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Pupillary responses and attentional allocation problems on the backward masking task in schizophrenia

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Abstract

Early visual information processing impairment has consistently been found on the backward masking task in patients with schizophrenia, but the nature of this impairment remains unclear. Pupillometry was used to measure attentional allocation during visual backward masking task performance in patients with schizophrenia ($n=16$) and nonpsychiatric controls ($n=16$). The extent of pupil dilation recorded during a cognitive task reflects the processing load placed on the nervous system by the task. Schizophrenia patients detected significantly fewer targets than controls only when the stimulus onset asynchrony (SOA) between targets and masks reached 317 ms. For both groups, peak pupil dilation responses were also significantly larger in the 317 ms SOA condition relative to a no-mask condition, suggesting that the processing load of the 317 SOA masking condition was greater than the no-mask condition. In addition, a principal components analysis of pupillary response waveforms identified time-related factors that appeared to differentially index attentional allocation to targets vs. masks. Patients with schizophrenia showed less dilation than controls on a middle factor that appeared to index attentional allocation to targets, but patients showed greater dilation than controls on a late factor that appeared to index attentional allocation to masks. That is, controls attended more to targets than to masks, but patients attended more to masks than to targets. These findings suggest that masking impairments at SOA intervals greater than 100–200 ms may be due to abnormalities in attentional allocation mechanisms.

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1. Introduction

Information-processing models provide a strong theoretical basis for stimulating research on cognitive impairment in schizophrenia. These models also offer established laboratory tasks that are highly sensitive to cognitive impairment in schiz-

ophrenia (Nuechterlein and Dawson, 1984). One well-studied task, the visual backward masking task, taps the time course of information passing through the sensory register. A target stimulus (e.g. letter) is visually presented and rapidly followed by a mask (e.g. patterned or random lines) at varying stimulus onset asynchronies (SOAs) between the target and the mask (e.g. 10–700 ms). When the mask follows the target, it is called backward masking. Subjects typically must identify the target or its location. The masking stimulus

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is thought to interfere with the accurate identification or localization of the target, because it takes time for information to be transferred from the sensory register to short-term memory. If the mask is presented before this information transfer is completed, the target is less likely to be correctly identified.

When the mask is of similar energy (e.g. duration and luminance) to the target, studies have consistently reported impaired target identification at SOAs of approximately 120–350 ms in medicated and unmedicated, acute and remitted schizophrenia patients in more than 20 studies (Balogh and Merritt, 1987; Braff and Saccuzzo, 1981; Green et al., 1994; McClure, 2001; Miller et al., 1979; Rund et al., 1993). This masking deficit has also been found in schizophrenia-spectrum subjects with schizotypal or psychosis prone traits (Steronko and Woods, 1978; Balogh and Merritt, 1985; Saccuzzo and Schubert, 1981) and in unaffected siblings of schizophrenia patients (Green et al., 1997). Masking task deficits, therefore, may reflect vulnerability or predisposition for schizophrenia, rather than the symptoms of the illness itself.

Despite consistent findings of masking task deficits in patients with schizophrenia, the nature of this impairment remains unclear. In particular, attention mechanisms have received surprisingly little study as a possible source of masking deficits in schizophrenia. When the masking interval is greater than approximately 100 ms, identification of stimulus meaning of distinct target and mask stimuli is thought to occur in short-term visual memory (Phillips, 1974). At this point, limited controlled processes must be shifted and shared between targets and masks to identify both stimuli, so attentional allocation to one or the other stimulus is required (Loftus et al., 1988; Michaels and Turvey, 1979; Phillips, 1974). Michaels and Turvey (1979) referred to this as 'replacement', whereby target information is replaced by mask information as the main focus of attention. This is the first stage in early visual processing when masking deficits are attributed to attentional mechanisms or the allocation of processing resources, rather than to mechanisms influencing the quality of the stimulus percept, itself. Target identification suffers after approximately 100 ms intervals, not because

of the quality of the target icon is degraded (e.g. by integration or interruption), but because target and mask icons compete for common stimulus identification algorithms. Backward masking task impairments in schizophrenia, which are most commonly observed at masking intervals greater than 100 ms, may be due to abnormalities in these attentional allocation mechanisms in short-term visual memory (Knight, 1992, 1993). Wasteful allocation of processing resources to mask processing would leave fewer resources available for accurate target identification (Braff et al., 1991; Nuechterlein and Dawson, 1984).

Pupillometry methods can be used to examine attentional allocation during backward masking tasks. For many decades, the pupillary response has been used as a reliable and sensitive psychophysiological index of 'mental effort' or the amount of processing resources allocated to a cognitive task (Beatty, 1982; Beatty and Lucero-Wagoner, 2000; Kahneman, 1973). Increased pupil dilation during cognitive task performance reflects increased allocation of processing resources to the task. In numerous studies (for a review, see Beatty, 1982; Beatty and Lucero-Wagoner, 2000), increased task processing load (e.g. more difficult perceptual discriminations, greater sentence syntax complexity, more arithmetic multiplicands) reliably evoked greater pupillary dilation responses, regardless of the cognitive domain tapped by different tasks.

In our previous research (Verney et al., 2001; Verney, 2001; Verney et al., submitted for publication), task-evoked pupillary responses of healthy undergraduates were recorded during a visual backward masking task. By comparing pupillary responses recorded in masked and no-mask conditions, it was possible to identify the processing load associated with targets vs. targets with masks, because pupil dilation in the no-mask condition reflected only target processing, while pupil dilation in masked conditions reflected both target and mask processing. Pupil dilation was significantly greater in longer (317 and 717 ms) SOA conditions than in the no-mask condition, suggesting that the mask only demanded additional processing when it was presented more than 100 ms after the target. This finding is consistent with masking

models that predict processing demands are highest in SOA conditions greater than approximately 100–200 ms, because attention must be shifted and shared between targets and masks in order to identify both stimuli in short-term visual memory (Loftus et al., 1988; Michaels and Turvey, 1979; Phillips, 1974).

