Diminished Parietal Cortex Activity Associated with Poor Motion Direction Discrimination Performance in Schizophrenia

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The results of multiple investigations indicate visual motion–processing abnormalities in schizophrenia. There is little information, however, about the time course and neural correlates of motion-processing abnormalities among these subjects. For the present study, 13 schizophrenia and 13 healthy subjects performed a simple motion direction discrimination task with peripherally presented moving grating stimuli (5 or 10 deg/s). Dense-array electroencephalography data were collected simultaneously. The goal was to discern whether neural deviations associated with motion-processing abnormalities among schizophrenia patients occur early or late in the visual-processing stream. Schizophrenia patients were worse at judging the direction of motion gratings, had enhanced early neural activity (about 90 ms after stimulus onset), and deficient target detection–related late neural activity over parietal cortex (about 400 ms after stimulus onset). In addition, there was a strong association (accounting for 36% of performance variance) between poor behavioral performance and lower target detection–related brain activity among schizophrenia patients. These findings suggest that abnormalities in later stages of motion-processing mechanisms, perhaps beyond extrastriate cortex, may account for behavioral deviations among schizophrenia subjects.

Keywords: ERP, motion, motion processing, P1, parietal cortex, schizophrenia, smooth pursuit, visual

Introduction

Problems with smooth-pursuit eye-movement performance have been consistently observed among schizophrenia subjects (e.g., Clementz and McDowell 1994; Hutton and Kennard 1998). Smooth pursuit is a complex behavior that requires constantly following a moving stimulus by keeping it on the fovea. Successfully performing smooth-pursuit tasks requires, at the least, sufficient motion-processing abilities (Lisberger et al. 1987; Stanton et al. 2005), primarily an extrastriate cortex function (Newsome and Pare 1988; Pasternak and Merigan 1994) and successful use of this perceptual motion information for generation of the correct motor response, primarily a frontal cortex and cerebellar function (Lencer and Trillenberg 2008). Determining if perceptual motion processing is normal, separate from the ability to generate the proper motor response, therefore, is important for understanding the cause of schizophrenia subjects' smooth pursuit–related deficits. The present study adds to this literature by directly measuring neural activity during motion processing among schizophrenia patients to determine when in the motion analysis hierarchy abnormalities accounting for their poor behavioral performance occur.

Behavioral studies suggest a motion perception deficit among schizophrenia patients (Stuve et al. 1997; Chen et al. 2003, 2004, 2005; Slaghuis et al. 2005; Kim et al. 2006; O'Donnell et al. 2006; Clementz et al. 2007). Blood flow–based functional neuroimaging studies also are consistent with the conclusion of a motion-processing problem because schizophrenia patients have lower activity in cortical area V5 that is associated with poor smooth-pursuit performance (Hong et al. 2005; Lencer et al. 2005). The temporal resolution of functional magnetic resonance imaging, with a sampling rate slower than the visual processing pathway's neural transmission time, limits the determination of "when" abnormalities occur in the visual-processing stream.

Existing behavioral and brain imaging data on motion perception in schizophrenia are also consistent with a theory of magnocellular (M) pathway dysfunction (Schechter et al. 2003; Butler et al. 2005; Butler and Javitt 2005; Kim et al. 2005, 2006), because this pathway provides the majority input to motion area V5 (Maunsell et al. 1990). Kim et al. (2006) proposed that motion-processing deficits in schizophrenia are caused by impaired bottom-up M-pathway input to V1. Indeed, early visual processing deficits have been reported in schizophrenia (Butler et al. 2008), with some specific functions reported as being intact or even enhanced among patients (Dakin et al. 2005). A complication for this theory is that schizophrenia patients have accurate saccades to smooth-pursuit targets (Clementz 1996; Kim et al. 1997) and can accurately perform a motion direction detection task in central vision (Chen et al. 2003), indicating adequacy of at least some later V5-supported functions (Newsome et al. 1985; Dursteler and Wurtz 1988).

