Abnormal Visual Motion Processing in Schizophrenia: A Review of Research Progress

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Motion processing represents a perceptual domain in which dynamic visual information is encoded to support the perception of movement. Research over the last decade has found a variety of abnormalities in the processing of motion information in schizophrenia. The abnormalities span from discrimination of basic motion features (such as speed) to integration of spatially distributed motion signals (such as coherent motion). Motion processing involves visual signals across space and time and thus presents a special opportunity to examine how spatial and temporal information is integrated in the visual system. This article surveys the behavioral and neuroimaging studies that probe into the spatial integration of motion information in schizophrenia. An emerging theme from these studies points to an imbalanced regulation of spatial interaction processes as a potential mechanism mediating different levels of abnormal motion processing in schizophrenia. The synthesis of these mechanism-driven studies suggests that further investigation of the neural basis and functional consequences of this abnormal motion processing are needed in order to render a basic biomarker for assessment and intervention of cognitive dysfunction in this mental disorder.

Key words: schizophrenic/movement perception/visual integration/speed/coherent motion

Introduction

The study of motion processing in schizophrenia was initially inspired by the search for the perceptual mechanisms responsible for the eye-tracking dysfunction associated with the mental disorder.1–6 Within the last decade, this line of study has been extended as an avenue of research for understanding how dynamic visual information is processed in schizophrenia and how the brain mechanisms underlying this perceptual domain are altered. This review briefly surveys the findings of psychological and neuroimaging studies on motion processing and then focuses on those studies that probe into specific underlying brain mechanisms. This review also offers a description of the tentative relationship between basic motion processing and other relevant visual cognitive and visuomotor functions in this mental disorder. The goal here is to provide a summary of the literature available in this field up to this stage of the research progress and to highlight the promise of this dynamic visual domain to both uncover the involved brain mechanisms of and guide the assessment and intervention for this mental disorder.

From Feature Detection to Spatiotemporal Integration

To support movement perception, the visual system develops a special capacity for processing motion information.7,8 This capacity comprises encoding of motion features (such as speed and direction) and integration of distributed spatiotemporal signals (such as coherent motion). Along with the classification of these features, a series of studies on basic motion processing has been conducted in schizophrenia (table 1).

Speed is a salient visual feature that is uniquely encoded in the motion processing system. One empirical paradigm for studying the visual processing of speed signals is speed discrimination, in which perceptual judgments about 2 moving targets are rendered based upon respective speeds of the targets. Deficient speed discrimination has been found in schizophrenia.10,11,19,20,23 Several aspects of the findings converge to suggest that the perceptual deficit is genuine to the motion processing, rather than to non-motion visual processing, domain. First, the speed discrimination deficit is present regardless of the type of moving targets used, whether they are gratings,10,23 a singular dot,20 or random dot patterns19; suggesting that the perceptual problem is not related to the spatial contents (or forms) of the visual targets involved. Second, the deficit is present not only in patients but also in their biological relatives,11 suggesting that the
perceptual problem is not directly associated with clinical manifestations in patients. Third, the deficit is mostly prominent in intermediate speeds (eg 10 degrees/s). Unlike slow speeds with which position cues are dominant or fast speeds with which temporal frequency cues are dominant, intermediate speeds are the conditions where speed cues dominate perceptual judgments of movements.24,25 Thus, the selective deficit in intermediate speeds indicates a perceptual problem specific to the motion processing domain.

Integration of visual information across space and time is an inherent component of motion processing. This particular visual domain thus provides an opportunity to study the brain mechanisms responsible for integrating spatiotemporal signals on basic perceptual levels. Coherent motion, which represents such a signal, consists of 2 components—one generated from a group of spatially distributed dots moving coherently along one direction (signal) and the other generated from a different group of superimposed dots moving in random directions (noise). Several studies have examined the detection of coherent motion in schizophrenia.9,13,21 Patients generally showed lower accuracies when performing this task and required higher levels of motion signal strength in order to achieve similar levels of accuracies (elevated thresholds) as compared with controls. Given that combining spatially distributed signals is a prerequisite for the performance of the task, this result indicates that spatial integration of the motion signals is deficient in this mental disorder. On a separate front, a preliminary study showed that first-degree relatives of schizophrenia patients and patients with bipolar disorder had normal performance on this task, suggesting a specificity of the motion integration deficit in clinical schizophrenia.15 This latter result, however, remains to be confirmed in independent and larger populations.

