Compromised Late-Stage Motion Processing in Schizophrenia

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Background: Visual motion processing is compromised in schizophrenia, as shown in deficient velocity discrimination. Processing of motion signals comprises progressive stages along the geniculate-striate-extra-striate-cortex pathway. Based on neurophysiologic and brain lesion studies, a velocity discrimination deficit can implicate early-stage motion processing if it is contrast-dependent or late-stage motion processing if it is contrast-independent.

Methods: To determine which stage underlies the deficient velocity discrimination in schizophrenia, we examined the effects of visual contrast on velocity discrimination. We measured velocity discrimination thresholds in schizophrenia patients (n = 34) and normal control subjects (n = 17) at both low and high contrasts, using each subject's contrast detection threshold to equate contrast levels.

Results: Schizophrenia patients showed poor velocity discrimination that improved little with high contrast, whereas normal control subjects showed enhanced velocity discrimination with increased contrast.

Conclusions: The finding that the velocity discrimination deficit in schizophrenia is independent of contrast modulation implicates the later, rather than the earlier, stages of motion processing, which is mediated in the extrastriate cortex.

Key Words: Contrast, psychiatry, velocity discrimination, visual system

Schizophrenia, a complex mental disorder, is associated with many sensory, cognitive, affective, and motor dysfunctions (Bleuler 1950; Kraepelin 1919). Although no accompanying gross organic brain lesions have been documented in this brain disorder, studying those dysfunctions that are associated with schizophrenia and that appear to have identified central nervous system mechanisms is useful in probing the special underlying brain mechanisms. A study of these dysfunctions narrows the field of observation from schizophrenic symptoms to parsable and experimentally manageable psychobiologic behaviors.

One of the dysfunctions implicated in schizophrenia is a disorder of smooth pursuit eye movements (Clementz and Sweeney 1990; Holzman et al 1973; Iacono and Koenig 1983; Levin et al 1986; Levy et al 1993; Sweeney et al 1998; Thaker et al 1998). This smooth pursuit abnormality occurs in about 50% to more than 80% of schizophrenia patients and in about 25%–40% of their clinically unaffected first-degree relatives. Smooth pursuit eye movements are generated and supported by the sensory, cognitive, and motor systems (Keller and Heinen 1991; Lisberger et al 1987; Lisberger and Movshon 1999; Tanaka and Lisberger 2001). One of the components of smooth pursuit eye tracking is the ratio of eye speed to target speed, or gain, which reflects the integrity of the motion processing system. Both motion perception and eye tracking require neural processing of sensory motion signals. To understand the sensory component of eye tracking dysfunction in schizophrenia, an effective (and noninvasive) approach to examine motion processing is to measure the performance of motion perception in patients. Several studies have shown that visual motion processing is indeed compromised in patients with schizophrenia (Chen et al 1999b, 1999c; O’Donnell et al 1996; Stuve et al 1997). For example, velocity discrimination thresholds were shown to be significantly elevated in schizophrenia patients compared with those in nonpatient control subjects (Chen et al 1999b). The exact relation of the motion processing deficits to the pathophysiology of schizophrenia, however, remains to be elucidated. Here, we explore whether the early or the late stage of motion processing is affected in schizophrenia.

Information required for velocity discrimination is processed in progressive stages of the motion pathway (Chen et al 1998; McKee et al 1986; Merigan et al 1991; Pasternak et al 1989; Pasternak and Merigan 1994; Plant and Nakayama 1993). Motion processing begins with neural activity in subcortical areas, such as the retina and the lateral geniculate nucleus of the thalamus (LGN, which includes two layers of magnocellular cells), and in the striate cortex (primary visual cortex—V1). The visual motion signals are then transmitted to the middle temporal area (MT), an extrastriate cortical area, for motion-specific processing. Other cortical areas, including the medial superior temporal area (MST), ventral intraparietal area (VIP), and the fundus superior temporal area (FST), receive projections from MT for more global motion processing and motion-related cognitive and motor processing.