An alternative model (Chen et al. 2003, 2004) suggested that schizophrenia patients have a "late-stage" motion-processing abnormality. According to this theory, motion deficits in schizophrenia are accounted for by dysfunction in the motion-processing stream "no earlier" than V5. Chen et al. (2003) compared local (simple moving gratings) and global (stochastic motion of random dot patterns) motion direction judgments in schizophrenia. Animal studies indicate that global motion processing occurs at a later stage in the motion hierarchy (i.e., in MT/MST, part of the V5 complex; Born and Tootell 1992; Movshon and Newsome 1996). Chen et al. (2003) reported that schizophrenia subjects had elevated thresholds on a global but not local direction detection task and concluded that these patients had intact bottom up (V1) but impaired later stage (V5) motion processing. Chen et al. (2004) provided additional support for a late-stage motion-processing problem in schizophrenia by demonstrating that their velocity discrimination performance was independent of stimulus

contrast. Animal studies indicate that neural responses to motion stimuli increase with stimulus contrast at early (V1) but not late stages of motion processing (Sclar et al. 1990; Pasternak and Merigan 1994), so Chen et al. (2004) again concluded that schizophrenia patients' motion-processing problems must occur at a later stage of motion analysis.

Given the available information, it is uncertain when and where motion-processing difficulties among schizophrenia subjects are manifest neurophysiologically. Two competing theories have sound empirical support. An important piece of missing information relevant to both theories is high temporal resolution assessment of brain activity during the perception of motion stimuli among schizophrenia subjects. For the present study, neural activity from early in the visual-processing stream during the viewing of motion stimuli was directly measured using dense-array EEG. Motion-processing performance was evaluated by using a simple motion direction detection task similar to the one used by Chen et al. (2003) where only a simple motion direction judgment of a grating stimulus is required (there is no need to either judge the speed of or generate an eye movement to the stimulus). In addition, these stimuli were presented within the context of a visual oddball design (1 motion direction, defined as the target, was infrequent and required a button press when perceived) to determine if target-identification abnormalities could account for difficulties with behavioral measures of motion perception (e.g., Alain et al. 1998; van der Stelt et al. 2004; Clementz et al. 2008). The results of this study will be important because of the ability to evaluate, using a "combination" of behavioral and high temporal resolution brain-activity measurements, whether schizophrenia subjects' motion-processing deficits occur early or late in the processing stream.

Materials and Methods

Participants

Thirteen chronic outpatients with Diagnostic and Statistical Manual of Mental Disorders (4th Ed) (DSM-IV) (American Psychiatric Association 1994) schizophrenia (mean age = 43 years; standard deviation (SD) = 8; range = 26–55; 6 females) and 13 healthy (mean age = 41 years, SD = 8; range = 27–54; 7 females) persons participated in this study. All participants were right handed and had normal or corrected-to-normal vision. Diagnoses were confirmed by Structured Clinical Interview for DSM-IV (First et al. 1995) and to rule out Axis I disorders in healthy subjects. Participants had no neurological hard signs, clinically confounding treatments, history of head trauma, and current psycho-active substance use disorders. All patients were clinically stable (Global Assessment of Functioning $M = 34$, SD = 4) on antipsychotic medications (11 on atypical and 2 on typical; Mean chlorpromazine (CPZ) equivalent dose = 502 mg, SD = 235) for >8 weeks prior to participation (i.e., based on diagnostic interview, patients' clinical and medication status had not changed during this period). A host of previous studies suggest that visual-processing deficits observed in schizophrenia are not associated with antipsychotic medication treatments (see, e.g., Butler et al. 2007, for a discussion). Indeed, there were no significant associations between CPZ equivalent dose and any behavioral/brain-imaging measure for schizophrenia patients in this study. After the study, participants were paid $15/h for their participation. The UGA Institutional Review Board approved this study, and participants provided informed consent prior to testing.

Stimuli and Procedure

Stimuli were presented on a 21'' high-resolution flat surface color monitor with a refresh rate of 100 Hz that was 60 cm from the participants' eyes. A centrally located fixation cross on which subjects were instructed to remain fixed was visible throughout testing. The relevant visual stimulus was a light/dark vertically oriented sinusoidal grating (0.4 cycles/deg), 3.75 by 3.75 deg, presented at 100% contrast with a mean luminance of 20 cd/m² against a 0.1 cd/m² background (see Fig. 1). To allow for assessment of possible laterality effects in motion-processing differences between-groups, a grating was presented with its inside edge 5 deg to the left or right of central fixation. This peripheral presentation had the added benefit of strongly activating the M-pathway (e.g., Kremláček et al. 2006), which provides the bulk of the input to cortical motion areas. Each trial started with a 1500 ms (± 150 ms) fixation period. The grating then randomly appeared either to the left or right of fixation for 500 ms, moved horizontally leftward or rightward (via temporally modulating the bars), which, depending on the side of presentation, was either toward or away from fixation. Grating speed was either 5 or 10 deg/s. We employed relatively slower speed stimuli because speed discrimination at higher speeds may allow for the use of changes in perceived contrast (Pantle 1978). Subjects were instructed to respond with a key press when the gratings moved outward (25% of trials). Participants were thoroughly practiced until they understood the nature of the task and understood the importance of maintaining central fixation throughout testing. Each participant completed 640 trials (320 trials for each speed; half in each direction).

