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Impaired Visual Recognition of Biological Motion in Schizophrenia

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Abstract

Background: Motion perception deficits have been suggested to be an important feature of schizophrenia but the behavioral consequences of such deficits are unknown. Biological motion refers to the movements generated by living beings. The human visual system rapidly and effortlessly detects and extracts socially relevant information from biological motion. A deficit in biological motion perception may have significant consequences for detecting and interpreting social information.

Methods: Schizophrenia patients and matched healthy controls were tested on two visual tasks: recognition of human activity portrayed in point-light animations (biological motion task) and a perceptual control task involving detection of a grouped figure against the background noise (global-form task). Both tasks required detection of a global form against background noise but only the biological motion task required the extraction of motion-related information.

Results: Schizophrenia patients performed as well as the controls in the globalform task, but were significantly impaired on the biological motion task. In addition, deficits in biological motion perception correlated with impaired social functioning as measured by the Zigler social competence scale (Zigler and Levine, 1981). **Conclusion:** The deficit in biological motion processing, which may be related to the previously documented deficit in global motion processing, could contribute to abnormal social functioning in schizophrenia.

1.1 INTRODUCTION

Accumulating evidence suggests that perceptual and cognitive abnormalities may be among the core deficits of schizophrenia. Among these abnormalities are a host of deficits pointing to abnormal visual information processing. A large proportion of schizophrenia patients report visual abnormalities (e.g. affecting the way colors, people, space and facial expression) during at least some stages of the illness (Bunney et al., 1999; Cutting & Dunne, 1986). In line with these subjective reports are the results from perceptual studies documenting deficits on visual tasks such as detection of spatial location (Cadenhead et al., 1998), spatial frequency discrimination (O'Donnell et al., 2002), velocity discrimination and motion perception (Chen et al., 1999a,b).

One particularly intriguing visual impairment in schizophrenia is the relative difficulty of detecting weak translational motion in animations portraying a circumscribed set of coherently moving dots embedded within a larger array of randomly moving "noise" dots (Chen et al., 1999b, Chen et al., 2003; Li, 2002). However, in the natural environment we are seldom confronted with these kinds of visual motion conditions, so it is difficult to relate these deficits to the core features of schizophrenia. What we do routinely encounter, however, is dynamic optical information specifying the activities of people and animals, known as 'biological motion". Biological motion contains information about the identity of the moving stimulus, his or her actions, intentions, and even emotions. The human visual system is fine-tuned to detect biological motion rapidly and effortlessly and biological motion provides socially relevant information. Our ability to efficiently process social signals is crucial for effective social interactions. Therefore, a deficit in biological motion perception may have wide ranging consequences for social perception and interpersonal functioning.

It has been hypothesized that the brain has specialized networks for processing the unique patterns of optic flow specifying biological motion. These networks are thought to include the superior temporal sulcus (STS) and surrounding regions (Bonda, Petrides, Ostry, & Evans, 1996; Grossman et al., 2000; Hoffman & Haxby, 2000; Jellema, Baker, Wicker, & Perrett, 2000; Puce et al., 1998; Vaina et al, 2001). The superior temporal cortex is a central component of the neural circuitry that mediates our ability to utilize the "Theory of Mind" (ToM) (Baron-Cohen et al, 2000), which refers to ability to represent the mental states of others. Schizophrenia patients show deficits in tasks that demand the use of the ToM (Frith and Cocoran, 1996). Structural imaging data from schizophrenic patients reveal reduced volume of the left superior temporal gyrus that is correlated with increased psychotic symptoms, especially formal thought-disorder (Shenton et al 1992). Given that patients with schizophrenia have structural abnormalities of the superior temporal gyrus and show difficulties on tasks that are associated with the functional integrity of the superior temporal cortex, we hypothesized that they would show deficits in biological motion perception, which is supported by the neural circuitry that includes the superior temporal cortex.

