

Exploring the short term visual store in schizophrenia using the attentional blink

Jonathan K. Wynn^{a,b,*}, Bruno Breitmeyer^c, Keith H. Nuechterlein^a, Michael F. Green^{a,b}

^a Department of Psychiatry and Biobehavioral Sciences, Geffen School of Medicine at UCLA, United States

^b Veterans Affairs Greater Los Angeles Healthcare System, 11301 Wilshire Blvd.,
(MIRECC 210A) Building 210, Los Angeles, CA 90073, United States

^c Department of Psychology, University of Houston, United States

Received 28 December 2005; received in revised form 11 April 2006; accepted 8 June 2006

Abstract

Schizophrenia patients exhibit numerous deficits on visual processing tasks, ranging from very early stages of visual processing (e.g., backward masking) to the later working memory stages (e.g., delayed match-to-sample, N-back). However, little is known about deficits in an intermediate stage of visual information processing, namely short term visual memory (STVM). The attentional blink (AB) paradigm is considered to be a valid way to assess the STVM, and recent studies have reported AB deficits in schizophrenia. However, it is not clear whether the reported AB deficit in schizophrenia patients is due to their increased susceptibility to backward masking or increased vulnerability in the STVM. In this study we first found poorer performance in the AB task in 37 schizophrenia patients compared to 26 normal controls. To examine the effects of increasing and decreasing mask strength on AB performance in patients and controls, we next systematically varied the masking effect by varying the length of the distracters immediately following the targets. The manipulation had relatively little effect on the patient – control differences and patients continued to show an enhanced AB effect across conditions. The findings suggest that the enhanced AB effect in schizophrenia reflects an abnormality in their short term visual memory, as opposed to their enhanced susceptibility to visual masking.

© 2006 Elsevier Ltd. All rights reserved.

Keywords: Schizophrenia; Visual processing; Attentional blink; Short term visual memory

1. Introduction

Schizophrenia patients exhibit several neurocognitive impairments that range from deficits in low-level information processing to high-level cognition. For example, patients show consistent deficits on backward masking tasks which reflect the earliest stages of visual processing (e.g., Butler et al., 2002; Cadenhead et al., 1998; Green et al., 1999; Green et al., 1994). Patients also show deficits on working memory tasks which reflect later cognitive

stages of visual processing, such as the spatial delayed match-to-sample task (e.g., Lencz et al., 2005; Park and Holzman, 1992) and the N-back task (e.g., Krieger et al., 2005; Perlstein et al., 2001). Despite an extensive literature regarding deficits in schizophrenia in these relatively early and late stages of visual information processing, little is known about a separate intermediate stage, namely short-term visual memory (STVM).

Short-term visual memory is a capacity-limited stage of processing that follows initial visual input processing (Bachmann and Hommuk, 2005; Knight et al., 1985; Phillips, 1974; Rabinowicz et al., 1996). This stage provides a brief working period to consolidate visual information into a form that can be passed on to later stages of processing, such as retention and recall (Potter, 1975, 1976). One

* Corresponding author. Address: Veterans Affairs Greater Los Angeles Healthcare System, 11301 Wilshire Blvd., Building 210, Los Angeles, CA 90073, United States. Tel.: +1 310 478 3711x44957; fax: +1 310 268 4056.
E-mail address: jkwynn@ucla.edu (J.K. Wynn).

accepted method to precisely assess the STVM is the attentional blink paradigm (AB).

During AB, two successively presented targets (T1 and T2) are presented in a rapid serial visual presentation of stimuli, including non-targets (referred to as distracters). The detection of T1 interferes with successful detection of T2 when T2 is delayed by a period of 200–500 ms (Chun and Potter, 1995; Raymond et al., 1992), which is called a “blink” in attention. A well-accepted theory for this phenomenon involves a two-stage model (Chun and Potter, 1995; Potter et al., 2002). This model proposes that in stage 1, detection of targets rapidly takes place in an attentionally labile period. In stage 2, targets are identified in a serial stage of processing that consolidates the targets into working memory (Vogel and Luck, 2002; Vogel et al., 1998) in an attentionally stable period. However, if T2 is presented while T1 is in stage 2, T2 cannot enter the second stage until T1 is completely processed. During this delay (approximately 200–500 ms) the representation of T2 decays during its stage 1 processing, resulting in a momentary “blink” of T2 detection.