EEG Recording

EEG data were measured using a 256-channel Geodesic Sensor Net and NetAmps 200 amplifiers (Electrical Geodesics Inc; EGI, Eugene, OR). Recordings were referenced to the vertex sensor (Cz). As is standard with high input impedance amplifiers like those from EGI, sensor impedances were below 50 kΩ. Data were analog filtered from 0.1 to 100 Hz, digitized at 500 Hz, stored on a disk for later off-line analysis, and recorded continuously throughout the testing.

Behavioral and EEG Data Analyses

For behavioral assessments, d-prime, response bias, and reaction time were calculated for each moving speed and for each group. For EEG scoring, raw data were checked for bad channels (less than 5% for any participant), which were replaced using a spherical spline interpolation method (as implemented in BESA 5.1; MEGIS Software, Gräfelfing, Germany). Data were transformed to an average reference and digitally filtered from 1 to 50 Hz (12 db/octave roll-off, zero phase). Eye blink and cardiac artifact correction was achieved by using the ICA toolbox in EEGLAB 4.515 (Delorme and Makeig 2004) under Matlab (Version 7.0, MathWorks, Natick, MA).

Only the visual event-related potentials (VERPs) elicited by non-target events (centripetal gratings) without a key press response (correct rejections) and target events (centrifugal gratings) with a key-press response (correct detections) were included in the analyses. Trials with an eye movement to the peripheral target also were eliminated from the analysis. Individual trials of 800-ms duration (beginning 200 ms before event onset) were averaged separately for nontargets and targets. Trials with activity greater than 75 µV were automatically eliminated from further processing. Grand averages were baseline corrected using the 200 ms pre-event period.

Direct comparison of VERPs for nontarget and target stimuli is supported and facilitated by the fact that motion onset VERPs are not specific for motion direction (Maurer et al. 2004). VERP-component latency identification was performed using programs written in Matlab.
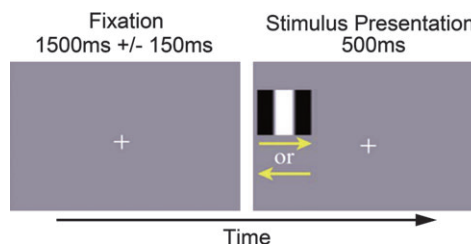

Fixation 1500ms +/- 150ms **Stimulus Presentation** 500ms

Time

Figure 1. Illustration of the stimuli.

To identify components above baseline noise level, global field power (GFP) plots were derived for every subject and condition. The only identifiable components in the GFP plots for all subjects in all conditions were the P1, N1, P2, and N2 (see Figs. 2 and 3). The latency for the P1, N1, and P2 components for each condition were determined from the peak in the individual GFP plots. Magnitudes of the P1, N1, and P2 (in μV) were quantified by using the maximal positive or negative potentials (positive for P1 and P2, negative for N1) over the hemisphere contralateral to the visual field of stimulation (averaged over 5 sensors that included and surrounded this peak, see Fig. 2) at the peak latency of the component (±4 ms). As is typical for quantifying target-related activity in VERP studies (e.g., Urban et al. 2008), a difference wave was created by subtracting the nontarget from the target responses. This resulted in an area of target-related negativity over the superior parietal area around the time of the N2. The magnitude of this activity was quantified by averaging over 15 sensors that best captured this

difference (see Fig. 3) averaged from 350 to 525 ms following stimulus onset. The similarities in spatial distributions of the VERP components also were determined by calculating Pearson correlations between schizophrenia and healthy groups using the sensors as observations and voltage as the dependent variable.