A principal components analysis (PCA) of pupillary response waveforms was also computed in these previous studies (Verney, 2001; Verney et al., submitted for publication). PCA is often used as a method of reducing the large number of time points in psychophysiological data to a small number of meaningful factors. Three time-linked factors were found: early (0–0.7 s), middle (0.7–1.55 s) and late (1.55–3.0 s) factors. The early factor occurred when pupil constrictions to light (e.g. from the computer display) typically peak (Loewenfeld, 1999), so this factor may have indexed the pupillary light reflex. The early factor may also reflect the rapid inhibition of parasympathetic constriction pathways, which is thought to contribute to an early component of dilation responses during cognitive tasks (Steinhauer and Hakerem, 1992; see Verney and Granholm, 2004). The middle factor occurred when pupil dilation responses to cognitive tasks typically peak and reflect task processing load (Beatty, 1982; Beatty and Lucero-Wagoner, 2000; Steinhauer and Hakerem, 1992). In addition, dilation on the middle factor was greater in the no-mask condition (target processing only) relative to masking conditions (target and mask processing). In contrast, dilation on the late factor was significantly greater in masking conditions (target and mask processing) relative to a no-mask condition (target processing only), especially in longer SOA conditions. This pattern of results suggested that the middle factor indexed attentional allocation to targets, while the late factor indexed attentional allocation to masks.

In the present study, pupillary responses of patients with schizophrenia and age- and education-comparable healthy control participants were recorded during performance of a target-identification version of the visual backward masking with an equal-energy mask. Consistent with previous studies using similar masking tasks, we predicted that schizophrenia patients would show

target identification deficits only in a longer (317 ms) SOA condition. We also predicted that the PCA factor structure for pupillary response waveforms found in our previous masking task research (Verney, 2001; Verney et al., submitted for publication) would be replicated and that the patients with schizophrenia would show less dilation than controls on the middle (target) factor and greater dilation than controls on the late (mask) factor. This finding would be consistent with the hypothesis that attentional allocation problems (over-allocation to masks) at least partially contribute to masking task deficits in patients with schizophrenia.

2. Method

2.1. Participants

Outpatients who met *Diagnostic and Statistical Manual* (fourth edition, or DSM-IV; American Psychiatric Association, 1994) criteria for a diagnosis of schizophrenia based on the Structured Clinical Interview for DSM-IV or SCID-patient version (First et al., 1995) were recruited. These patients were recruited from the Veterans Affairs San Diego Healthcare System, University of California, San Diego, Outpatient Psychiatry Services, San Diego County Mental Health Services and private physicians. All patients were community-dwelling outpatients (i.e. not institutionalized). Symptom severity ratings were acquired for the patients using the Scales for the Assessment of Positive and Negative Symptoms (SAPS: $M=5.2$, $S.D.=5.1$; and SANS: $M=6.0$, $S.D.=4.1$; Andreasen and Olsen, 1982). Nonpsychiatric controls with no DSM-IV diagnoses of past or current mood or psychotic disorders (based on the SCID-nonpatient version; First et al., 1995) were also recruited from the general community, using local advertisements.

Patients and nonpsychiatric comparison participants were excluded for the following: (1) neurologic disorders (e.g. seizure disorder; head injury with loss of consciousness > 30 min); (2) history of any alcohol or substance dependence diagnosis (DSM-IV criteria) other than nicotine or caffeine

Table 1
Demographics of the two participant groups

Group	Age (years)	Education (years)	WAIS-R vocabulary scaled score	Gender (% male)	Ethnicity (%)	
Schizophrenia patients (<i>n</i> = 16)	50.0	12.8	10.2	81	Caucasian	69
	(8.4)	(2.3)	(3.7)		African American	6
					Hispanic	6
					Asian American	0
					Other/unknown	19
Nonpsychiatric participants (<i>n</i> = 16)	48.1	12.8	11.9	56	Caucasian	56
	(12.6)	(1.3)	(3.0)		African American	25
					Hispanic	13
					Asian American	0
					Other/unknown	6

Values represent means (with S.D.) and percentages where indicated. WAIS-R, Wechsler Adult Intelligence Scale—Revised (nonpsychiatric: *n* = 13; schizophrenia: *n* = 14). The groups did not differ significantly on any variable.

in the past year; (3) uncorrected/corrected visual acuity less than 20/30 based on a Snellen wall chart exam; (4) ocular medications, diseases or surgery that might affect pupil function; (5) near-chance performance in the no-mask condition (two patients with 55 and 65% accuracy and two controls with 45 and 50% accuracy) and (6) abnormal pupil measurements (resting diameter and/or pupillary response outliers > 2 S.D. or excessive artifacts in recordings). Table 1 displays the demographic characteristics of the final sample of 16 patients and 16 controls.

Controls did not differ significantly from patients on age, $t(30) = 0.51$, $P > 0.05$, education, $t(30) = 0.00$, $P > 0.05$, Wechsler Adult Intelligence Scale—Revised Vocabulary subtest scaled scores (Wechsler, 1981; missing for two patients and three controls), $t(25) = 1.26$, $P > 0.05$, or ethnicity (Caucasian vs. non-Caucasian), $\chi^2(1) = 0.53$, $P > 0.05$. There was a fairly large difference between the groups in gender composition, but the groups did not differ significantly in gender, $\chi^2(1) = 2.33$, $P > 0.05$, and analysis of variance (ANOVA) showed no main effect of gender or any significant gender X SOA condition interaction on any dependent variable.