It has been established that, for AB to occur, each target must have a trailing distracter stimulus, similar to a visual mask (Giesbrecht and Di Lollo, 1998; Raymond et al., 1992; Seiffert and Di Lollo, 1997; Vogel and Luck, 2002). The distracter places additional processing demands on the target. If this distracter is eliminated, processing of the target is completed rapidly and with little or no interference (Chun and Potter, 1995; Raymond et al., 1992). However, AB is not considered to be merely a variant of backward masking (Bachmann and Hommuk, 2005; Raymond et al., 1992). Rather, backward masking functions at early stages of visual processing (20–100 ms), typically interrupting processing of the first stimulus (Breitmeyer, 1984; Breitmeyer and Ganz, 1976). AB, while influenced by backward masking, operates at a later stage of visual processing (200–500 ms) in which attentional demands are more dominant. Theoretically, increased susceptibility to masking could create an abnormal AB in samples that have increased susceptibility to the presence of a visual mask, such as schizophrenia (e.g., Butler et al., 2002; Cadenhead et al., 1998; Green et al., 1999; Green et al., 1994) by placing extra demands on attentional processing not seen in normal populations.

Only two published studies to date have examined AB in schizophrenia patients (Cheung et al., 2002; Li et al., 2002). In these studies, schizophrenia patients exhibited a larger AB effect (i.e., lower correct T2 identification at each lag) and a protracted AB effect (i.e., lower correct T2 identification at later lags) compared to normal controls. These results imply that schizophrenia patients are less efficient at processing information presented in a rapid stream. However, the specific interpretation of the deficit is unclear as there are two possible explanations. The problem could be an abnormality in the STVM, or a reflection of the increased susceptibility of patients to visual masking (Cheung et al., 2002).

The two studies of AB in schizophrenia are interesting, but their findings are difficult to generalize to the broader schizophrenia population. Both studies were: (1) conducted on a Chinese population (Taiwan and Hong Kong); (2) conducted with inpatients or day patients; (3) conducted with patients mainly on first-generation antipsychotic medications or with no specific medication information available; and (4) included patients who were relatively young (mean ages of 32 and 34). We tested the AB effect in a distinctly different type of sample: ethnically diverse U.S. schizophrenia outpatients, who were older and mainly on second-generation antipsychotic medications.

In this paper, we examined the nature of the AB deficit in schizophrenia patients with two AB conditions. First, we tested whether we could replicate previous reports and detect an enhanced AB effect in schizophrenia patients. We expected to see an AB effect in the time range of 200–500 ms in both groups, with the patients either showing an enhanced AB, a protracted AB, or both effects within this time range. Second, the main goal of this study is to clarify the nature of the enhanced AB effect by manipulating the strength of backward masking (e.g., by decreasing or increasing the length of the distracter following the two targets). We hypothesized that if the AB performance in schizophrenia patients is attributable to increased masking susceptibility, then increasing the masking strength of the distracters will result in greater patient control differences in the AB performance functions.

2. Methods

2.1. Participants

Participants included 37 schizophrenia patients (6 female) and 26 normal controls (8 female). Participants were recruited from a larger study “Early Visual Processing in Schizophrenia” (PI: M.F. Green). Patients were recruited from outpatient clinics at the VA Greater Los Angeles Healthcare System and through presentations in the community. Non-patient control participants were recruited through approved flyers posted in the local community and through newspaper advertisements. All participants gave written informed consent after all procedures were fully explained, in compliance with procedures approved by the UCLA and VA Institutional Review Boards.

Patients met criteria for schizophrenia based on the Structured Clinical Interview for DSM-IV (SCID; First et al., 1996b). All patients were receiving antipsychotic medication. Psychiatric symptoms in the patient group were assessed with the 24-item Brief Psychiatric Rating Scale (BPRS; Ventura et al., 1993). Interviewers were trained to a minimum intraclass correlation of 0.80 on ratings in the VISN 22 Mental Illness Research Education and Clinical Center (MIRECC) Treatment Unit. Table 1 provides the means and standard deviations of the items (range 1–7) that comprise the positive symptom (unusual thought content, hallucinations, and conceptual disorganization)

Table 1
Group demographics and symptom ratings for schizophrenia patients

	Mean	SD
<i>Normal controls (18 male/8 female)</i>		
Age	40.4	8.5
Age range	26.7–52.7	
Education	13.4	1.0
<i>Schizophrenia patients (31 male/6 female)</i>		
Age*	47.7	10.2
Age range	25.8–61.5	
Education	12.6	1.9
BPRS total	42.6	11.5
BPRS subscales		
Thinking disturbance	2.3	1.2
Withdrawal/retardation	1.8	0.7

* Schizophrenia patients were significantly older than normal controls ($t(61) = 3.01, p < .01$).

and negative symptom (emotional withdrawal, blunted affect, and motor retardation) factors of the BPRS (Overall et al., 1967), as well as the total BPRS score.