There are several differences in the functional properties of the various stages of motion processing. Neural activities in the earlier stages depend on the contrast of visual motion stimuli; at the earlier stage (e.g., subcortical areas and the striate cortex), neural responses to motion signals increase monotonically with contrast over a considerable range (Sclar et al 1990), in addition to the motion aspects of a visual target (e.g., direction or velocity). After damage to the magnocellular pathway at LGN (early-stage motion processing), velocity discrimination in monkeys is impaired for visual targets at low, but not at high, contrasts (Merigan et al 1991), indicating that the deficient motion processing at the level of LGN is contrast-dependent. At the later stage (e.g., the extra-striate cortex), however, neural responses are largely independent of contrast once the stimulus contrast exceeds a low threshold level (e.g., Sclar et al 1990). After damage to area MT (the late stage), velocity discrimination in monkeys is impaired regardless of the contrast level of a moving target (Pasternak and Merigan 1994), indicating that deficient motion processing at this later stage is not contrast-dependent.

The difference in contrast dependency between the early and late stages of motion processing can be used experimentally to dissociate the functional integrity of the two stages (i.e., subcortical and striate cortical vs. extra-striate cortical mechanisms).
Methods and Materials

Subjects

Thirty-four schizophrenia patients participated in this study. The patients had been hospitalized about 1 year earlier and, at the time they were tested, all were outpatients in various degrees of remission from their psychosis. All patients met DSM-IV criteria for schizophrenia or schizoaffective disorder. Consensus diagnoses were made independently by experienced clinicians based on a review of a standardized interview (Structured Clinical Interview for the DSM-IV-SCID-P) conducted by trained interviewers, and an evaluation of all available hospital records. All but two patients were receiving antipsychotic medication (mean daily chlorpromazine dose equivalent: 651 mg [SD = 44.7]). Among those patients receiving antipsychotic medication, all but two patients were receiving antipsychotic medication that included both first- and second-generation antipsychotic drugs, and the rest took only second-generation antipsychotic drugs. The average score of the Brief Psychiatric Rating Scale of the patients was 44.7 (SD = 14.3).

Seventeen normal control subjects participated in this study. None met DSM-IV criteria for a psychotic condition (lifetime) according to the Structured Clinical Interview for DSM-IV or for schizotypal personality disorder or schizoid or paranoid personality disorder according to the Structured Interview for Schizotypal Symptoms (Kendler 1989). None of the normal control subjects had a family history of psychosis. The following exclusion criteria applied to all participants: 1) lack of English fluency, 2) diagnosis of organic brain disease, 3) history of substance abuse or dependence during the past 2 years, 4) presence of tardive dyskinesia, 5) use of alcohol or other recreational drugs within 2 weeks before study participation, or 6) estimated verbal IQ less than 70.

Specifically, by controlling the contrast level of a moving target during a velocity discrimination task, we can determine the stage of motion processing that is implicated in schizophrenia. If velocity discrimination of schizophrenia patients is impaired at low contrasts but not at high contrasts, we can conclude that motion processing at the earlier stages is compromised (Merigan et al 1991). If, however, increasing target contrast fails to normalize velocity discrimination of the patients, motion processing at the later stages is compromised (Pasternak and Merigan 1994). The experiments presented here were based on this rationale.

Stimuli

The visual target used for velocity discrimination was a patch of moving gratings (Figure 1). The stimuli were generated on a Macintosh computer, used with a luminance attenuator that allows fine gradations in contrast. The display had a mean luminance of 35 cd/m². The gratings patch was oriented vertically and was shown through a circular window with 19° of visual angle. Viewing was binocular at a distance of .5 m. Base velocity of the stimuli was 10°/sec. The spatial frequency and the temporal frequency of the gratings were .5 cycles/deg and 5 Hz, respectively. Each presentation of the gratings lasted 300 msec. A small central circle was provided for fixation.

We used two levels of contrast to elicit effects of contrast modulation on velocity discrimination in this study. As a primary controlling variable, contrasts of the gratings were set at 4 times (low contrast) and 20 times (high contrast) the detection thresholds. The thresholds were measured individually in all subjects (discussed later). The selection of the two levels of contrast (4X and 20X the detection threshold) was based on our pilot data in a small number of normal subjects who showed decreased velocity discrimination thresholds (improved performance) when target contrast was increased from 4 to 20 times contrast the detection threshold. The data from the sample of normal control subjects in this study (n = 17) also showed a significant decrease of velocity discrimination thresholds with the same increase in target contrast (see Results).