We investigated whether patients with schizophrenia could distinguish biological from non-biological motion that is portrayed by point-light animation sequences (Johansson, 1973). Since deficits on a biological motion task could also reflect general deficits visual processing, we included a difficult perceptual grouping task (global-form task) that requires subjects to detect global form against background noise. Its difficulty would allow us to assess the motivational and attentional state of our subjects, and furthermore, this task would provide additional confirming evidence of relatively spared visual information processing when processing motion signals is not required (e.g. O'Donnell et al., 1996).

1.2. EXPERIMENTAL MATERIALS AND METHODS

1.2.1. Subjects

Fourteen outpatients (5 females) who met criteria for a DSM-IV diagnosis of schizophrenia were recruited from a private psychiatric hospital; diagnosis was determined on the basis of Structured Clinical Interview for DSM-IV (Spitzer &

Williams, 1985). The mean age of the patients was 38.3 years (SD = 7.8 years), mean education level was 12.6 years (SD = 2.0 years), and they had been ill for an average of 14.5 years (SD = 8.7 years). All patients were taking atypical antipsychotic medication at the time of testing (risperidone, clozapine or olanzapine). The CPZ equivalent dose was calculated (Bezchlibnyk-Butler and Jeffries, 1999) Clinical symptoms were assessed with the Brief Psychiatric Rating Scale (BPRS, Overall & Gorham, 1962). Positive and negative symptoms were assessed using the Scale for Assessment of Positive Symptom (SAPS, Andreasen, 1982), the Scale for Assessment of Negative Symptom (SANS, Andreasen, 1982) respectively. Mean BPRS, SAPS, and SANS scores were 28.3 (SD = 11.7), 30.8 (SD = 20.2) and 30.9 (SD = 20.7), respectively.

Fifteen control subjects (9 females) with no history of DSM-IV Axis 1 disorder as determined from SCID were recruited from the community. No control subject had neurological disorders or had first-degree relatives with DSM-IV Axis 1 disorders. No control subject was receiving psychotropic medications. Their mean age was 36.6 years (SD = 11.8 years) and their mean education level was 13.5 years (SD = 2.3 years), values not significantly different from those of the patient group. Control subjects were also screened to rule out schizotypal personality using the Schizotypal Personality Questionnaire (SPQ, Raine, 1991) before the experiment. SPQ is a 74-item true-false self-report questionnaire that assesses syndromes of schizotypal personality. According to Raine's data, 10% of the population scored above 41 (out of 74) and those individuals may have elevated risk for schizotypal personality disorder. The mean score of our control subjects on the SPQ was 18.1 (SD = 11.1), which is well below the cut-off score published by Raine (1991).

We assessed global social functioning in all subjects with the Zigler social competence scale (Zigler & Levine, 1981), which measures social competence. Variables that are used to calculate the Zigler score are age, education level, marital status, occupation, and employment history. Each variable is scored

between 0 (low/poor) to 2 (high/good). Then they are summed to yield a total score. Overall low score indicates reduced social functioning.

All subjects had normal or corrected-to-normal (eye glasses) vision. Practice trials were given before the experiment. During practice trials, all subjects responded they could discriminate dots and their motions from the background very clearly. The experimental protocol was approved by the Institutional Research Board of Vanderbilt University. All subjects gave written informed consent. All subjects were paid for their participation

INSERT TABLE 1

1.2.2. Stimuli and procedure

Displays were presented on a TFT-LCD monitor of a Macintosh computer, and controlled by Matlab© and the Psychophysics Toolbox (Brainard, 1997). All experiments were conducted in a room illuminated only by light from the video screen. In both biological motion task and global-form task, all stimuli appeared black against a gray background, because using black on gray minimizes afterimage persistence compared to white on gray in case of motion task, although persistence is not a concern when dealing with point-light animations.