Control participants were screened with the SCID and SCID II (First et al., 1996a) and were excluded if they met criteria for any psychotic disorder, bipolar mood disorder, recurrent depression, substance dependence, or paranoid, schizoid, borderline, avoidant or schizotypal personality disorders. Control participants were also excluded if there was a history of a psychotic disorder among first-degree relatives.

Additionally, all patient and control participants were excluded for active substance dependence in the past six months, substance abuse in the past month, identifiable neurological disorder, seizure disorder, or mental retardation. Patients were included if they were between the ages of 18–62 years old and controls were included if they were between 25 and 55 years old. Diagnostic interviewers were trained to a minimum kappa of 0.75 for rating psychotic mood symptoms. Approximately 54% of patients and 19% of controls had a history of substance abuse.

2.2. Procedures

Subjects were seated in a comfortable chair approximately 1 m from a computer monitor set at 150 Hz. All stimuli were generated and presented through E-Prime (Pittsburgh, PA). Two sets of stimuli served as targets and distracters. Targets consisted of the capital letters A, C, E, J, K, R, T and Y presented in Times New Roman font. Distracters consisted of the numerals 2, 3, 4, 5, 6, 7, 8 and 9.

In the first experimental condition (standard), all stimuli were presented for 27 ms with a stimulus onset asynchrony of 94 ms. Five lags between the first (T1) and second (T2) targets were utilized: either a lag of 1, 2, 3, 4 or 5 after T1 (i.e., a lag of 1 means T2 is displayed immediately after T1, a lag of 2 means T2 is displayed after T1 with one distracter in between, etc.; see Fig. 1). There were 12 trials for each lag, resulting in a total of 60 trials for the standard condition. At least two distracters were always presented

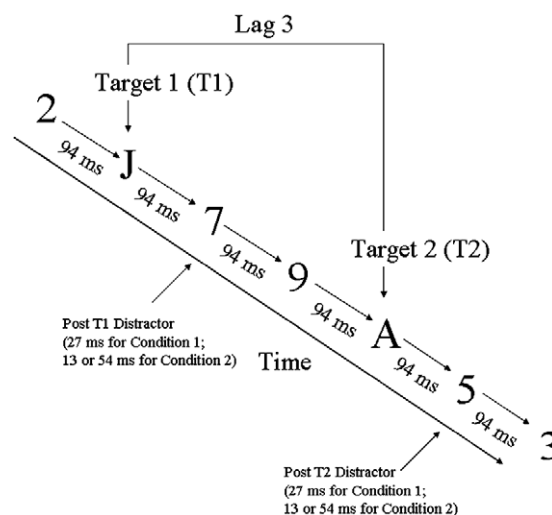


Fig. 1. Sequence and timing of events for the attentional blink. In the standard condition all stimuli are 27 ms. In the variable distracter length condition, post-T1 and post-T2 distracter lengths are either 13 ms or 54 ms.

before T1 and at least two distracters after T2 with a total of 11 stimuli presented in each visual stream. Each 11-stimulus visual stream constituted a single trial. All stimuli were presented in a fixed, pseudo-random order. Participants were instructed to name the first and second targets in the order they were presented and the experimenter logged their responses into the computer. If they were not sure, they were instructed to guess. They were not given a time limit to make their choices. Participants were given a sheet containing the eight possible target stimuli to which they could refer when giving their responses. The next trial commenced after the experimenter logged the results.

The second experimental condition (distracter length manipulation) was administered immediately after the first. It consisted of the same set of stimuli as the first session and the same parameters, with the following exceptions: the length of the distracters immediately following the targets was varied and a different set of lags were used to assess the AB. The length of the post-target distracter was systematically varied to be either half the length (13 ms) or twice the length (54 ms) of the target. Four possible combinations of post-target distracter length for T1 and T2 resulted from this manipulation: 13 ms/13 ms, 13 ms/54 ms, 54 ms/13 ms or 54 ms/54 ms. Four lags were utilized: 2, 3, 5 or 7 (a lag of 1 could not be used as the duration of T2 would be different from that of T1). Twelve trials were presented for each lag and each combination of post-target distracter length, resulting in a total of 192 trials. There were 12 stimuli (targets and distracters) presented in each visual stream. Each 12-stimulus visual stream constituted a single trial. As before, participants were asked to name the targets in the order that they were presented and the experimenter logged their responses into the computer.