Procedure

Contrast Detection

Contrast detection thresholds were determined using a psychophysical procedure, a two-alternative, forced-choice method in conjunction with a staircase. The procedure started with the initial contrast of the gratings set at 1.5%, an adequate level to detect the presence of the moving target. The task was to indicate which of two intervals in a trial contained the target (one interval contained the moving gratings, and other contained only a blank field of the same mean luminance). The amount of contrast was decreased by 30% of the current level after three consecutive correct responses or increased by 30% whenever a single erroneous response occurred, a procedure that identified the
79% correct point on each subject’s psychometric function, on the basis of 12 up–down reversals (Levitt 1972). By initiating the visual tasks at an easy level (high contrast), this staircase procedure is particularly useful in studying psychiatric populations because it gives subjects confidence, minimizes the subjective criterion bias that may be associated with clinical symptoms such as interpersonal withdrawal, and identifies a threshold point in a relatively short time, which is always advantageous when testing patients with schizophrenia.

**Velocity Discrimination.** After contrast detection thresholds were obtained, contrast levels for velocity discrimination, the principal task, were set individually for each subject. For the low-contrast condition, the contrast level of the moving gratings was set at four times the contrast detection thresholds of each individual. For the high-contrast condition, the contrast level of the moving gratings was set at 20 times the contrast detection thresholds. In each trial, a pair of moving gratings was presented sequentially for velocity comparison (interstimulus interval: 500 msec). Of the two gratings, one moved at a base velocity of $10^\circ$/sec, and the other moved at a faster velocity. The task for each subject was to indicate which of the two sequentially presented targets moved faster. The presentation order of the two types of gratings was randomized across trials. The initial velocity for the faster grating was set at $20^\circ$/sec, that is, 100% velocity difference: $(20-10)/10 = 1$, or 100%. The same psychophysical procedure that was used to determine the contrast detection thresholds was used to determine velocity increment thresholds (the just-noticeable difference in velocity, $\Delta V$), except that now the velocity difference between two moving targets, rather than the amount of contrast, became the variable to be adjusted. The velocity difference between the two moving targets was decreased by 30% of the current level after three consecutive correct responses or increased by 30% whenever a single erroneous response was made. Twelve up–down reversals in the staircase determined an increment threshold, from which the Weber fraction ($\Delta V/V$) was computed.

Before formal data collection, brief practice sessions were provided. These sessions were not meant to train subjects to achieve perfect performance but were sufficient to allow them to understand the procedure of the tasks. Completion of all procedures required about 1 hour. Rest periods, as needed, were provided within the testing session.

**Results**

**Velocity Discrimination at High Contrast**

Schizophrenia patients showed significantly higher mean Weber thresholds for velocity discrimination than normal control subjects when the contrast of moving targets was set at 20 times the contrast detection threshold (Figure 2). Statistical analysis

![Figure 1. Schematic stimulus configuration for velocity discrimination. The faster of the two comparison gratings may appear either in the first or in the second time interval in a trial. The length of arrows signifies velocity of the moving gratings. The actual contrast levels of gratings used in this study were set experimentally for each individual subject.](#)

![Figure 2. Scatter plot of velocity discrimination thresholds at the high-contrast level. Each point in the plot represents a Weber threshold from an individual subject who compared the velocities of two moving gratings. Filled circles represent the schizophrenia patients and open circles the normal control subjects. The thresholds of the patient group were significantly higher than those of the control group ($t_{(50,1)} = 3.17, p < .005$).](#)
yields highly significant difference between the thresholds of the two groups ($t_{(50,1)} = 3.17, p < .005$).

**Velocity Discrimination at Low Contrast**

Figure 3 shows the Weber thresholds of velocity discrimination when the contrast was set at 4 times the contrast detection threshold. The velocity discrimination thresholds of some, but not all, schizophrenia patients were higher than those of normal control subjects. Statistical analysis yields a significant difference between the two groups ($t_{(50,1)} = 2.24, p < .05$).

Two characteristics emerge when we compare the velocity discrimination thresholds obtained at low and at high contrasts. First, velocity discrimination between the two contrast levels was significantly correlated among the schizophrenia patients (see Figure 4); the Pearson correlation coefficient was $.70\ (p < .001)$.