Eleven patients were taking typical antipsychotic medications, two were taking atypical antipsychotic medications, seven were taking anticholinergic medications, and two were not taking any psychotropic medications. Medication status was unavail-

able for one patient. The following current medication values were calculated: (1) average daily dosage (mg) of antipsychotic medication as clinically recommended chlorpromazine equivalents (CPZE; Ereshefsky and Richards, 1990; Sadock and Sadock, 2000) and (2) average daily dosage (mg) of anticholinergic medication as clinically recommended bztropine equivalents (BZTE; Ereshefsky and Richards, 1990; Sadock and Sadock, 2000). The mean current daily dosage of CPZE for the 13 patients receiving antipsychotic medications was 309.8 mg (S.D. = 247.0) and the mean current daily dosage of BZTE for the 7 patients receiving anticholinergic medications was 3.4 mg (S.D. = 1.4).

2.2. Apparatus

A PC-compatible microcomputer and standard 15-inch color monitor was used to administer the masking task and store responses. A standard joystick equipped with two response buttons was used to collect responses. Participants sat facing the monitor with their head stabilized in a chin rest to maintain a distance of 77 cm between the subject and the computer monitor. Pupil area was recorded from the left eye using a Micromasurements System 1200 corneal-reflection-pupil-center infrared pupillometer (Micromasurements, Inc., Farmington, CT). A video camera sensitive to infrared light and an infrared light source were

positioned 24 cm from the participant below the field of view between the participant and the computer monitor. Analog pupil area was digitized at a 60 Hz sampling rate and saved for later offline processing. For consistency with the literature, area was then converted to diameter for all analyses. The resolution of the pupillometer was 0.05-mm diameter.

3. Procedure

On the backward masking task, two vertical lines of different length (2 and 3 cm) were presented side-by-side 3 cm apart randomly offset (i.e. not aligned at the top or bottom) in the center of the monitor. Subjects were asked to identify the longer of the two lines by pressing either the right or left response button corresponding to the right and left line. The targets were randomly and equally distributed to the right and left side. Both detection accuracy and speed were emphasized with the instruction, 'Try to be as accurate as you can, but also be as fast as you can.' The target stimuli were followed by longer masking lines of 4 cm presented in the same locations as the target stimuli. In addition to a no-mask condition, the interval between the onset of the target and onset of the mask (stimulus onset asynchrony or SOA) was 50, 67, 100, 134 and 317 ms, for a total of six conditions. These SOAs were chosen to be similar to those used in previous studies, within the constraints of the video refresh rate of the monitor (≈ 17 ms cycles). Due to the video refresh rate limitations, targets and masks were each presented for 17 ms.

Prior to the masking task, a calibration was conducted to ensure participant–pupillometer agreement on center of visual field. At the beginning of each trial, a green fixation square (0.85×0.85 cm, 0.63° of visual angle and 7 lux) was presented in the center of the monitor (with a black screen background) along with a high-pitched tone (1500 Hz for 500 ms). The fixation square and tone served as visual and auditory cues to warn the participant to prepare for the trial's target stimulus. When the participant's left pupil was detected as fixating on the green square for at least 200 ms, the program would terminate the

fixation square and administer the trial. This procedure ensured that the participant was attending to the center of the screen, where stimuli were presented, and not blinking when the targets were flashed. Three seconds after the onset of the target stimulus, a low-pitched tone (800 Hz for 500 ms) served as an auditory cue signaling the end of the trial. Participants were asked to try to refrain from blinking during this trial period and only blink during a 3-s inter-trial interval.

Following 24 practice trials (4 per condition), 20 trials per condition were randomly presented, for a total of 120 test trials. The practice trials began with the easiest conditions, namely, two no-mask trials followed by two 317 ms SOA trials. The SOA durations of the remaining 20 practice trials were randomly blocked. For the first half of the practice, computer-automated feedback was provided regarding correctness of participant's response. Feedback was not provided during the test phase of the study. During test trials, a moment of rest was allowed after each presentation of six trials since the computer required time to periodically save strings of pupillary response data. In addition, each participant was allowed a few minutes to rest halfway through the test. The entire task (i.e. instructions, practice and test) typically required less than 30 min. The percentage of correct target identifications and median response time for the 20 trials for each condition were recorded.

3.1. Data analysis

Graphic displays of raw pupil diameter data were first visually inspected for gross artifacts by a trained technician and discarded for major artifacts and excessive blinking. A computer algorithm was then used to remove eye blinks and other minor artifacts from other trials by linear interpolation. A seven-point smoothing filter was then passed over the data. For each participant, an average pupillary-response waveform was then calculated for the artifact-free trials of each SOA and no-mask condition. To remove individual differences in resting pupil size, *baseline pupil size* for each condition waveform (i.e. average of five pupil diameter samples recorded 100 ms prior to

onset of the target display) was then subtracted from all subsequent samples for that waveform. *Peak dilation* for each mean condition waveform was then determined by picking the largest pupil dilation sample (change from baseline) within a 500–2500 ms window after onset of the target display. A PCA with varimax rotation was also performed on the 180 time point samples (i.e. 3 s) of the mean pupillary response waveform for each of the six conditions for each participant. Three factors were expected based on our previous research (Verney, 2001; Verney et al., submitted for publication).