After VERP analyses calculated on voltage data at the sensors, we used L2 minimum norm (Hämäläinen and Ilmoniemi 1984) to estimate brain regions accounting for "between-group differences" on each component observed in the sensor space data. For the minimum norm approach, the source configuration is fixed a priori (fixed-source locations are specified on the surface from which EEG signals emanated, e.g., the cerebral cortex). Given the measured data, source strength values are estimated for each location at each time point. In BESA 5.1, 713 locations are evenly distributed on the surface of a smoothed standard MRI of the brain. At each location, sources are located 10% and 30% below the cortical surface (for a total of 1426
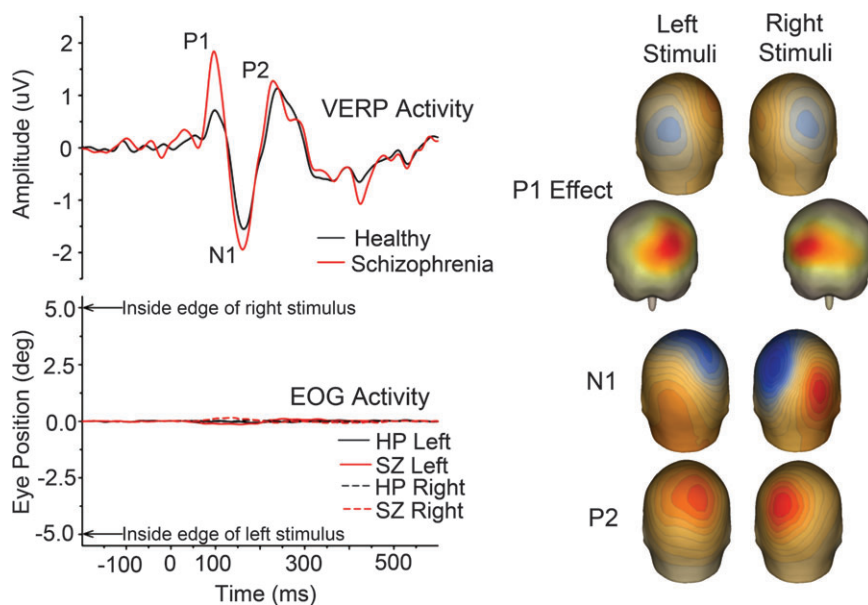

Figure 2. Neural activities associated with early VERP components during motion processing. The upper time by voltage plot shows the P1, N1, and P2 time courses for schizophrenia and healthy subjects averaged over left and right stimuli presentations. The lower time by voltage plot shows horizontal eye movements in response to left and right stimuli and illustrates that both schizophrenia and healthy subjects followed the instruction to maintain central fixation. All plots of neural activities are seen from the occipital view. The upper voltage topography shows the main effect of group on P1 VERP amplitude (difference between schizophrenia and healthy subjects) in response to both left and right stimuli. The associated brain plot shows the source activities associated with that voltage difference main effect. The lower 2 voltage topographies show the spatial distributions of the N1 and P2 VERP responses, averaged over group and stimuli types, for which there were no effects of group membership.
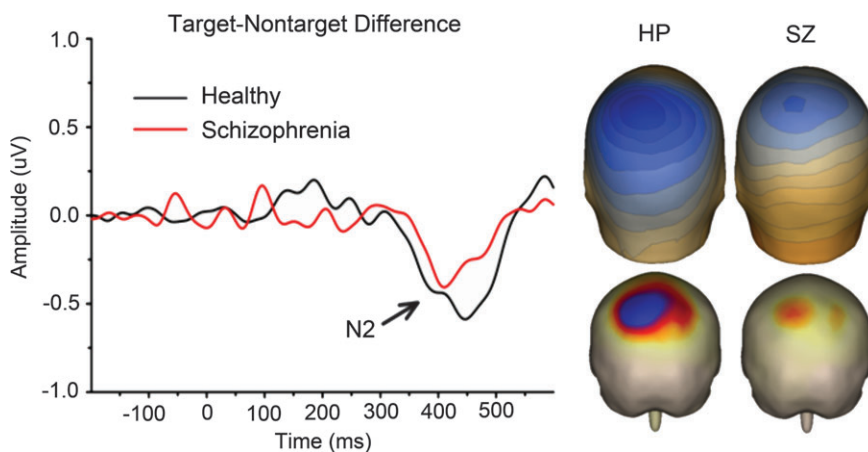