That is to say, if a patient performed poorly at one contrast level, he or she was likely to perform poorly at the other contrast level. In the normal control group, the correlation between the thresholds at the low and high contrasts was .33 and not statistically significant ($p > .1$). Second, velocity discrimination in the schizophrenia patients changes little from the low- to the high-contrast conditions, whereas velocity discrimination in the normal control subjects was considerably better for the same increase in contrast. As a group, the schizophrenia patients showed a much smaller and statistically insignificant velocity discrimination threshold decrease (from $.27\ [SD = .16]$ to $.25\ [SD = .13]$) or 7% decrease; $t_{(66,1)} = .66, p > .1$) than did normal control subjects, who showed a statistically significant threshold decrease (.18 $[SD = .06]$ to .13 $[SD = .06]$ or 28% decrease; $t_{(52,1)} = 2.6, p < .05$), as the stimulus contrast was changed from low to high. When a change index of velocity discrimination threshold ($([\text{Threshold}_\text{low contrast} - \text{Threshold}_\text{high contrast}] / \text{Threshold}_\text{low contrast})$) was computed for individual subjects, the schizophrenia group and the normal control group showed slightly negative (worse) and substantially positive (better) magnitudes, respectively (Figure 5). The combination of the very small decrease in mean threshold (better) and the very small negative magnitude in change index (worse) in the patient group indicates that change in their velocity discrimination thresholds with stimulus contrast is negligible. Taken together, both characteristics of the results illustrate that contrast modulation had little effect on velocity discrimination in the schizophrenia patients.

We also used analysis of variance (ANOVA) to compare the results of two subject groups (schizophrenia patients and normal control subjects) at two levels of stimulus contrasts (low and high). The results are as follows: 1) there was a significant effect of groups ($F = 16.530, p < .001$), 2) there was no significant effect of high versus low contrast ($F = .038, p = .16$), and 3) there was no significant interaction between subject groups with contrast level ($F = .730, p = .39$). The ANOVA is consistent with the previous t test analysis and confirms the conclusion that the velocity discrimination deficit in patients is independent of contrast.
low contrast and .16 and .11 at the high-contrast level. The average velocity discrimination threshold change in these two patients is a 26% decrease from the low to the high contrast, which is similar to the average 28% decrease in the entire patient group. The CPZ equivalent dose of antipsychotic drugs and the velocity discrimination thresholds in patients were not significantly correlated. In the patient group, age, year of education, and SES were not correlated with velocity discrimination thresholds; verbal IQ and velocity discrimination thresholds were moderately correlated ($r_{\text{high contrast}} = .38$ and $r_{\text{low contrast}} = .57, p < .05$). An analysis of covariance by the general linear model (GLM) procedure of the SAS software program indicated that the group differences in velocity discrimination thresholds remain after using the IQ scores as the covariate.

### Discussion

Velocity discrimination thresholds at both low- and high-contrast levels were significantly elevated in schizophrenia patients compared with normal control subjects. The independence of the velocity discrimination deficit from contrast provides evidence relevant to the specific stages of motion processing implicated in schizophrenia.

Although contrast is vital to many attributes of vision, its roles differ in various stages of visual processing. In the motion pathway, neural responses to low-contrast moving gratings are different from those to high-contrast moving gratings, but the difference occurs primarily at the early stages (such as in subcortical areas) of motion processing. At the late stages (such as in extra-striate cortical areas), neural responses are relatively insensitive to the changes in contrast of visual targets but are sensitive to the movement aspects of visual target, such as direction or velocity of movement (Sclar et al. 1990). In light of the different roles that contrast plays in the early and the late-stage motion processing, the performance of velocity discrimination, when measured as a function of contrast, yields information about the functional integrity of each motion processing stage. Our results showed that the schizophrenia patients’ performance on the velocity discrimination task was impaired at both low and high contrasts. Moreover, the extent of this performance impairment did not depend on contrast levels. Thus, the independence of velocity discrimination deficits from contrast modulation is consistent with the notion that the later, rather than the earlier, stage motion processing in the visual pathway is implicated in schizophrenia.

Several issues merit further discussion. The first concerns whether contrast detection itself is normal in schizophrenia. Contrast detection of schizophrenia patients has been variously reported to be worse than that of normal control subjects (Slaghuis 1998), equivalent to that of normal control subjects (Chen et al. 1999c, 2000, 2003a), and better than that of normal control subjects (Keri et al. 2000). It is possible that when judging the velocities of targets, stimuli of various contrasts may have differential effects on the performance of individuals who differ in contrast detection. The design of our paradigm, however, precludes this possibility. In our study, the low- and high-contrast levels were defined by the multiples of contrast detection thresholds determined for each individual, not by a nominal value (say, 10%) of a stimulus contrast level selected arbitrarily. Because each subject’s own contrast detection threshold is used as baseline, the definition and measurement applied in this study takes into account the variation in contrast detection among individuals, schizophrenia patients and normal control subjects. Thus, the velocity discrimination deficit seen at the low and the
high contrasts cannot be attributed to individual variations in contrast detection.