To test the hypothesis that the patients would show significantly impaired detection accuracy in longer (317 ms), but not shorter, SOA conditions, planned group comparisons of detection accuracy within each condition were computed using Bonferroni-corrected, independent samples, one-tailed *t*-tests ($P < 0.05/6 = 0.008$). Median response time was examined in a 2 (groups) \times 6 (conditions) split-plot ANOVA, which treated group as a between subjects factor and conditions as a within subjects factor. Pupillary responses were examined by computing a separate 2 (groups) \times 6 (conditions) split-plot ANOVA, which treated group as a between subjects factor and conditions as a within subjects factor, for baseline pupil size and peak dilation. In addition, a 2 (groups) \times 6 (conditions) \times 3 (PCA factors) split-plot ANOVA was computed for the factor scores from the pupil waveform PCA. Greenhouse–Geisser correction was used when sphericity assumptions were violated. To follow-up significant interaction effects for each ANOVA, Dunnett's procedure was used to compare each masked condition with the no-mask condition, because the difference in processing demands between masked and no-mask conditions was of primary interest. Finally, a planned 2 (groups) \times 2 (middle vs. late PCA factors) contrast was computed to test the hypothesis that the patients with schizophrenia would show less pupil dilation than controls on the middle (target) factor, but greater dilation than controls on the late (mask) factor. Effect sizes were computed as the proportion of variance accounted for by ANOVA effects (η^2) and Cohen's *d* for between-group *t*-tests.

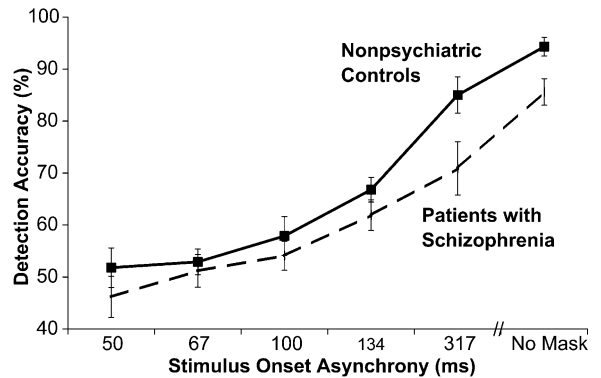


Fig. 1. Percent correct target detections in each masking and no-mask condition for each participant group. Error bars are ± 1 S.E.

4. Results

4.1. Task performance

Fig. 1 presents target detection accuracy in each condition for each group. As predicted, the patients with schizophrenia detected significantly fewer targets than controls in the 317 ms SOA condition, $t(30) = 2.56$, $P = 0.008$, $d = 0.93$, but did not differ significantly in any other masking condition. The patients with schizophrenia also detected fewer targets than controls in the no-mask condition, $t(30) = 3.08$, $P = 0.004$, $d = 1.12$. To control for this group difference in the ability to accurately perceive the target lines even when no mask was present, the groups were again compared in the 317 ms condition using no-mask condition detection accuracy as a covariate, and the groups still differed significantly, $F(1, 29) = 2.86$, $P = 0.05$, $\eta^2 = 0.09$.

Table 2 shows average median response time in each condition for each group. The ANOVA showed no main effect of group on overall response time, $F(1, 30) = 1.80$, $P > 0.05$, $\eta^2 = 0.06$, and the interaction between-group and condition was not significant, $F(5, 150) = 1.44$, $P > 0.05$, $\eta^2 = 0.05$, but response time did differ significantly across conditions, $F(5, 150) = 8.45$, $P > 0.05$, $\eta^2 = 0.22$. Significantly slower response times were found in all masking conditions relative to the no-mask condition (Dunnett's tests, $P < 0.05$).

Table 2
Average median response times in each condition for each group

Group	SOA condition					
	50 ms	67 ms	100 ms	134 ms	317 ms	No mask
Schizophrenia patients	954 (169)	979 (192)	927 (141)	917 (140)	1017 (239)	825 (137)
Nonpsychiatric participants	936 (310)	877 (260)	856 (268)	853 (257)	842 (272)	716 (170)

Values represent means (with S.D.).

Table 3
Pupillary response measures for the two participant groups

Dependent measure	SOA condition					
	50 ms	67 ms	100 ms	134 ms	317 ms	No mask
<i>Schizophrenia patients</i>						
Baseline pupil diameter (mm)	3.92 (0.73)	3.90 (0.75)	3.92 (0.72)	3.90 (0.74)	3.93 (0.75)	3.91 (0.73)
Peak dilation (mm)	0.16 (0.09)	0.15 (0.08)	0.15 (0.08)	0.15 (0.07)	0.16 (0.09)	0.15 (0.08)
Early PCA factor	0.29 (0.36)	0.29 (0.23)	0.27 (0.47)	0.26 (0.57)	0.24 (0.39)	0.33 (0.46)
Middle PCA factor	-0.31 (0.43)	-0.41 (0.43)	-0.40 (0.45)	-0.35 (0.42)	-0.42 (0.48)	-0.28 (0.64)
Late PCA factor	-0.14 (0.40)	-0.03 (0.62)	-0.09 (0.45)	-0.19 (0.34)	-0.09 (0.33)	-0.23 (0.24)
<i>Nonpsychiatric controls</i>						
Baseline pupil diameter (mm)	3.76 (0.69)	3.76 (0.68)	3.77 (0.68)	3.76 (0.67)	3.75 (0.70)	3.74 (0.67)
Peak dilation (mm)	0.21 (0.09)	0.20 (0.07)	0.22 (0.07)	0.23 (0.08)	0.25 (0.09)	0.23 (0.08)
Early PCA factor	0.25 (0.29)	0.34 (0.24)	0.25 (0.34)	0.33 (0.33)	0.37 (0.23)	0.37 (0.29)
Middle PCA factor	-0.24 (0.20)	-0.27 (0.19)	-0.26 (0.19)	-0.24 (0.19)	-0.26 (0.21)	-0.18 (0.22)
Late PCA factor	-0.36 (0.21)	-0.34 (0.26)	-0.31 (0.22)	-0.32 (0.21)	-0.24 (0.23)	-0.36 (0.23)

4.2. Baseline pupil size

Baseline pupil size (Table 3) did not differ significantly between the groups, $F(1, 30)=0.49$,