Figure 3. Neural activities associated with late VERP activity during motion processing. The time by voltage plot shows the N2 target–nontarget response for schizophrenia and healthy subjects averaged over left and right stimuli presentations (there was no effect of stimulus laterality on topography of this response). Neural activities are seen from the occipital view. The voltage topographies show the N2 voltage difference separately for schizophrenia and healthy subjects. The associated brain plot shows the source activities associated with those voltage differences and illustrate the group main effect as strength of the target-detection response in parietal cortex.

sources). The source used at each location in the final analyses is the one with the largest magnitude.

Results

Behavioral Results

There was no difference between groups on the number of usable trials for either nontarget (schizophrenia $M = 443$, SD = 31; healthy $M = 453$, SD = 19) or targets events (schizophrenia $M = 137$, SD = 23; healthy $M = 146$, SD = 5). A Group (schizophrenia, healthy) by Grating Speed (5 deg/s, 10 deg/s) by Stimulus Location (left, right) repeated-measures analysis of variance (ANOVA) was used to test for differences on d-prime, response bias, and reaction time. For d-prime, the main effect of Group was significant, $F(1,24) = 12.7$, $P = 0.002$; healthy participants ($M = 4.1$, SD = 0.3) were better than schizophrenia patients ($M = 3.2$, SD = 0.3) at detecting target events. There were no other significant effects involving group membership, and there were no significant effects involving group membership on either response bias (schizophrenia $M = 0.11$, SD = 0.36; healthy mean = 0.04, SD = 0.17) or reaction time (schizophrenia $M = 669$ ms, SD = 114; healthy $M = 628$ ms, SD = 118).

EEG Results

A Group (schizophrenia, healthy) by Grating Speed (5 deg/s, 10 deg/s) by Stimulus Location (left, right) by Event Type (target, nontarget) repeated-measures ANOVA was used to test for effects on the latency and amplitude for each of the 3 early VERP components (P1, N1, and P2). For P1 latency, there was a significant main effect of Event Type, $F(1,24) = 5.0$, $P = 0.034$, with subjects having earlier responses to targets ($M = 90$ ms, SD = 7) than to nontargets ($M = 94$ ms, SD = 8). For P1 amplitude, there were only significant main effects of 1) Group, $F(1,24) = 16.3$, $P < 0.001$ (schizophrenia patients, $M = 2.4$ μV, SD = 0.6, had larger responses than healthy subjects, $M = 1.4$ μV, SD = 0.4) and 2) Event Type, $F(1,24)=8.4$, $P=0.008$ (subjects had larger responses to targets, $M = 1.9$ μV, SD = 0.6, than to nontargets, $M = 1.6$ μV, SD = 0.6). The schizophrenia and healthy subjects had highly similar spatial distributions of P1 voltage over the head ($r = 0.93$), suggesting that similar source activities between-groups were generating this response. The between-group difference on P1 amplitude was accounted for by greater occipital–parietal cortex activity in the hemisphere contralateral to the visual field of stimulation among schizophrenia compared with healthy subjects (see Fig. 2).

For N1 latency, there were no significant effects. For N1 amplitude, there was only a significant main effect of Event Type, $F(1,24) = 6.7$, $P = 0.016$ (subjects had larger responses to targets, $M = -2.5$ μV, SD = 0.7, than to nontargets, $M = -2.3$ μV, SD = 0.8). Again, the schizophrenia and healthy subjects had highly similar spatial distributions of N1 voltage over the head ($r = 0.92$), suggesting that similar source activities between-groups were generating this response (Fig. 2). There were no significant effects involving group membership or event type on either P2 latency or P2 amplitude (Fig. 2), but the 2 groups, again, had highly similar P2 voltage patterns over the head ($r = 0.84$).