The second issue concerns the schizophrenia patients' deficient performance in velocity discrimination at low contrasts. That is, whether the group difference in velocity discrimination at the low-contrast level is due to the contrast detection factor. This would be a significant issue if the study had set a standard contrast level for all of the subjects, as is done in many conventional studies. In this study, however, the low- and the high-contrast levels of the stimuli used in velocity discrimination were tailored to the individual subjects' previously established contrast detection thresholds. This procedure takes into account experimentally any differences in contrast detection ability among subjects. Thus, the group difference found in velocity discrimination at low contrast level cannot be attributed to a difference in contrast detection but can properly be attributed to velocity discrimination. In a previous study (Chen et al. 1999c), we assessed velocity discrimination using contrast as a dependent variable and found that schizophrenia patients needed much higher contrasts to discriminate a small difference in velocity (difficult), but not to discriminate a large difference (easy). The high-contrast reliance of schizophrenia patients in velocity discrimination, found in the previous study, may at first seem paradoxical in light of the contrast-independence of the velocity discrimination deficit, found in the study presented here. When one compares the paradigms and the results between the two studies, however, it becomes clear that schizophrenia patients showed a genuine velocity discrimination deficit that is not based on contrast. In the previous study, the velocity difference in the difficult condition was set at 20%, a condition that significantly exceeds normal Weber thresholds for velocity discrimination, which are about 10%. It is thus possible that when velocity discrimination is made easy by presenting a suprathreshold level (20% velocity difference), the less precise and contrast-dependent early stage of motion processing may be responsible for the velocity discrimination in schizophrenia patients, especially when the late stage of motion processing is defective. In another study, Chen et al. (1999b) randomized the contrast levels of the moving targets used for velocity discrimination and found little change in the thresholds at intermediate base velocities, the range in which the velocity discrimination deficit occurred in schizophrenia patients. The negligible effect of contrast randomization on velocity discrimination is consistent with the results of this study—namely, that velocity discrimination deficit in schizophrenia does not depend on contrast.

The third issue concerns whether the result that increase of contrast did not normalize velocity discrimination in schizophrenia can be taken as evidence that only the late-stage motion processing is implicated. One could argue that an abnormality in the early-stage motion processing also contributes to the deficient velocity discrimination in patients. Although this argument may be raised on theoretical grounds, the physiologic data obtained from studies using monkeys suggest otherwise. As described in the Introduction, after an experimental lesion was made to LGN (early in the motion pathway) in monkeys, a velocity discrimination deficit is induced, but this deficit can be compensated for by increase of the target contrast (Merigan et al. 1991). This signature result of impaired early-stage of motion processing was not present in schizophrenia patients; however, instead, they showed persistent velocity discrimination deficits in this study despite an increase of target contrast by five times. Thus, although we cannot completely rule out any effect of abnormal input from earlier stages of the visual pathway, the pattern of the velocity discrimination deficit shown in schizophrenia patients is more consistent with compromised motion processing at later stages when increase of target contrast has little effect on neural responses (e.g., Sclar et al. 1990). That the patients failed to improve their deficient velocity discrimination from increases in stimulus contrast actually reflects a signature property of damaged MT, in which the velocity discrimination deficit of monkeys cannot be improved by increase of stimulus contrast (Pasternak and Merigan 1994); this is not, however, a property of damaged LGN, in which the velocity discrimination deficit can be improved by increases in stimulus contrast (Merigan et al. 1991).

The fourth issue concerns the complexity of the late-stage of motion processing. It has been recognized that neural processing in the late stage of the motion pathway deals with complex properties of visual signals (such as the independence of spatial frequency in processing the motion of complex patterns). The transformation from processing basic features, such as contrast, of motion stimuli at the earlier stage to processing complex patterns of motion stimuli at the later stage also suggests that the late stage of motion processing is contrast-independent. In our study, the contrast of the stimuli was modulated, but the spatial frequency of the stimuli was kept constant at .5 cycles/degree. Spatial frequency and contrast, both aspects of visual signals, are processed differently within the motion sensitive areas in the superior temporal sulcus, such as MT. It is the relative independence of motion from contrast modulation in the late but not at the early stage that provides us with the opportunity to study the functional properties of the motion system in schizophrenia. We were interested in seeing whether the difference in contrast made a difference in velocity discrimination thresholds after its detection levels were equated across subjects. Thus, in our study, the results are based not on spatial frequency but on whether the contrast level affects motion discrimination. The complexity of neural processing in the late stage of the motion pathway does raise a related issue—whether the deficit in the late-stage motion processing in schizophrenia is present in nonmotion domains that deal with complex patterns. It has been reported that visual processing of complex patterns in nonmotion domains, such as in perceptual closure, is affected in schizophrenia patients (Doniger et al. 2001). It remains to be examined whether a common or related neural mechanism in the late stage of the visual system is responsible for the behavioral deficits of schizophrenia patients in the motion and nonmotion domains.