$P>0.05$, $\eta^2=0.016$, or across conditions, $F(5, 150)=0.49$, $P>0.05$, $\eta^2=0.016$, and the group by condition interaction was not significant, $F(5,$

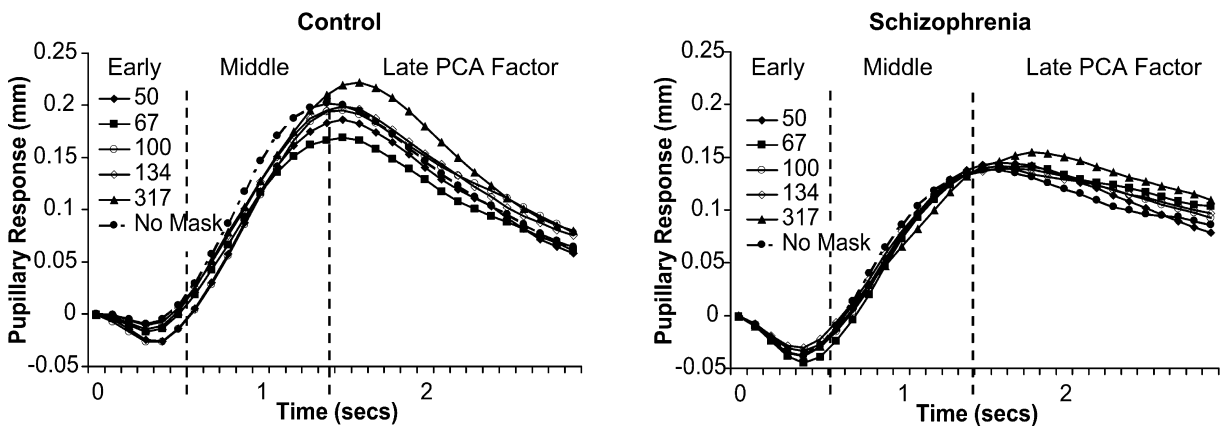


Fig. 2. Pupillary response waveforms recorded during a visual backward masking task are shown in each masking and no-mask condition for healthy controls (left) and patients with schizophrenia (right). Waveforms are divided into early, middle and late time-linked PCA factors.

150)=0.28, $P>0.05$, $\eta^2=0.009$. The groups, therefore, did not differ significantly in baseline pupil size.

4.3. Pupillary response

Fig. 2 shows the pupillary response waveforms and Table 3 shows peak dilation scores for all conditions for each group. Patients with schizophrenia showed significantly smaller overall peak dilation relative to controls, $F(1, 30)=6.96$, $P<0.05$, $\eta^2=0.188$. Peak dilation also differed significantly across SOA conditions, $F(3.4, 150)=2.61$, $P<0.05$ (Greenhouse–Geisser corrected), $\eta^2=0.080$, but the group \times condition interaction was not significant, $F(3.4, 150)=1.57$, $P>0.05$ (Greenhouse–Geisser corrected), $\eta^2=0.050$. The significant condition effect was examined using Dunnett's tests to compare each masking condition with the no-mask condition for the combined sample of both groups. Consistent with our previous research (Verney et al., 2001), peak dilation was significantly greater in the 317 ms SOA condition than in the no-mask condition ($P<0.05$), but peak dilation in all other masking conditions did not significantly differ from the no-mask condition.

4.4. Pupillary response waveform factor structure

Consistent with our previous research (Verney, 2001; Verney et al., submitted for publication), three prominent stable PCA factors accounted for 97.0% of the variance in the pupillary response data for the total sample. The factor structure for each group was identical to that of the total sample. All factors were internally consistent and well defined by the data (the lowest of the squared multiple correlations for factors from data was 0.69). The three factors formed a linear time course of the pupillary response waveform (Fig. 2): (1) an early factor from 0 to 0.7 s (eigenvalue=6.8); (2) a middle factor from 0.7 to 1.62 s (eigenvalue=21.8) and (3) a late factor from 1.62 to 3.00 s (eigenvalue=68.4). Table 3 presents PCA factor scores in each condition for both groups.

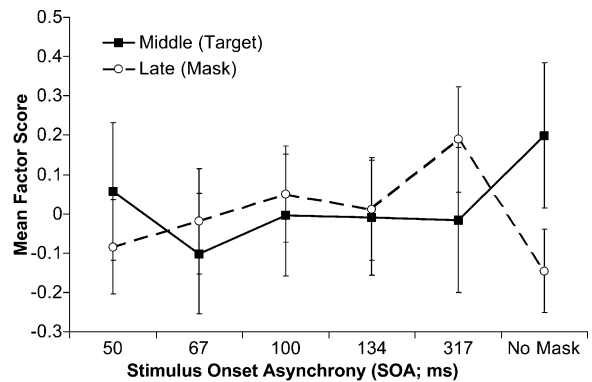


Fig. 3. Middle (left) and late (right) pupillary response PCA factor scores in masking and no-mask conditions for the combined sample of both groups. Error bars are ± 1 S.E.

The 2 (groups) \times 6 (masking conditions) \times 3 (PCA factors) ANOVA showed significant effects for the condition \times PCA factor interaction, $F(10, 300)=2.93$, $P<0.01$, $\eta^2=0.089$, and the group \times PCA factor interaction, $F(2, 60)=3.92$, $P<0.05$, $\eta^2=0.116$. None of the other main effects or interactions were significant. The significant condition \times PCA factor interaction was examined using Dunnett's tests ($P<0.05$) to compare each masking condition with the no-mask condition for the combined sample of both groups. For the early factor (Table 3), no masking condition differed significantly from the no-mask condition. For the middle factor (see Table 3 and Fig. 3), significantly less pupil dilation was found for all masking conditions relative to the no-mask condition ($P<0.05$). In contrast, for the late factor (see Table 3 and Fig. 3), pupil dilation tended to be greater in all masking conditions relative to the no-mask condition, but the only difference to reach statistical significance was the 317 SOA vs. no-mask comparison ($P<0.05$). The middle and late factors, therefore, showed the opposite pattern of dilation responses relative to the no-mask condition: less dilation was shown for masking relative to no-mask conditions for the middle factor, and greater dilation was shown for masking relative to no-mask conditions for the late factor, especially in the longest SOA condition.