The dependent variable was the conditional probability at each lag of accurately detecting the second target given accurate detection of the first target ($Pr T2/T1$). Data were

analyzed with a repeated measures ANOVA separately for each condition. Age was significantly different between groups (see Table 1). Because age did not meet the homogeneity of slopes assumption between groups, we did not use age in an ANCOVA. Instead, to determine if age affected the results, we conducted additional analyses in which we removed patients older than 55 years ($n = 11$), which yielded groups of comparable age. As a check on the assumption of homogeneity of variance in the repeated measures ANOVA, we reran all analyses with a general linear mixed effects model with a fully unstructured covariance matrix using SAS PROC MIXED, using the same analysis structure as the repeated measures ANOVA, and the results were not meaningfully different from the ANOVA. All analyses were conducted with an *a priori* significance level of .05. Greenhouse-Geisser epsilon (ϵ) correction factors were used to adjust probability levels for repeated measures ANOVAs for within-subject factors with more than two levels. We report the uncorrected degrees of freedom, the corrected significance level, and ϵ value.

3. Results

3.1. Demographics

As can be seen in Table 1, patients were significantly older ($t(61) = 3.01, p < .01$) and tended to have lower education ($t(61) = 1.92, p < .06$) than normal controls. The gender and ethnicity distributions between groups did not significantly differ. As can be seen by the symptom ratings in Table 1, patients had relatively low levels of positive and negative symptoms. Twenty-six patients were on second-generation antipsychotic medications, 7 on first-generation, 1 on mixed, 2 were not taking medication at time of testing and medical information could not be confirmed for 1

Table 2
Group target accuracy by lag for the standard attentional blink condition

	Lag	Patients		Controls	
		Mean	SD	Mean	SD
Target 1	1	4.3	2.2	7.1	2.1
	2	7.2	2.5	8.7	2.4
	3	7.4	2.7	8.8	2.4
	4	7.5	2.4	9.0	2.7
	5	8.0	2.3	9.4	2.5
Target 2	1	3.2	2.1	6.5	2.4
	2	3.3	2.2	5.9	3.4
	3	3.5	2.8	5.5	3.7
	4	3.7	2.7	6.0	3.4
	5	4.5	2.9	7.0	3.3

Means are number correct out of 12.

patient. Limiting the analyses to the 26 patients on confirmed second generation antipsychotic medication did not affect any of the results; therefore, medication status was not considered further in the analyses.

3.2. Attentional blink: standard distracter length

Table 2 presents the mean and standard deviation of the number of correctly identified targets at each lag. As can be seen, the patients correctly identify fewer targets at each lag compared to the controls. It can also be seen that both groups identify fewer T1 targets at lag 1, possibly reflecting a backward masking effect.

The AB effect was analyzed using conditional probability of T2 (Pr T2/T1) with a repeated measures ANOVA, with lag (1, 2, 3, 4 or 5) serving as the repeated factor and group (patients vs. controls) as the between-subjects factor. The analysis revealed a significant group effect, $F(1, 61) = 10.41, p < .01$, and a significant effect of lag, $F(4, 244) = 23.57, p < .001, \epsilon = .90$. There was no interaction between group and lag. As can be seen in Fig. 2,

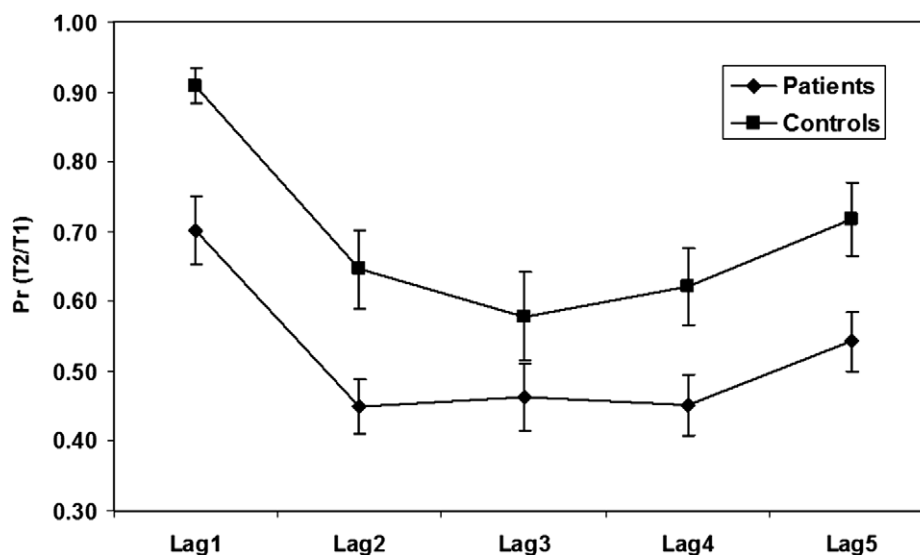


Fig. 2. The standard attentional blink (AB) effect in normal controls and schizophrenia patients. Schizophrenia patients correctly identified fewer T2 targets across all lags compared to controls.