### Table 1. A Summary of Psychophysical Studies on Basic Motion Processing in Schizophrenia

<table>
<thead>
<tr>
<th>Article</th>
<th>Other Subjects</th>
<th>Paradigms</th>
<th>Key Results (in SZ, Unless Otherwise Noted)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N = 19</td>
<td>Healthy controls (HC) (n = 19)</td>
<td>Detection of coherent motion</td>
<td>Elevated detection thresholds</td>
</tr>
<tr>
<td>N = 15</td>
<td>HC (n = 18)</td>
<td>Contrast-based speed discrimination (gratings)</td>
<td>Elevated contrast thresholds</td>
</tr>
<tr>
<td>N = 20</td>
<td>Relatives (n = 24) and HC (n = 20)</td>
<td>Speed discrimination (gratings)</td>
<td>Elevated discrimination thresholds in mid-speeds (such as 10 degrees/s) in SZ and relatives</td>
</tr>
<tr>
<td>N = 13</td>
<td>HC (n = 14)</td>
<td>Detection of coherent motion</td>
<td>Reduced sensitivities</td>
</tr>
<tr>
<td>N = 23</td>
<td>HC (n = 26)</td>
<td>Direction discrimination (local) and detection of coherent motion (global)</td>
<td>Intact detection thresholds for local motion and elevated detection thresholds for global motion</td>
</tr>
<tr>
<td>N = 34</td>
<td>HC (n = 17)</td>
<td>Speed discrimination at low and high contrasts</td>
<td>Elevated discrimination thresholds for both contrasts</td>
</tr>
<tr>
<td>N = 29</td>
<td>Relatives (n = 20), bipolar patients (BP) (n = 19), and HC (n = 33)</td>
<td>Detection of coherent motion</td>
<td>Elevated detection threshold in SZ but not in relatives or BP</td>
</tr>
<tr>
<td>N = 12</td>
<td>HC (n = 12)</td>
<td>Contrast-based detection of direction of motion</td>
<td>Reduced sensitivity in SZ with negative symptoms, but not in SZ with positive symptoms</td>
</tr>
<tr>
<td>N = 44</td>
<td>HC (n = 40)</td>
<td>Speed discrimination</td>
<td>Elevated discrimination thresholds, independent of age</td>
</tr>
<tr>
<td>N = 25</td>
<td>BP (n = 16) and HC (n = 25)</td>
<td>Speed discrimination (gratings)</td>
<td>Elevated discrimination thresholds for mid-speeds in SZ and for fast speeds in BP</td>
</tr>
<tr>
<td>N = 14</td>
<td>HC (n = 16)</td>
<td>Speed discrimination (random dots)</td>
<td>Elevated discrimination thresholds</td>
</tr>
<tr>
<td>N = 18</td>
<td>HC (n = 17)</td>
<td>Speed discrimination (a singular dot)</td>
<td>Elevated discrimination thresholds</td>
</tr>
<tr>
<td>N = 35</td>
<td>HC (n = 35)</td>
<td>Detection of coherent motion</td>
<td>Elevated detection thresholds at 6, but not 12 or 24, degrees/s</td>
</tr>
<tr>
<td>N = 34</td>
<td>HC (n = 34)</td>
<td>Detection of apparent motion</td>
<td>No group difference, yet SZ’s task performance related to clinical measures</td>
</tr>
<tr>
<td>N = 38</td>
<td>HC (n = 33)</td>
<td>Speed discrimination with 150- and 300-ms stimulus intervals</td>
<td>Elevated discrimination thresholds for comparison stimuli with 300 but not with 150-ms intervals</td>
</tr>
</tbody>
</table>