Biological motion task

Biological motion sequences were generated using techniques detailed elsewhere (Grossman & Blake, 1999; Blake et al, 2003). 24 familiar activities, including running, kicking, climbing, throwing and jumping, were captured by videorecording an adult engaged in those activities. Video clips were imported to a computer to create biological motion stimuli. We placed markers on the joints in each frame of the sequence. These successive frames were converted to matrices that could be manipulated and animated in Matlab©. An example of several (non-successive) frames from a 'walking' biological motion sequence is shown on the left in Figure 1. Frames on the right in figure 1 show a series of frames (not successive) of a phase-scrambled version of the walker. Scrambled motion sequences were created from the normal biological animations and, thus, consisted of the same individual dots undergoing the same local motions as the biological counterparts. Scrambling was produced by randomizing the temporal phases and spatial locations of the dots in a given animation, thereby perturbing the hierarchical, pendular motions that are characteristic of biological motion. The size of each dot was approximately 12arc min, and the average speed within a sequence was 4 deg/s. Each animation sequence lasted 1 s. Viewing distance was 57 cm and the visual angle of each sequence was approximately 7 deg.

The subject's task was to judge whether each of a series of briefly presented animations depicted biological or scrambled motion. Before testing, the task was described to each subject and examples of biological and scrambled animations were shown to the subject to make sure that they understood the task by making oral responses to those examples Then a series of 100 trials was presented with each entailing the brief presentation of either a "biological" sequence or a "scrambled" (the order of trials was random, with the stipulation that exactly 50 of the presentations be biological). Following each trial, the subject indicated whether or not the animated dots portrayed "human" activity by pressing one of two preassigned computer keys; auditory feedback was provided after each trial. There was no time limit to respond. When subjects made a decision, he/she pressed a key to move on to the next trial. All fourteen schizophrenic patients finished this task, which lasted between 8-15 minutes depending on each individual's pace.

To derive an index of performance on this task, for each subject we tallied the number of 1) hits ("biological" response to biological motion sequences) and 2) false alarms ("biological" responses to scrambled sequences). Hit-rate and false alarm-rate were used to compute an unbiased measure of sensitivity, \underline{d}' , which is expressed as d' = | z (hit rate) – z (false alarm rate)| where z is a z-score calculated using the standard deviation from the mean.

INSERT FIGURE 1

Global-form task

This four-alternative, forced choice task measured the subject's ability to group small, stationary line elements into a larger, global form. The entire screen of the computer monitor was divided into four equal-sized quadrants whose boundaries were delineated by thick black lines, and the screen was filled with short lines most of which were oriented randomly. Each line subtended a visual angle approximately 30 min length \times 2 min width. In one of the four quadrants, a small group of six lines formed a guasi-circular shape within a randomly selected region of the guadrant, and the probability of the appearance of the guasi-circular shape in any of the quadrants was equal over the trials. Distracter lines and shapeforming lines were the same in color and size, thus the only cue for detecting target was the spatial arrangement termed "good continuation" in the Gestalt tradition. To manipulate the clarity of the target, we introduced jitter in the orientation of each line segment forming the quasi-circular shape; jitter comprised an angular deviation among target contours from the canonical value specified by their positions on the circle. Thus, larger degrees of jitter lessened the clarity of the target, impairing subjects' ability to detect the target and, therefore, designate in which quadrant it appeared. Displays remained visible until the subject responded. Viewing distance was the same as that of the biological motion task (57cm), and the visual angle of the target was approximately 2.5 deg. Examples of the displays are shown in the figure 2.

Each subject was instructed to locate the quasi-circular shape that looked like 'stop-sign' and to indicate in which quadrant it appeared. They indicated the quadrant by pressing one of four keys assigned to each quadrant (i.e., top left, top right, bottom left or bottom right). Although no sample stimuli were presented before the formal testing, the test began with a series of trivially easy trials (jitter = 0) so that each subject quickly became accustomed to the task. The degree of jitter over trials was adjusted by a staircase procedure to find the level of jitter at which the subject could identify the location of the target with greater than 70% accuracy. Thus, the target became more difficult to detect following correct answers and less difficult following incorrect answers. Visual feedback showing correct location was provided after each trial. The total number of trials was 100, and the mean and standard deviation of the jitter from the last 8 trials of the staircase were recorded as the estimate of the threshold. The subject could rest at any time during trials.