controls performed better across all lags compared to patients, with both groups showing the standard AB effect (i.e., poorer performance at intermediate lags compared to the earliest and latest lags). Rerunning the analyses without the oldest patients did not affect the results.

We examined the correlation between education and AB performance separately for each group. As there were 5 possible correlations, we used an alpha level of .01 to correct for experiment-wise Type 1 error. Education did not significantly correlate with AB performance at any lag in either group.

3.3. Attentional blink: varied distracter length

The variable distracter length condition was analyzed with a 2 (post-T1 distracter length: 13 or 54 ms) × 2 (post-T2 distracter length: 13 or 54 ms) × 4 (lag: 2, 3, 5 or 7) × 2 (group: patients vs. controls) repeated measures ANOVA. Analyses revealed a significant group effect, $F(1, 61) = 9.34, p < .01$, a significant lag effect, $F(3, 183) = 12.84, p < .001, \epsilon = .82$, and a significant effect of post-T2 distracter length, $F(1, 61) = 13.57, p < .01$. There was also a significant three-way interaction (lag, post-T1 distracter length and post-T2 distracter length, $F(3, 183) = 12.47, p < .001, \epsilon = .94$). Rerunning the analyses without the oldest patients did not affect the results.

As in the standard distracter length condition, normal controls performed better across all lags as compared to patients (see Fig. 3). Furthermore, all subjects showed the

standard AB effect, as revealed by the significant lag effect. The significant effect of post-T2 distracter length revealed that significantly fewer second targets were accurately identified in both groups (mean probability = .664) with the long distracter, compared to the short distracter (mean probability = .706). This finding demonstrates that, by increasing the masking strength of the distracter, the AB effect was increased comparably across groups. Hence, there was no significant interaction between T2 duration and group, $F(1, 61) = 1.06, p < .31$.

The significant three-way interaction was due to differential effectiveness of the distracter length at different lags and was examined with a series of paired *t*-tests. Notably, when the post-T1 distracter at lag 2 was short and the post-T2 distracter was long, the AB effect was stronger than that with a short post-T2 distracter, $t(62) = 5.22, p < .01$. When the long distracter appeared after both T1 and T2, at lags 3 and 5, the AB effect was stronger than that with a short post-T2 distracter, $t(62) = 2.28$ and 4.74 respectively, $p < .05$. However, this finding did not interact with group.

We examined the correlation between education and AB performance separately for each group. As there were 16 possible correlations, we used an alpha level of .01 to partially correct for experiment-wise Type 1 error. Only one correlation was significant: Education correlated with AB performance in the schizophrenia patients at lag 2 when both post-target distracters were short, $r = .45, p < .01$. There were no significant correlations in the controls.