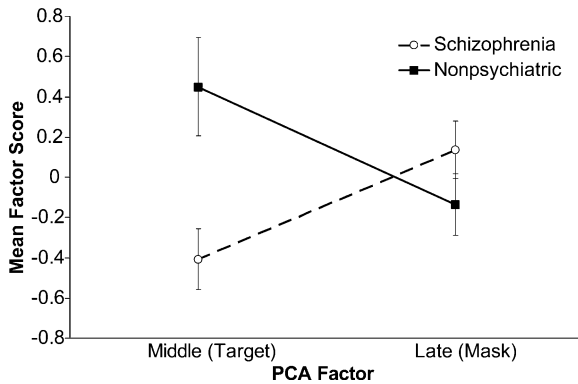


Fig. 4. Overall (collapsing across masking and no-mask conditions) middle and late pupillary response PCA factor scores for each group. Error bars are ± 1 S.E.

The significant group \times PCA factor interaction was due to the different pattern of middle and late factor scores shown by the two groups (Fig. 4). Consistent with the study hypothesis, controls showed greater overall dilation (collapsing across conditions) than patients with schizophrenia on the middle (target) factor, but showed less overall dilation than patients with schizophrenia on the late (mask) factor, $F(1, 30) = 8.75$, $P < 0.01$, $\eta^2 = 0.226$. For patients with schizophrenia, overall dilation on the late factor was significantly greater than that on the middle factor, $t(15) = 3.78$, $P < 0.01$, $d = 0.93$, whereas for controls overall dilation on the middle factor was marginally greater than on the late factor, $t(15) = 1.65$, $P = 0.12$, $d = 0.74$. Groups did not differ significantly on the early factor, and overall dilation for the early factor did not differ significantly from the middle or late factors for either group ($P > 0.05$).

4.5. Relationships between pupillary response and performance

To further explore the meaning of the different pupillary response PCA factor scores, post hoc correlations between task performance and the factor scores were computed for the total sample. To reduce the number of potential correlations, mean early, middle and late factor scores were computed for all conditions and correlated with mean target detection accuracy and mean response

time for all conditions. The early factor score did not correlate significantly with either detection accuracy ($r = 0.18$) or response time ($r = 0.02$). The middle factor was significantly correlated with response time ($r = -0.42$, $P < 0.05$) and a trend was found for detection accuracy ($r = 0.33$, $P = 0.067$). The late factor did not correlate significantly with either detection accuracy ($r = -0.07$) or response time ($r = 0.25$). In addition, if the greater late factor dilation found in the 317 SOA condition relative to the no-mask condition reflected attentional allocation to masks, then the difference between late factor scores in the 317 ms SOA condition and the no-mask condition (i.e. 317 SOA—no mask late factor scores) should be inversely correlated with detection accuracy. That is, more wasteful allocation of limited processing resources to masks should leave fewer spared resources for accurate target processing. Consistent with this prediction, the late (mask) factor difference score was significantly inversely correlated with detection accuracy ($r = -0.40$, $P < 0.05$), but not with response time ($r = 0.12$). In sum, greater dilation on the middle factor was correlated with faster, more accurate target detection, and greater allocation of attention to masks on the late (mask) factor difference score was correlated with poorer target detection.

4.6. Medication and symptom effects

Patients receiving anticholinergic medications ($n = 7$) did not differ significantly (two-tailed t -tests) from patients not receiving these medications ($n = 9$) on baseline pupil size, detection accuracy, peak dilation or any PCA factor score in any condition. In addition, current daily dosage of anticholinergic medication (BZTE) did not correlate significantly with any of these dependent variables in the patients. Similarly, daily dosage of antipsychotic medication (CPZE) did not correlate significantly with any of these dependent variables, except that greater CPZE dosage was associated with a smaller (more normal) factor score on the early factor, $r = -0.50$, $P < 0.05$. Positive symptom severity (SAPS total) was not significantly correlated with any dependent variable. Greater negative symptom severity (SANS total) was significantly

correlated with poorer detection accuracy in the 67 ms SOA condition, $\rho = -0.54$, $P < 0.05$. In summary, no meaningful, consistent associations between medications or symptoms and any of the dependent measures were found.

5. Discussion

The pupillary responses of patients with schizophrenia and healthy nonpsychiatric participants were recorded, while they performed a target-identification version of the visual backward masking task with an equal-energy mask. Consistent with numerous previous studies using similar types of masking tasks, patients with schizophrenia showed impaired detection accuracy only in the longest (317 ms) SOA condition (Balogh and Merritt, 1987; Braff and Saccuzzo, 1981; Braff et al., 1991; Green et al., 1994; McClure, 2001; Miller et al., 1979; Rund et al., 1993). To investigate the role of attentional mechanisms in producing this commonly observed impairment, pupillary responses were recorded as an index of attentional allocation to targets and masks. Consistent with our previous research with healthy undergraduates (Verney et al., 2001; Verney, 2001; Verney et al., submitted for publication), peak dilation was significantly greater in the 317 ms SOA condition than in the no-mask condition. This finding is consistent with masking models that predict attentional demands are highest in SOA conditions greater than 100–200 ms, because selective attention must be shifted and shared between targets and masks in order to identify both stimuli in short-term visual memory (Loftus et al., 1988; Michaels and Turvey, 1979; Phillips, 1974). Taken together, these behavioral and psychophysiological data indicate that the greatest masking task impairment was found in the SOA condition with the highest processing load. This suggests that masking impairments at SOA intervals greater than 100–200 ms may be due to abnormalities in resource allocation and/or resource availability (Nuechterlein and Dawson, 1984).