INSERT FIGURE 2

1.3. RESULTS

Biological motion task

Mean <u>d'</u> values and standard deviation (SD) in schizophrenia patients and controls are shown in figure 3. Clearly, the patients did not find the task impossible, which is hardly surprising given how easy it is. Still, discrimination of biological motion in the patient group was worse than in the control group (<u>d'</u> = 2.80 (0.84) vs. 2.21 (0.40)), with the difference between the two groups being statistically significant (<u>t</u> (27) = 2.41, <u>p</u> = 0.023).

The correlations between d' values and current symptom severity (BPRS), positive and negative symptom (SAPS, SANS) scores were examined for the schizophrenia group. No significant correlation was observed. Furthermore, no significant correlation between CPZ dose equivalent and d' was found. On the other hand, significant correlations were observed between \underline{d} values and social

functioning measured by the Zigler scores (figure 4): Zigler scores and <u>d'</u> values were significantly correlated ($\underline{r} = 0.71$, $\underline{p} < .0001$, n = 29) and the group difference in Zigler scores was also significant (t (27) = 5.84, p< 0.0001). Those subjects with impaired social functioning (as indicated by low Zigler scores) tended to have worse performance on the biological motion task.

Global-Form task

The mean (SD) jitter threshold values in the two groups are shown in figure 3. Jitter threshold (deg) in the patients group was comparable to that of normal subjects (16.9 (3.74) vs. 18.5 (3.42), <u>t</u> (27) = 1.19, <u>p</u> = 0.24). Performance of the patients on the global-form task did not correlate with BPRS, SAPS, SANS, illness duration, CPZ equivalent or Zigler scores.

INSERT FIGURE 3

INSERT FIGURE 4

1.4. DISCUSSION

Patients with schizophrenia showed a deficit in recognizing biological motion sequences whereas their performance on the global form task was comparable to that of healthy subjects, even though the global-form task was difficult and challenging. Note that in the global form task, there were only 6 small lines that were indistinguishable from the other numerous "noise" lines, and the only cue for target detection was their spatial arrangement. Normal performance on this task suggests that the patients were able to maintain their motivation and attention on a

perceptually difficult task. Results from the global-form task provide further support for the notion that visual processing in schizophrenia is relatively intact when motion detection is not required. Consistent with this interpretation, Chen et al. (1999b) showed that orientation and contrast detection was unimpaired in schizophrenia. Similarly, O'Donnell et al. (1996) found that schizophrenia patients processed form attributes such as high spatial frequencies and patterns just as well as controls did. The neural interconnections among orientation-selective neurons at early visual processing stages, including visual area V1 (Gilbert, 1993), are thought to be involved in the integration of contour information for perception of targets in displays such as those used by us (Field, Hayes, & Hess, 1993). These results suggest that general abnormalities of the primary visual cortex do not account for the deficits in biological motion perception in schizophrenia

From earlier work we know that schizophrenic patients have no trouble perceiving "local" motion, where "local" refers to linear contours moving in a single direction (Chen et al, 2003). However, these same patients do experience difficulty integrating local motion signals to achieve a sense of global, coherent translational motion (Chen et al, 2003), a task believed to involve the middle temporal area (MT) of the dorsal stream visual pathway (Born & Tootell, 1992). It is possible; of course, that the difficulty experienced by schizophrenic patients discriminating biological motion is another manifestation of their difficulty with global motion. After all, biological motion, too, requires integrating local motion signals over space and time, but it is unlikely that MT is selectively involved in registering the hierarchical pendular motions unique to point-light animations of the sort used in this study. Previous studies have revealed that MT responds just as strongly to presentation of scrambled motion sequences as it does to presentation of biological motion sequences (Grossman et al., 2000; Howard et al., 1996). Therefore MT does not seem to have the requisite sensitivity for discriminating between the two classes of animations. On the other hand, a region located in and near the superior temporal sulcus (STS) seems to be strongly and selectively activated by biological motion

signals (Jellema & Perrett, 2003). Damage to the STS region produces impairment in the ability to recognize biological motion while sparing the ability to perceive other types of visual motion (Schenk & Zihl, 1997a,b). It is possible that the impaired biological motion processing found in our study may arise from functional deficits within the superior temporal cortex. This hypothesis should be tested using functional imaging methods in the future. Whatever the neural bases of these perceptual deficits, our results underscore that these deficits in global motion processing in schizophrenic individuals may have potential social consequences because accurate and efficient processing of socially relevant stimuli forms the basis of social perception.