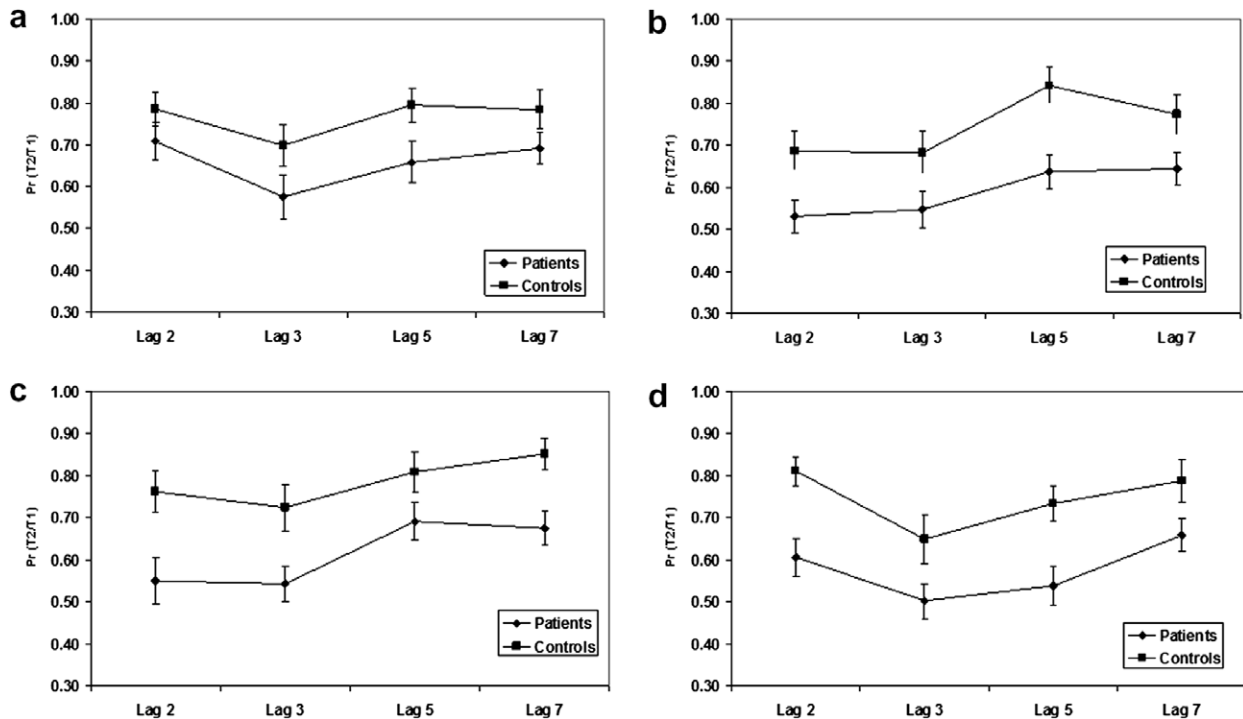


Fig. 3. Effects of masking strength of distracters after the targets in the attentional blink. In all four conditions, schizophrenia patients show poorer AB performance compared to normal controls. (a) Post T1 and T2 distracter lengths 13 ms; (b) post T1 distracter length 13 ms, post T2 distracter length 54 ms; (c) post T1 distracter length 54 ms, Post T2 distracter length 13 ms; and (d) post T1 and T2 distracter lengths 54 ms.

4. Discussion

In the first experimental condition, schizophrenia patients exhibited poorer performance on the AB task compared to normal controls, consistent with previous studies of AB in schizophrenia (Cheung et al., 2002; Li et al., 2002). However, as the groups significantly differed at the first lag, the enhanced AB effect was difficult to interpret. A potential masking effect might explain why patients show an enhanced AB across all lags, and particularly at lag 1, in the first condition (see Fig. 2).

As reported in normal populations, at short SOAs between targets (i.e., lag 1), backward masking effects are more dominant than AB effects (Bachmann and Hommuk, 2005; Potter et al., 2002), possibly explaining the lower than expected performance at lag 1 in the patients. Cheung et al. (2002) also showed patient–control differences at lag 1. They accounted for baseline differences by factoring in performance from an additional single-target condition, and still found an enhanced AB in schizophrenia patients.

To determine whether the performance of the schizophrenia patients on the AB task was due to increased susceptibility of schizophrenia patients to backward masking, we varied the strength of the masking effect by modifying the length of the distracter immediately following the targets. The results again revealed an overall significant group effect, and the duration of the distracters did not interact with group, suggesting that increasing the masking strength did not increase the differences between groups. Hence we cannot attribute AB performance in patients to enhanced susceptibility to backward masking. Instead, the AB deficits in patients appear to be due to problems in the STVM. However, it should be noted that this study was not able to fully dissociate masking effects from the AB, and future studies with schizophrenia patients may better address this issue (e.g., Potter et al., 2002). For example, a recent study demonstrated working memory consolidation deficits in schizophrenia in the absence of masking (Fuller et al., 2005).

The conclusion that AB performance is separable from visual masking is consistent with recent findings from functional neuroimaging that suggest that the AB paradigm involves somewhat different neural regions than those in backward masking tasks or working memory tasks. Functional magnetic resonance imaging (fMRI) findings for AB have implicated involvement of frontal and parietal areas during conscious perception of the targets, namely superior frontal gyrus, inferior frontal gyrus, anterior cingulate cortex and inferior parietal lobe (Kranzioch et al., 2005). In an fMRI study of visual backward masking, we found that ventral and dorsal lateral occipital complex (LO), located early in the visual processing stream, is the likely source of the masking effect (Green et al., 2005). Furthermore, in both the *n*-back (Perlstein et al., 2001) and spatial delayed match-to-sample task (Habeck et al., 2005), fMRI findings have implicated the prefrontal cortex as the area responsible for maintenance

in working memory of visual information. Therefore, it appears that AB, indexing the separate stage of STVM, has partially distinct neural substrates from visual masking and visual working memory.

