp. 329–348). Washington, DC: American Psychological Association.
- Hess, R., Schu, U., Muller, P., & Schuttler, R. (1992). Preattentive perception? Limited capacity channel system. In M. Spitzer, F. A. Uehlin, M. A. Schwartz, & C. Mundt (Eds.), *Phenomenology, language, and schizophrenia* (pp. 290–302). New York: Springer-Verlag.
- Irwin, D. E., & Andrews, R. V. (1996). Integration and accumulation of information across saccadic eye movements. In T. Inui & J. L. McClelland (Eds.), *Attention and Performance XVI. Information Integration in Perception and Communication* (pp. 125–155). Cambridge, MA: MIT Press.
- Javitt, D. C., Liederman, E., Cienfuegos, A., & Shelley A. M. (1999). Panmodal processing imprecision as a basis for dysfunction of transient memory storage systems in schizophrenia. *Schizophrenia Bulletin*, *25*, 763–775.
- Jiang, Y., Olson, I. R., & Chun, M. M. (2000). Organization of visual short-term memory. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *2*, 683–702.
- Keefe, R. S. (2000). Working memory dysfunction and its relevance to schizophrenia. In T. Sharma & P. D. Harvey (Eds.), *Cognition in schizophrenia: Impairments, importance, and treatment strategies* (pp. 16–50). Oxford, England: Oxford University Press.
- Keefe, R. S., Lees-Roitman, S. E., & Dupre, R. L. (1997). Performance of patients with schizophrenia on a pen-and-paper visuospatial working memory task with short delay. *Schizophrenia Research*, *26*, 9–14.
- Keefe, R. S., Silva, S. G., Perkins, D. O., & Lieberman, J. A. (1999). The effects of atypical antipsychotic drugs on neurocognitive impairment in schizophrenia: A review and meta-analysis. *Schizophrenia Bulletin*, *25*, 201–222.
- Kwon, J. S., O'Donnell, B. F., Wallenstein, G. V., Greene, R. W., Hirayasu, Y., Nestor, P. G., et al. (1999). Gamma frequency-range abnormalities to auditory stimulation in schizophrenia. *Archives of General Psychiatry*, *56*, 1001–1005.
- Lee, D., & Chun, M. M. (2001). What are the units of visual short-term memory, objects or spatial locations? *Perception and Psychophysics*, *63*, 253–257.
- Liang, K. Y., & Zeger, S. (1986). Longitudinal data analysis using generalized linear models. *Biometrika*, *73*, 13–22.
- Lisman, J. E., & Idiart, M. A. P. (1995). Storage of 7 ± 2 short-term memories in oscillatory subcycles. *Science*, *267*, 1512–1515.
- Luck, S. J., & Beach, N. J. (1998). Visual attention and the binding problem: A neurophysiological perspective. In R. D. Wright (Ed.), *Visual attention* (pp. 455–478). New York: Oxford University Press.
- Luck, S. J., & Vecera, S. P. (2002). Attention: From paradigms to mechanisms. In H. Pashler & S. Yantis (Eds.), *Stevens handbook of experimental psychology* (3rd ed., Vol. I, pp. 235–286). New York: Wiley.
- Luck, S. J., & Vogel, E. K. (1997). The capacity of visual working memory for features and conjunctions. *Nature*, *390*, 279–281.
- Luck, S. J., & Vogel, E. K. (1998). Response from Luck and Vogel (response to commentary by Nelson Cowan). *Trends in Cognitive Sciences*, *2*, 78–80.
- Manoach, D. S., Gollub, R. L., Benson, E. S., Searl, M. M., Goff, D. C., Halpern, E., et al. (2000). Schizophrenia subjects show aberrant fMRI activation of dorsolateral prefrontal cortex and basal ganglia during working memory performance. *Biological Psychiatry*, *48*, 99–109.
- Meltzer, H. Y., & McGurk, S. R. (1999). The effects of clozapine, risperidone, and olanzapine on cognitive function in schizophrenia. *Schizophrenia Bulletin*, *25*, 233–255.
- Mori, S., Tanaka, G., Ayaka, Y., Michitsuji, S., Niwa, H., Uemura, M., & Ohta, Y. (1996). Preattentive and focal attentional processes in schizophrenia: A visual search study. *Schizophrenia Research*, *22*, 69–76.
- Nuechterlein, K. H. (1991). Vigilance in schizophrenia and related disorders. In S. Steinhauer, J. H. Gruzeliier & J. Zubin (Eds.), *Handbook of schizophrenia: Neuropsychology, psychophysiology and information processing* (pp. 397–433). Amsterdam, the Netherlands: Elsevier Science.
- Park, S., & Holzman, P. S. (1992). Schizophrenics show spatial working memory deficits. *Archives of General Psychiatry*, *49*, 975–982.
- Park, S., Holzman, P. S., & Goldman-Rakic, P. S. (1995). Spatial working memory deficits in the relatives of schizophrenic patients. *Archives of General Psychiatry*, *52*, 821–828.