A Group (schizophrenia, healthy) by Grating Speed (5 deg/s, 10 deg/s) by Stimulus Location (left, right) repeated-measures ANOVA was used to test for N2 amplitude differences
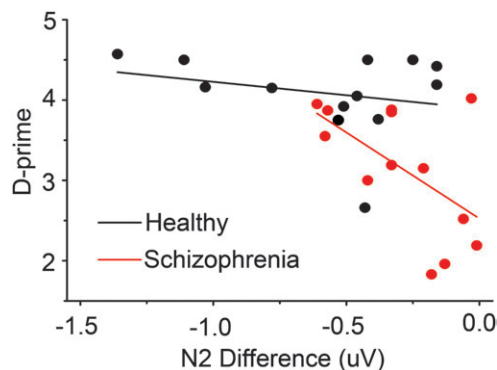

Figure 4. Scatter plot of the relationship between behavioral performance (d-prime) and target-related neural activity (target–nontarget voltage over parietal cortex) at the time of the N2 VERP response.

(target–nontarget). There was only a significant main effect of Group, $F(1,24) = 6.0$, $P = 0.022$, with healthy subjects ($M = -0.6$ μV, SD = 0.4) having larger responses than schizophrenia patients ($M = -0.3$ μV, SD = 0.2).[1] The groups were less similar on spatial distribution of the N2 ($r = 0.53$) than they were for the earlier VERPs, suggesting that as stimulus processing progressed to a later stage, the neural architecture supporting the response may have become less similar between groups (see Fig. 3). The between-group difference on N2 was accounted for by greater superior parietal cortex activity among the healthy compared with the schizophrenia subjects, in a region beyond area MT (Fig. 3).

Relationships between Behavior and Brain Activity

Pearson correlations were used to investigate the relationship between behavioral responses (d-prime) and brain activity measures that differentiated the groups (P1 amplitude and the N2 amplitude difference). The only significant correlations involved the N2 amplitude difference (see Fig. 4). For schizophrenia patients, the N2 amplitude difference was significantly correlated with d-prime, $r(13) = -0.6$, $P = 0.022$, indicating that schizophrenia subjects had better target discrimination ability as the N2 amplitude difference increased. This same correlation was not statistically significant for the healthy subjects, $r(13) = -0.2$, $P = 0.211$, although the 2 groups did not significantly differ on magnitude of this correlation (by Fisher's z).

Discussion

Schizophrenia patients had poor target-detection performance when judging the motion direction of peripheral grating stimuli. This compromised performance is not easily attributable to an early visual pathway processing deficiency (Kim et al. 2006) because 1) we found "enhanced" activity at the time of

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[1] At the request of one reviewer, we also analyzed these data using a Group by Grating Speed by Stimulus Location by Event Type (target, nontarget) repeated-measures ANOVA. The Group by Event Type interaction was significant, $F(1,24) = 6.0$, $P = 0.022$, indicating that healthy subjects had a larger target–nontarget difference than did the schizophrenia patients.

P1 among schizophrenia subjects (see also Alain et al. 1998; Sponheim et al. 2006), and 2) there was no relationship between this enhancement and accuracy. Alternatively, poor behavioral discrimination of target motion direction was strongly associated with a compromised ability to generate a later, target detection–related neural response (at the time of the N2). This pattern may be most consistent with a theory of a late-stage, post-MT deviation in neural functioning being associated with behavioral indications of motion-processing abnormalities in schizophrenia.

An enhanced P1 response in schizophrenia is consistent with some (e.g., Alain et al. 1998; Sponheim et al. 2006; Clementz et al. 2008) but inconsistent with other (e.g., Foxe et al. 2001; Doniger et al. 2002; Butler et al. 2007; Yeap et al. 2008) studies of visual sensory processing in schizophrenia (see Yeap et al. 2008, for a review). The thesis that schizophrenia patients have hyperactivity in transient visual-processing channels figures prominently in multiple conceptualizations of schizophrenia neuropathology (e.g., Green et al. 1994; Kéri et al. 2005). A number of features could account for the considerable between-study differences on visual P1 amplitude deviations in the schizophrenia literature, including stimulus characteristics, task/attentional/response requirements, and VERP quantification scheme. An integrated and comprehensive investigation of these possibilities has the potential to illuminate the neurophysiological nature of schizophrenia-specific visual sensory processing deficits.

In addition to the present manuscript, Alain et al. (1998) and Clementz et al. (2008) used bar-type stimuli requiring little in the way of visual integration for their proper sensory registration. Such stimuli have the desirable property of being optimal for activating simple cells at the initial stages of processing in primary visual cortex (Hubel and Wiesel 1962). These studies found, at the least, a trend toward enhanced activation of the initial visual response among schizophrenia subjects. Studies reviewed by Yeap et al. (2008) that resulted in smaller initial visual responses among schizophrenia subjects used complex stimuli requiring considerable visual integration (see, e.g., Butler et al. 2008, for a discussion of this issue). Whether stimulus complexity could account for different results across studies concerning V1 activation among schizophrenia subjects should be specifically addressed.