The current study adds to a relatively small literature on STVM deficits in schizophrenia (e.g., Knight et al., 1985; Rabinowicz et al., 1996) and is complementary to the two previous reports of AB deficits in schizophrenia, though we did not replicate the finding of a protracted AB at longer lags in schizophrenia patients compared to controls. The failure to find a protracted AB in patients may be due to the different stimulus parameters used in the current study (e.g., the relatively shorter stimulus duration) or differences in patient sample. Our results extend previous findings in finding AB performance differences in an ethnically diverse US population of stable schizophrenia outpatients who were mainly taking second generation antipsychotic medications.

This study had a few limitations. First, the schizophrenia patients were significantly older than the normal controls. Age can be a factor in AB performance as at least one study has reported an increased AB effect in an older (e.g., >60 years old) population (Maciokas and Crognale, 2003). Another limitation is that the patients in our study were chronic, stabilized outpatients on second generation antipsychotic medications. It is not known whether an AB deficit would also be seen in early-onset or never-medicated schizophrenia patients. Educational level is a possible confound; however, it is not likely that educational level can entirely account for the performance differences as it was only seen in one specific correlation in the patient group and was not seen in the normal controls. Finally, a high proportion of patients had a history of substance abuse (though not in the prior month before participation). It is not known whether substance abuse affects presentation of the AB, though this should be considered in future studies in normal controls as well as schizophrenia patients.

In conclusion, this study examined in schizophrenia short term visual memory, a stage that falls between the earliest sensory perception stage and the later cognitive and working memory stage. We demonstrated poor performance on an AB task in schizophrenia patients that seems to be reflecting a deficit in their STVM and is not due to their increased susceptibility to backward masking.

Acknowledgements

This research was supported by grants MH-43292 and MH-65707 to Michael F. Green, Ph.D. and by the Department of Veterans Affairs, Veterans Integrated Services Network 22 Mental Illness Research, Education, and Clinical Center (MIRECC). Jonathan K. Wynn was supported by a Ruth L. Kirschstein NIMH NRSA Training Grant MH14584 (PI - Keith H. Nuechterlein, Ph.D.) during preparation of this manuscript. The authors wish to thank Patrick Carlyle for technical assistance, and Poorang Nori and Kelly Tillery for help in data collection. Portions of this

paper were presented at the 59th Annual Meeting of the Society of Biological Psychiatry, New York, NY, April, 2004.