- Pashler, H. (1988). Familiarity and visual change detection. *Perception and Psychophysics*, *44*, 369–378.
- Perry, W., Heaton, R. K., Potterat, E., Roebuck, T., Minassian, A., & Braff, D. L. (2001). Working memory in schizophrenia: Transient “online”

- storage versus executive functioning. *Schizophrenia Bulletin*, 27, 157–176.
- Pfohl, B., Blum, N., & Zimmerman, M. (1997). *Structured Interview for DSM-IV Personality*. Washington, DC: American Psychiatric Press.
- Place, E. J. S., & Gilmore, G. C. (1980). Perceptual organization in schizophrenia. *Journal of Abnormal Psychology*, 89, 409–418.
- Raffone, A., & Wolters, G. (2001). A cortical mechanism for binding in visual working memory. *Journal of Cognitive Neuroscience*, 13, 766–785.
- Rizzo, L., Danion, J.-M., Van Der Linden, M., Grange, D., & Rohmer, J.-G. (1996). Impairment of memory for spatial context in schizophrenia. *Neuropsychology*, 10, 376–384.
- Salame, P., Danion, J.-M., Peretti, S., & Cuervo, C. (1998). The state of functioning of working memory in schizophrenia. *Schizophrenia Research*, 30, 11–29.
- Schmidt, B. K., Vogel, E. K., Woodman, G. F., & Luck, S. J. (2002). Voluntary and automatic attentional control of visual working memory. *Perception and Psychophysics*, 5, 754–763.
- Schweickert, R., & Boruff, B. (1986). Short-term memory capacity: Magic number or magic spell? *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 12, 419–425.
- Silverstein, S. M., Knight, R. A., Schwarzkopf, S. B., West, L. L., Osborn, L. M., & Kamin, D. (1996). Stimulus configuration and visual context effects in perceptual organization in schizophrenia. *Journal of Abnormal Psychology*, 105, 410–420.
- Simons, D. J., & Levin, D. T. (1997). Change blindness. *Trends in Cognitive Sciences*, 1, 261–267.
- Singer, W., Engel, A. K., Kreiter, A. K., Munk, M. H. J., Neuenschwander, S., & Roelfsema, P. R. (1997). Neuronal assemblies: Necessity, signature, and detectability. *Trends in Cognitive Sciences*, 1, 252–261.
- Stanislaw, H., & Todorow, N. (1999). Calculation of signal detection theory measures. *Behavioral Research Methods, Instruments and Computers*, 31, 137–149.
- Stevens, A. A., Goldman-Rakic, P. S., Gore, J. C., Fulbright, R. K., & Wexler, B. E. (1998). Cortical dysfunction in schizophrenia during auditory word and tone working memory demonstrated by functional magnetic resonance imaging. *Archives of General Psychiatry*, 55, 1097–1103.
- Stone, M., Gabrieli, J. D. E., Stebbins, G. T., & Sullivan, E. V. (1998). Working and strategic memory deficits in schizophrenia. *Neuropsychology*, 12, 278–288.
- Strauss, M. E. (2001). Demonstrating specific cognitive deficits: A psychometric perspective. *Journal of Abnormal Psychology*, 110, 6–14.
- Tallon-Baudry, C., Bertrand, O., & Fischer, C. (2001). Oscillatory synchrony between human extrastriate areas during visual short-term memory maintenance. *Journal of Neuroscience*, 21, RC177, 1–5.
- Tek, C., Gold, J. M., Blaxton, T., Wilk, C., McMahon, R. P., & Buchanan, R. W. (2002). Visual perceptual and working memory impairments in schizophrenia. *Archives of General Psychiatry*, 59, 146–153.
- Treisman, A. (1996). The binding problem. *Current Opinion in Neurobiology*, 6, 171–178.
- Treisman, A. M., & Gelade, G. (1980). A feature-integration theory of attention. *Cognitive Psychology*, 12, 97–136.
- Vogel, E. K., Woodman, G. F., & Luck, S. J. (2001). Storage of features, conjunctions, and objects in visual working memory. *Journal of Experimental Psychology: Human Perception and Performance*, 27, 92–114.
- Walker, P., & Cuthbert, L. (1998). Remembering visual feature conjunctions: Visual memory for shape–colour associations is object-based. *Visual Cognition*, 5, 409–455.
- Weiss, K. M., Vrtunski, P. B., & Simpson, D. M. (1988). Information overload disrupts digit recall performance in schizophrenics. *Schizophrenia Research*, 4, 299–303.
- Wheeler, M., & Treisman, A. M. (2002). Binding in short-term visual memory. *Journal of Experimental Psychology: General*, 131, 48–64.
- Wilkinson, G. S. (1993). *Wide Range Achievement Test-3: Administration manual*. Wilmington, DE: Wide Range.
- Xu, Y. (2002). Limitations of object-based feature encoding in visual short-term memory. *Journal of Experimental Psychology: Human Perception & Performance*, 28, 458–468.

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