The present study replicated the PCA factor structure found for pupillary response waveforms in two previous masking studies of healthy undergraduates (Verney, 2001; Verney et al., submitted

for publication). Three time-linked factors were found: early (0–0.7 s), middle (0.7–1.62 s) and late (1.62–3.0 s) factors. No interesting group or condition differences were found for the early factor. The middle factor occurred when the extent of task-evoked peak pupil dilation is typically found to reflect the processing load associated with key task operations (Beatty, 1982; Beatty and Lucero-Wagoner, 2000; Steinhauer and Hakerem, 1992). The late factor occurred after this task processing load factor. Importantly, the middle and late dilation factors showed the opposite pattern of results when masking and no-mask conditions were compared. Less dilation was found for masking relative to no-mask conditions for the middle factor, but greater dilation was found for masking relative to no-mask conditions for the late factor, especially in the longest SOA condition. An index of target processing should show greater processing load in the no-mask condition, where only targets are presented, while an index of mask processing should show greater processing load in masking conditions. This was exactly the pattern of results found for the middle and late factors, respectively. In addition, greater dilation on the middle factor was correlated with faster, more accurate target detection, whereas greater allocation of attention to masks on the late factor (mask) difference score was correlated with poorer target detection. Taken together, these findings suggest that the middle factor indexed target processing and the late factor indexed mask processing.

This interpretation of the late factor as indexing mask processing is consistent with the Michaels and Turvey (1979) ‘replacement’ hypothesis that target processing is replaced by mask processing at SOAs greater than approximately 100–200 ms. Late (mask) factor dilation was greater in longer SOA conditions and differed significantly from the no-mask condition only in the longest (317 ms) SOA condition. This finding is also consistent with Phillips’ (1974) hypothesis that masking impairments after approximately 100 ms SOAs are due to limitations on controlled processes that select stimuli for identification in short-term visual memory.

An alternative interpretation of late factor dilation is that it simply reflected psychophysical

aspects of viewing two distinct stimuli in longer SOA conditions. That is, the later peak in the waveform might occur whenever two stimuli are presented at this rate, regardless of whether they are actively processed in any way. However, in Verney et al. (2001), participants passively viewed the visual backward masking task stimuli, and were told not to process them in any way (cognitive no-load condition). In that study, middle and late factor scores did not differ significantly between any SOA and no-mask conditions in the no-load condition. Therefore, simply viewing targets and masks in rapid succession did not produce greater dilation on the late factor in longer SOA conditions.

Another alternative interpretation of the late factor is that it reflected slowed or more sustained target processing in masked relative to no-mask conditions. That is, more sustained effort may have been required for target processing in masked conditions, because less target information was transferred to short-term memory before the mask was presented. Several findings, however, are not consistent with this interpretation. First, if greater late factor dilation reflected greater sustained processing for more degraded targets, greater late factor dilation should have been found in shorter, not longer, SOA conditions, because less target information is transferred prior to mask presentation in shorter intervals. Greater late factor dilation was found in longer, not shorter, intervals. In addition, the two groups also did not differ significantly in response times across conditions, so the increased late factor scores in the patients did not simply reflect slowed response time. Finally, the late factor score and the late (mask) factor difference score (i.e. 317 SOA—no-mask condition) were not significantly correlated with response time. The late factor, therefore, did not likely index slowed or sustained processing of targets.

The nonpsychiatric participants showed greater dilation on the middle (target) factor than on the late (mask) factor, suggesting that they selectively attended to targets more than masks. In contrast, patients with schizophrenia showed less dilation on the middle (target) factor than on the late (mask) factor, suggesting that they selectively attended more to masks than to targets. That is,

patients with schizophrenia wastefully allocated more processing resources to masks, leaving fewer resources spared for target identification. In the language of the Michaels and Turvey (1979) model, target processing was 'replaced' by mask processing to a greater extent for patients relative to controls. This finding is consistent with an attentional allocation problem that occurs in a later stage of processing (e.g. short-term visual memory) when inputs compete for stimulus identification resources (Knight, 1992, 1993). This finding is also consistent with the hypothesis of Green et al. (1997) of an 'attentional disengagement' deficit that occurs at interstimulus intervals greater than 70 ms. Thus, an attentional allocation problem in schizophrenia may partially contribute to masking task deficits on this type of masking task.

Research on the nature of masking task deficits in schizophrenia has typically focused on two other masking mechanisms: integration and interruption (or inhibition; Breitmeyer, 1984; Breitmeyer and Ganz, 1976; Breitmeyer and Ogmen, 2000). Integration is the fusing together of the target and mask representations into an indecipherable image and is maximal at shorter (e.g. 20 ms) intervals. Interruption occurs when processing of the target is disrupted by the incoming processing signals of the mask, resulting in only partial processing of the target, and typically occurs between 20 and 70 ms intervals. In a neural system model of these two factors (Breitmeyer, 1984; Breitmeyer and Ganz, 1976; Breitmeyer and Ogmen, 2000), a transient visual pathway (magnocellular tract) rapidly detects and transmits low spatial frequency information about stimulus onset, offset and location, while a sustained visual pathway (parvocellular tract) responds more slowly and transmits high spatial frequency information about the finer details of stimulus processing. Integration occurs when information about the target and mask combine in sustained channels, and interruption occurs when transient mask activity inhibits sustained target activity, which inhibits correct identification of the target. There is some evidence that masking task deficits in schizophrenia may involve deficits in transient magnocellular pathways (Butler et al., 2001; Cadenhead et al., 1998; Green et al., 1994). However, a more recent expanded masking model

(Breitmeyer and Ogmen, 2000; Purushothaman et al., 2000) emphasizes that synchronized gamma frequency oscillations in sustained channels may bind features of visual stimuli together, and there is some evidence that aberrant gamma range activity in schizophrenia may lead to sustained channel deficits (Green and Nuechterlein, 1999; Green et al., 1997, 2003; Kwon et al., 1999). Deficits in both channels may also be present.