References

- Bachmann T, Hommuk K. How backward masking becomes attentional blink. *Psychological Science* 2005;16:740–2.
- Breitmeyer BG. *Visual masking: An integrative approach*. New York: Oxford University Press; 1984.
- Breitmeyer BG, Ganz L. Implications of sustained and transient channels for theories of visual pattern masking, saccadic suppression, and information processing. *Psychological Review* 1976;83:1–36.
- Butler PD, DeSanti LA, Maddox J, Harkavy-Friedman JM, Amador XF, Goetz RR, Javitt DC, Gorman JM. Visual backward-masking deficits in schizophrenia: Relationship to visual pathway function and symptomatology. *Schizophrenia Research* 2002;59:199–209.
- Cadenhead KS, Serper Y, Braff DL. Transient versus sustained visual channels in the visual backward masking deficits of schizophrenia patients. *Biological Psychiatry* 1998;43:132–8.
- Cheung V, Chen EYH, Chen RYL, 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. *Schizophrenia Bulletin* 2002;28:443–58.
- Chun MM, Potter MC. A two-stage model for multiple target detection in rapid serial visual presentation. *Journal of Experimental Psychology: Human Perception & Performance* 1995;21:109–27.
- First MB, Gibbons M, Spitzer RL, Williams JBW. *Structured Clinical Interview for DSM-IV Axis II Personality Disorders*. New York: Biometrics Research Department; 1996a.
- First MB, Gibbons M, Spitzer RL, Williams JBW. *Users guide for the Structured Clinical Interview for DSM-IV Axis I Disorders-Research Version (SCID-I, Version 2.0, February 1996 Final Version)*. New York: Biometrics Research Department; 1996b.
- Fuller RL, Luck SJ, McMahon RP, Gold JM. Working memory consolidation is abnormally slow in schizophrenia. *Journal of Abnormal Psychology* 2005;114:279–90.
- Giesbrecht B, Di Lollo V. Beyond the attentional blink: Visual masking by object substitution. *Journal of Experimental Psychology: Human Perception & Performance* 1998;24:1454–66.
- Green MF, Nuechterlein KH, Mintz J. Backward masking in schizophrenia and mania: II. Specifying the visual channels. *Archives of General Psychiatry* 1994;51:939–44.
- Green MF, Nuechterlein KH, Breitmeyer BG, Mintz J. Backward masking in unmedicated schizophrenic patients in psychotic remission: Possible reflection of aberrant cortical oscillation. *American Journal of Psychiatry* 1999;156:1367–73.
- Green MF, Glahn D, Engel SA, Nuechterlein KH, Sabb F, Strojwas M, Cohen MS. Regional brain activity associated with visual backward masking. *J Cogn Neurosci* 2005;17:13–23.
- Habeck C, Rakinin BC, Moeller J, Scarmeas N, Zarahn E, Brown T, Stern Y. An event-related fMRI study of the neural networks underlying the encoding, maintenance, and retrieval phase in a delayed-match-to-sample task. *Cognitive Brain Research* 2005;23:207–20.
- Knight RA, Elliot DS, Freedman EG. Short-term visual memory in schizophrenics. *Journal of Abnormal Psychology* 1985;94:427–42.
- Krancioch C, Debener S, Schwarzbach J, Goebel R, Engel AK. Neural correlates of conscious perception in the attentional blink. *NeuroImage* 2005;24:704–14.
- Krieger S, Lis S, Cetin T, Gallhofer B, Meyer-Lindenberg A. Executive function and cognitive subprocesses in first-episode, drug-naive schizophrenia: an analysis of N-back performance. *Am J Psychiatry* 2005;162:1206–8.
- Lenz T, Bilder RM, Turkel E, Goldman RS, Robinson D, Kane JM, Lieberman JA. Impairments in perceptual competency and maintenance on a visual delayed match-to-sample test in first-episode schizophrenia. *Archives of General Psychiatry* 2005;60:238–43.
- Li CR, Lin W, Yang Y, Huang C, Chen T, Chen Y. Impairment of temporal attention in patients with schizophrenia. *Neuroreport* 2002;13:1427–30.
- Maciokas JB, Crognale MA. Cognitive and attentional changes with age: Evidence from attentional blink effects. *Experimental Aging Research* 2003;29:137–53.
- Overall JE, Hollister LE, Pichot P. Major psychiatric disorders: A four-dimensional model. *Archives of General Psychiatry* 1967;16:146–51.
- Park S, Holzman PS. Schizophrenics show spatial working memory deficits. *Archives of General Psychiatry* 1992;49:975–82.
- Perlstein WM, Carter CS, Noll DC, Cohen JD. Relation of prefrontal cortex dysfunction to working memory and symptoms in schizophrenia. *Am J Psychiatry* 2001;158:1105–13.
- Phillips WA. On the distinction between sensory storage and short-term visual memory. *Perception and Psychophysics* 1974;16:283–90.
- Potter MC. Meaning in visual search. *Science* 1975;187:965–6.
- Potter MC. Short-term conceptual memory for pictures. *Journal of Experimental Psychology: Human Learning and Memory* 1976;2:509–22.
- Potter MC, Staub A, O'Connor DH. The time course of competition for attention: Attention is initially labile. *Journal of Experimental Psychology: Human Perception & Performance* 2002;28:1149–62.
- Rabinowicz EF, Opler LA, Owen DR, Knight RA. Dot enumeration perceptual organization task (DEPOT): Evidence for a short-term visual memory deficit in schizophrenia. *Journal of Abnormal Psychology* 1996;105:336–48.
- Raymond JE, Shapiro KL, Arnell KM. Temporary suppression of visual processing in an RSVP task: An attentional blink? *Journal of Experimental Psychology: Human Perception & Performance* 1992;18:849–60.
- Seiffert AE, Di Lollo V. Low-level masking in the attentional blink. *Journal of Experimental Psychology: Human Perception & Performance* 1997;23:1061–73.
- Ventura J, Lukoff D, Nuechterlein KH, Liberman RL, Green MF, Shaner A. *Brief Psychiatric Rating Scale (BPRS)*. *International Journal of Methods in Psychiatric Research* 1993;3:227–43.
- Vogel EK, Luck SJ. Delayed working memory consolidation during the attentional blink. *Psychonomic Bulletin and Review* 2002;9:739–43.
- Vogel EK, Luck SJ, Shapiro KL. Electrophysiological evidence for a postperceptual locus of suppression during the attentional blink. *Journal of Experimental Psychology: Human Perception & Performance* 1998;24:1656–74.