Note: BP, bipolar patients; HC, healthy controls.
The important issue of exactly how the spatial and temporal contents of visual signals modulate motion processing has yet to be systematically examined in schizophrenia. The stimulus properties, including size and display time, play a direct role in determining the extent of spatiotemporal integration during motion processing. A major reason for studying this modulation effect is that despite the fact that motion processing itself requires integration in spatial and temporal domains, other factors such as generalized deficit or medication treatment may also have potential influences on perceptual performance of patients. If the non-integration factors were primarily responsible for the motion processing deficits, modulation of spatial and temporal contents would have little effects. Alternatively, if the integration factors were responsible for the motion processing deficits, distributing motion signals over greater ranges of space and time would lead to more severe impairments in perceptual performance. Thus, the examination of motion processing as a function of spatial and temporal contents will provide a more deterministic approach to validating whether the spatiotemporal integration mechanism is indeed responsible for the motion processing deficits in schizophrenia.

In this regard, the center-surround interaction paradigm, which provides an evaluation of the modulation of the neural or perceptual responses to a central visual target by surrounding contexts, is particularly useful. A theoretical advantage of the paradigm is its direct employment of the neural computations for spatial interaction in circuitry levels.²⁶,²⁷ This paradigm is also advantageous methodologically for schizophrenia research in that it measures the response differences between the conditions where a particular surround is present or where it is absent (the latter serving as baseline). As such, the net measurements of response difference remove the baseline-associated factors including the generalized deficit. This paradigm is discussed in detail in a later section of this article.

Potential Neural Substrates

Motion processing is normally mediated in the posterior cortex (striate and extrastriate regions).⁸ In particular, middle temporal area (MT) in the extrastriate cortex responds selectively to motion signals and is accordingly identified as a center for visual motion processing.²⁸–³¹

Neuroimaging approaches have recently been applied to explore the neural substrates of motion processing in schizophrenia. In a functional magnetic resonance imaging (fMRI) study, schizophrenia patients showed decreased activation in MT and increased activation in the inferior convexity of the prefrontal cortex during detection of coherent motion and speed discrimination, but not during contrast discrimination.³² This pattern of results indicates that abnormal processing of motion information in schizophrenia involves a deficient sensory system, which yields weakened signals, and an abnormally activated cognitive system, which may tend to compensate for the sensory deficiency. It appears that the neural substrate of motion processing in schizophrenia includes a network of cortical systems beyond conventional perceptual processing. This intriguing result should be studied further in order to more fully understand its implications on the pathophysiology of schizophrenia.

One limitation of the fMRI paradigm is the relatively low resolution in the measurement of temporal dynamics, which is an evidently important aspect of motion processing. A recent study using an electroencephalography paradigm is illuminating in this regard; the performance of a motion perception task by schizophrenia patients evoked enhanced early-phase neural activity and diminished late-phase neural activity over the parietal cortex.³³ The abnormal late phase, but not the abnormal early phase, neural activity was associated with the performance levels in patients. This result indicates that an abnormality in the late stages of motion processing is associated with schizophrenia, which is consistent with the results of a previous psychophysical study.¹⁴

Using eye-tracking paradigms, several studies have found that fMRI responses in a network of cortical areas were altered in schizophrenia. One study showed that MT activity during eye tracking of a moving target was reduced in schizophrenia patients.³⁴ Other studies showed increased activity in posterior hippocampi and the right fusiform gyrus,³⁵ or in the medial occipitotemporal cortex,³⁶ during patients’ performance of an eye-tracking task. These diverse magnitudes of cortical activation in patients are intriguing but not entirely surprising, given that eye tracking typically involves 2 phases—the initial open loop and the sustained closed loop—during which different types of information are utilized. During the open loop phase, eye tracking is primarily driven by perceptual motion signals, which are processed in the posterior visual cortex. During the closed loop phase, however, perceptual motion signals are no longer available and eye tracking is primarily driven by non-perceptual signals such as extraretinal signals that are processed in other cortical areas, including the medial superior temporal area. Future studies of motion processing in schizophrenia should combine neuroimaging with psychometrically defined visual stimulation in order to dissect perceptual from non-perceptual inputs and maximize the interpretative powers of data.