The question of whether transient or sustained channel deficits or attentional resource allocation deficits better account for masking impairments centers on the question of whether input stages (e.g. quality of initial icon representation formation) or subsequent stages (e.g. competition for and allocation of higher cortical stimulus identification resources) can better account for deficits. This is an old controversy about peripheral vs. more central deficits in schizophrenia research, which may be a meaningless distinction (Saccuzo, 1977). For example, from the bottom-up, the magnocellular (transient) pathway helps orient attention toward salient stimuli, so overactive transient channel activity may be one mechanism by which a second input orients processing resources away from sustained processing of earlier relevant inputs (Butler et al., 2001, 2002). From the top-down, attention manipulations are known to modulate masking effects (Ramachandran and Cobb, 1995; Havig et al., 1998). Of note, patients with schizophrenia show normal performance on forward masking studies (i.e. when the mask precedes the target; Slaghuis and Bakker, 1995; Saccuzo et al., 1996), which may indicate a central, rather than peripheral transient channel, impairment (Saccuzo et al., 1996). Other studies have also implicated central rather than transient channel deficits in schizophrenia (Keri et al., 2000). Regardless of whether the impairment is more central or peripheral, the present study found an abnormal over-allocation of attention to masks rather than targets in patients with schizophrenia.

Bachmann (1994, 1997) described a neurophysiological masking model that emphasizes the role of attention and activation mediated by thalamic reticular systems (Bachmann, 1994, 1997). In this model, identification and conscious awareness of a stimulus requires both a specific (SP) input

carrying content information (e.g. orientation, color, form, spatial frequency, etc.) through classic visual pathways to visual cortex and a nonspecific (NSP) input from the thalamic reticular system. The NSP input is required to raise the level of activation of SP inputs to a level sufficient for awareness and identification. Because the NSP input is slower than the SP input, the timing of some masking intervals can lead to enhancement of mask SP inputs by target NSP inputs on backward masking tasks. Consistent with this model, several thalamic mechanisms are known to selectively enhance transfer and processing of sensory-perceptual data, such as sensory gating by the nucleus reticularis thalami (Scheibel, 1980; Crick, 1984) and cortical activity synchronization in cortico-thalamic loops (Alexander et al., 1986; Crick, 1984). Interestingly, gamma oscillations that may be involved in masking effects can be produced by stimulus-driven activation of cortico-thalamic loops (Tallon-Baudry et al., 1997).

It is possible that pupillary responses reflect these reticular-thalamic activation inputs. Pupil dilation is a function of the balance of activity between opposing sympathetic and parasympathetic systems. The brainstem nuclei driving these autonomic systems receive extensive inhibitory input from the reticular system and structures in cortico-thalamic loops. For example, electrical stimulation of the midbrain reticular formation in animals results in pupillary dilation (Loewenfeld, 1999). It is through these reticular connections that pupillary responses may provide an index of central brain systems that govern selective enhancement of sensory-perceptual inputs or attentional allocation (Beatty, 1986). In the present study, the reduced overall pupillary responses and abnormal distribution of pupillary responses between targets and masks found in patients with schizophrenia may implicate dysfunction in the general level of reticular system activation and/or the allocation of activation among inputs. Several investigators have emphasized thalamic mechanisms and cortico-thalamic loop dysfunction in schizophrenia (Andreasen et al., 1994; Carlsson and Carlsson, 1990; Swerdlow and Koob, 1987).

Poor premorbid status and severe negative symptoms have been found to be associated with

poorer masking task performance in other studies (Knight, 1992, 1993; Green and Walker, 1986). Perry and Braff (1994) also found that the poorer masking task performance was associated with more severe thought disorder. In the present study, no significant associations were found between positive or negative symptoms and masking task performance or pupillary responses. The patients in the present study, however, were community-dwelling outpatients with relatively mild symptoms, so restricted range of symptom severity may have reduced symptom correlations. We also did not find any effect of medications on performance. Some previous studies found better masking task performance in patients taking antipsychotic medications (Braff and Saccuzzo, 1982; Butler et al., 1996), but one found no differences between patients on and off medications (Butler et al., 2002). Consistent with previous studies (Granholm et al., 1997, 1998, 2000; Steinhauer and Hakerem, 1992), medications also did not significantly impact pupillary responses in the present study.

This study had several limitations. For example, a standard target exposure duration was used in this study, because the limitations of our computer display did not permit matching participants for no-mask detection accuracy using a critical stimulus duration (CSD) procedure. At the exposure used, the patients with schizophrenia detected significantly fewer targets in the no-mask condition. This suggests that the patients may not have received the same amount of target information in the no-mask condition. Any group might show masking deficits relative to another group, if not provided with an equivalent amount of target information. We attempted to account for this by using no-mask detection accuracy as a covariate in group comparisons, and still found impairment in the longer SOA condition. However, masking effects are less ambiguous when no-mask performance is equated using a CSD or other procedure (e.g. graded increase in the difference in length between target lines). Future studies should also further validate our interpretations of the pupil waveform PCA factors found in our masking studies. Additional evidence that the late factor reflects mask processing would be provided by manipulating the information value of the mask or

using a procedure that required greater attention to masks and testing for increased dilation on the late factor with increased mask load. These studies are currently under way in our laboratory.

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