Kim et al. (2006) proposed that motion-processing deficits in schizophrenia are caused by impaired bottom-up input to V1 based on a significant association between poor behavioral velocity discrimination performance and visual steady-state response strength to M-pathway-biased stimuli among patients. Although this theory is inconsistent with some findings (Clementz 1996; Kim et al. 1997; Chen et al. 2003), other data are consistent with, but do not prove, an M-pathway deficit in schizophrenia (Butler et al. 2005; Butler and Javitt 2005; Kim et al. 2005, 2006). Kim et al. (2006) obtained their behavioral and brain-activity measures from different tasks, unlike in the present study where brain activity was assessed in relation to behavioral motion–discrimination performance. In addition, Kim et al.'s (2006) steady-state flickering stimuli would maximally activate lower-level visual cortical regions (Di Russo et al. 2007) and minimally activate regions beyond extrastriate cortex (Sunaert et al. 1999; Claeys et al. 2003), so they may have had limited ability to detect the parietal cortex activity seen in the present report. Finally, Kim et al. (2006) only required subjects to passively view the flickering stimuli without requiring a response, so, unlike in the present investigation, their stimuli were not critically relevant for motion analysis performance. It is possible, therefore, that the present investigation and that of Kim et al. (2006) assessed minimally overlapping aspects of the relationship between motion-processing performance and its neural correlates among schizophrenia patients. Comparison of methodological differences between these studies illustrates the need to further investigate relationships between neural activity and motion analysis among schizophrenia subjects.

Another possible explanation for the P1 enhancement is the use of peripherally as opposed to centrally presented moving stimuli (e.g., Kremláček et al. 2006). Peripheral versus central presentation of motion gratings may account for performance differences between the present study and Chen et al. (2003), who reported normal motion direction discrimination among schizophrenia patients. Perhaps consistent with a bottom-up visual-processing dysfunction, schizophrenia patients' brains may try to enhance signal-to-noise ratio for particular stimulus types to overcome a constitutionally higher state of low-frequency "neural noise" in visual cortex (e.g., Clementz et al. 2008). Alternatively, schizophrenia subjects may be unable to maintain centrally located attentional focus, leading to increased responsiveness to peripheral stimuli, even if increased attention to those stimuli results in inappropriate behavioral responses (e.g., Reilly et al. 2008). In the present study, stronger early sensory amplification among schizophrenia patients did not obviously normalize neural activity in superior parietal cortex, a part of the dorsal visual stream associated with target-related brain activation (Ardekani et al. 2002) and identification of task-relevant stimuli (e.g., Simons et al. 2002). Impaired target-detection performance in schizophrenia, therefore, could be caused by inappropriately large signal amplification, as a secondary consequence of bottom-up visual processing dysfunction, resulting in a compromised ability to neurophysiologically differentiate targets from nontargets. This is a possibility that would be critically important to evaluate in subsequent investigations.

Previous work on motion processing in schizophrenia resulted in 2 competing hypotheses (an "early" visual input dysfunction versus a "late" MT-level dysfunction) for why such subjects demonstrate behavioral abnormalities during specific motion analysis tasks. Results of the present study, which directly assessed the relationship between motion processing and neural activity associated with that motion processing, suggest a third possibility. Schizophrenia patients' behavioral motion-processing abnormalities were associated with inability to generate a target-specific neural response in parietal cortex, beyond area MT, and this deficiency in neural processing was specifically and highly correlated with motion analysis behavior. Although consistent with an abnormality that is manifest late in the visual processing stream, these data indicate that schizophrenia patients may have a stimulus classification/target detection difficulty (see also Alain et al. 1998; van der Stelt et al. 2004; Clementz et al. 2008) that is associated with, although not a direct consequence of, problems generating the proper neural response to motion stimuli (see also Braus et al. 2002). The possibility that problems with stimulus classification/target detection may account for motion processing and other behavioral abnormalities in schizophrenia should be more fully investigated in subsequent research.

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