The results of neuroimaging studies highlight that motion processing in schizophrenia involves multiple cortical systems. Additional studies are urged in order to characterize the relationship among these potential neural substrates and to uncover specific underlying brain mechanisms.
Imbalanced Regulation of Spatial Interaction Across Motion Processing Levels

The psychophysical and neuroimaging studies aforementioned are helpful in the identification of specific motion processing domains and associated brain systems that are implicated in schizophrenia. The exact underlying brain mechanisms, however, have yet to be pinpointed and characterized in the neural computational levels.

Center-surround interaction is a ubiquitous neural mechanism mediating the processing of visual information within spatial contexts. As a hallmark of this mechanism, the response of a neural unit is not solely determined by the visual stimuli located within its classic receptive field. The stimuli falling outside the receptive field can also modulate the response due to the lateral connections between this unit and neighboring neural units. Note that the stimuli outside the receptive field would have no effect when presented alone. In motion processing, this spatial interaction mechanism is characterized by moving surround’s suppression and facilitation of the neural response to center motion. Suppression occurs when center and surround contain the same direction of motion, whereas facilitation occurs when they contain opposite directions of motion.

In schizophrenia, 2 studies have investigated this mechanism in the context of motion processing. Using moving gratings (a visual target primarily stimulating local motion processing), one study found significantly reduced surround suppression of motion in schizophrenia. This result suggests that inhibitory regulation in the local stage of motion processing is weakened in this mental disorder. Using a random dot pattern (a visual target primarily stimulating global motion processing), the other study found significantly increased surround suppression of motion in schizophrenia. This result suggests a heightened inhibitory regulation in the global stage of motion processing. The drastic change in regulating different types of motion signals suggests that neither a deficient nor excessive spatial interaction mechanism alone can explain the abnormal motion processing in schizophrenia. Instead, these results point to an imbalanced regulation of spatial interaction across the motion processing stages as a fundamental mechanism mediating these abnormalities. This mechanism, unlike straight deficiency mechanisms, highlights that non-equilibriums of primary (excitatory) and regulatory (inhibitory) responses shift their polarity from one motion processing stage to another due to the change in inhibitory modulation. While still requiring further validation, the imbalance of motion regulatory mechanisms at the perceptual level provides empirical support for the general theory of abnormal cognitive coordination in schizophrenia. An unanswered question, however, is whether this imbalanced regulatory mechanism for basic motion processing is also present in other visual and cognitive processes and to what extent it contributes to various disorganized cognitive and social behaviors found in patients.

Tentative Relationships Between Motion Processing and Eye Tracking, Cognitive Functioning, or Early Visual Processing

Motion processing provides critical perceptual signals for survival and success in life. In schizophrenia, abnormalities have been found in many motion-related perceptual and cognitive functions including trajectory discrimination, illusory motion, and recognition of biological motion. A few studies have attempted to explore the relationships between basic motion processing and various motion-relevant functional capacities in this mental disorder.

In schizophrenia patients, associations have been reported between eye-tracking dysfunction, an initial motivation behind motion processing studies, and speed discrimination or detection of coherent motion. Motion processing provides perceptual inputs for the generation of eye-tracking response to moving targets. Thus, the above associations in schizophrenia suggest that deficient motion processing contributes to eye-tracking dysfunction. A recent study asked whether the eye-tracking dysfunction contributes to deficient motion processing in this mental disorder. By recording eye movement simultaneously, this study found that compared with controls, patients had poor speed discrimination for the stimuli of a long display time (300 ms) but not for those of a short display time (150 ms) and suggested that the motion perception deficit may be a consequence of the eye movements generated by the moving stimuli with a long display time. This notion is both interesting and tenable. However, if eye movement led to poor speed discrimination, patients’ performance would be significantly worse for the stimuli with a long display time than for the stimuli with a short display time. This was not the case in the above study; for the stimuli with different display times, the patients’ performance levels were similar (Figure 2A and B of Hong LE, Turano KA, O’Neill HB, et al.), although controls performed better for the stimuli with the longer display time of 300 ms. The similar patient performance under these stimulus conditions suggests that eye movement may not be a primary factor of deficient speed discrimination here. This line of research merits further study.