The fifth issue concerns whether working memory may influence velocity discrimination. Indeed, spatial working memory is impaired in schizophrenia patients (Park and Holzman 1992). For the following reasons, however, we believe that the influence of working memory has limited, if any, influence on the velocity discrimination measures and instead reflects primarily the functional properties of the neural processing of motion signals within the visual system. First, the time interval between two comparison targets in our velocity discrimination paradigm was very brief (.5 sec), compared with those used in conventional working memory studies, which are usually set at 5–20 sec. Neural processing in the visual system (e.g., striate cortex) is capable of retaining the information for this short period of time (i.e., an order of 1 sec) (Super et al. 2001). Second, using two similar time-interval velocity discrimination paradigms, animal studies showed impaired velocity discrimination performance when the middle temporal area in the visual cortex is damaged (Pasternak and Merigan 1994). Third, in a separate study, we employed a paradigm that required subjects to judge a global
motion stimulus each time a random dot pattern was presented, that is, one with no memory element. Patients showed impaired performance in that motion task even when no working memory was required (Chen et al 2003b).

The results from this study point to the neural processes mediated in the extra striate cortex as the brain mechanisms responsible for motion processing deficits in schizophrenia. Only recently has this brain region been seriously considered in the pathophysiology of schizophrenia. In this part of the motion pathway, several other aspects of motion perception and motion-related activities are involved, including global motion, spatial and temporal interaction of motion information, and eye movements to moving targets. In a recent study (Chen et al 2003b), schizophrenia patients showed poor performance in detecting the direction of global, but not local, motion, a result that also implicates motion processing at later stages. It is noteworthy that the late-stage of motion processing supports both smooth pursuit eye movements and saccades to visual moving targets (Newsmoe et al 1988), yet only smooth pursuit is abnormal in schizophrenia (Chen et al 1999a; Clementz and McDowell 1994; Friedman et al 1995; Holzman et al 1973; Levin et al 1988; Sweeney et al 1998; Thaker et al 1998). This complex pattern of behavioral responses to motion signals suggests that an overall loss of neural processing in schizophrenia is unlikely because only some, but not all, aspects of the associated neural mechanisms are dysfunctional. Instead, the functioning of the neural mechanisms in the extra striate cortex in schizophrenia may depend on the way in which motion information is used. One case of the functional failures in the late-stage of motion processing may occur when continuous spatial and temporal integration of motion information is required, such as in smooth pursuit and in velocity processing, but not when discrete spatial information is sufficient, such as in saccadic eye movements. Schizophrenia patients may share functional impairments seen in other primates with organic damage to the extra-striate cortex, but only for the part in which continuous processing of motion information is necessary. This phase of neural processing may require intensive interaction among the neural units involved at different points in space and time and may be selectively lost in schizophrenia, in which case the associated cognitive and motor processing in the later stages will receive noncontinuous or even erroneous spatial and temporal signals from the motion processing system. Whether and how the deficient processing at the later stages of the motion pathway is related to cognitive dysfunctions in schizophrenia patients remains an open question, and thus an important spur for empirical studies. In this respect, studies of contextual processing of motion information (e.g., Albright and Stoner 1995), which includes short- and long-range interactions between motion signals and their spatial and temporal neighbors, should yield new insights into the special brain mechanisms that are linked to the pathophysiology of schizophrenia.

This research was supported in part by National Institutes of Health Grant Nos. MH 61824, 31154, 31340, 46987, 01020; a National Alliance for Research on Schizophrenia and Depression Young Investigator Award; a Rapaport Mental Health Scholar Award; and grants from the William T. Milton Foundation of Harvard University and the Roy A. Hunt Foundation.

We thank Drs. Steven Matthesy, Ken Nakayama, and Charles Stromeyer III and Ms. Cinnamon Bidwell, for their help in various aspects of this study. We also thank Dr. Laurie Terepsulsky and Ms. Anne Gibbs for recruiting subjects.


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