Associations have also been reported between basic motion processing (speed discrimination or detection of coherent motion) and several motion-related cognitive and social cognitive functions in schizophrenia patients. These cognitive functions include (1) tracking of multiple moving objects with attentive mind, which does not involve eye tracking, (2) recognition of biological motion, which requires identifications of various human actions (such as walking or running) solely based upon animated
motion signals, and (3) the capacity of theory of mind,\textsuperscript{49} which indexes one’s ability to infer other people’s mental states based upon their eyes. A potential confounding factor for these tentative relationships is the generalized deficit, whose presence may affect basic motion perception and cognitive functions in schizophrenia patients. If affecting basic motion processing and cognitive functions similarly, this factor could contribute to the tentative relationships of the 2 domains seen here. In order to evaluate this factor, one can examine the effects of modulating motion signals on cognitive processing in schizophrenia. If present, the generalized deficit presumably affects motion processing similarly, whether before or after the visual modulation, thus imposing no differential effects on cognitive processing. Any net effects yielded from visual modulation on cognitive functions should reflect the contribution of motion processing rather than generalized deficit. Such an approach can provide a direct assessment of the relationship between the 2 functional domains.

Motion perception receives inputs from early visual processing. A series of studies have found that cortical processing in the early visual system including the magnocellular pathway is deficient in schizophrenia.\textsuperscript{50–55} A natural question that emerges is whether the 2 types of visual deficits are related. A couple of studies showed that this may be the case. In one study, a correlation between speed discrimination and evoked potentials in response to simple visual stimuli was found in schizophrenia patients.\textsuperscript{19} In another study, a correlation between detection of coherent motion and visual masking was found in patients.\textsuperscript{56} Additional studies should be conducted to further evaluate the extent to which the early visual processing deficits contribute to motion processing in this mental disorder.

**Concluding Remarks**

Given its vital importance in understanding brain mechanisms and relevant social functions, motion processing has been an active area in schizophrenia research. The progress thus far suggests that the brain systems involved in motion processing can be a good model for evaluating spatiotemporal integration and sensory-cognitive relationships in this mental disorder.\textsuperscript{57} To translate the knowledge gained from this line of perceptual study into intervention, future research must further explore and link abnormal motion processing in the levels of neurochemistry, neural circuitry, cortical systems, and perception.

Future research in patients should also look into the relationships between basic motion processing and functional outcome in the real world. A recent study suggests that such relationships may be indirect, with social perception as mediator.\textsuperscript{38} A systematic assessment of this relationship will confirm the value of this line of translational research.

Another direction of translational research is to explore whether and to what extent deficient motion processing in schizophrenia patients can be improved through perceptual training. In healthy controls, motion perception can be enhanced through perceptual learning.\textsuperscript{38} If this neural plasticity-based training approach is effective in schizophrenia, not only motion perception but also relevant social cognitive functions may benefit.

The synthesis of the progress in this research area provides a glimpse of what studying motion processing can offer for the understanding of the basic brain mechanisms and for the development of targeted intervention for this mental disorder. Recently, one basic motion processing paradigm—detection of coherent motion—has been recommended as a promising perception paradigm for translation for use in clinical trials by the National Institute of Mental Health initiative on Cognitive Neuroscience Treatment Research to Improve Cognition in Schizophrenia.\textsuperscript{59} With continuing efforts in this area and in related research areas, our knowledge about and treatment strategies for this mental disorder will be further advanced.

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