Other factors might have affected performance on these tasks. First, one might argue that impairments of the patients on biological motion task are caused by psychotropic medication. It is possible but unlikely. Some studies indicate that psychotropic medications may have some effect on patients' performance on perception (Purdon et al., 2000), whereas others did not found such effect on visual perception and attention (Allen et al., 1997). Moreover, in a recent study (Joober et al., 2002), schizophrenic patients receiving atypical antipsychotic drugs showed unimpaired visual contrast detection compared to unmedicated patients. Those evidences indicate that psychotropic medication may not affect perceptual and cognitive abilities of schizophrenic patients in any specific manner. In our study, the patients were taking low dose atypical antipsychotic drugs and the CPZ dose equivalent was not correlated with their performance on biological motion perception (d'). Second, it could be argued that detecting black stimuli (dots and lines) against a gray background might be difficult for patients. However, both biological motion and global-form tasks had the same background color but the patients' performance on the global-form task was comparable to that of normal control subjects. If low-level vision problems were present in the patients, performance on both tasks should have been affected. Finally, there were unequal proportions of women in the two groups (36% women in the patient group and 50%

women in the control group). In both groups, men showed higher d' than women on biological motion task. However, no interaction between sex and groups was observed and in spite of the fact that the proportion of men was higher in the patient group, they showed deficits as a group compared with the normal controls.

It is noteworthy that the pattern of deficits, that is, impaired biological motion perception but intact global-form perception found in this study is similar to those observed in children with autism (Blake et al., 2003). An important characteristic of children with autism is impaired Theory of Mind (ToM), or the ability to represent one's own and other people's mental states. Indeed, there is evidence that some patients with schizophrenia have ToM impairments that are associated with negative symptoms (Corcoran, Mercer, & Frith, 1995; Doody et al., 1998; Frith & Corcoran, 1996; Pickup & Frith, 2001). Studies of the neural circuitry for ToM have implicated the amygdala, a limbic structure that is thought to affect social behavior, and its connection with the STS region as parts of such a brain network (Baron-Cohen et al., 2000: Review). Although at present we do not know the details of how this network operates, impaired biological motion perception in patients with schizophrenia may be a behavioral sign related to a ToM deficit. Moreover, although impaired biological motion perception has been observed in both schizophrenic and autistic patients, the degree of impairment seems to be different. More profound deficits of biological motion (Blake et al., 2003) were observed in autistic children. Similarly, Pickup and Frith (2001) and Pilowsky et al. (2000) found more severe ToM deficits in autistic subjects than in schizophrenic patients. Thus, impaired biological motion perception may be coupled with the severity of ToM deficit.

To summarize, the results of the present study suggest that patients with schizophrenia have impairments in recognizing biological motion and this deficit may contribute to some of the social dysfunction associated with schizophrenia. However, the nature of this association remains to be examined in detail.

REFERENCES

Allen, D.N., Gilbertson, M.W., van Kammen, D.P., Kelley, M.E., Gurklis, J.A. Jr., & Barry, E.J., 1997. Chronic haloperidol treatment does not affect structure of attention in schizophrenia. <u>Schizophrenia Research</u>, 25, 53-61.

Allison, T., Puce, A., & McCarthy, G. (2000). Social perception from visual cues: Role of the STS region. <u>Trends in Cognitive Science, 4</u>, 267-278.

Andreasen N.C., & Olsen S (1982) Negative v positive schizophrenia. Definition and validation. <u>Archives of General Psychiatry</u>, <u>39</u>,789-94.

Baron-Cohen, S., Ring, H.A., Bullmore, E.T., Wheelwright, S., Ashwin, C., & Williams, S.C.R. (2000). The amygdala theory of autism. <u>Neuroscience and</u> <u>Biobehavioral Reviews</u>, 24, 355-364.

Bezchlibnyk-Butler, K.Z and Jeffries J.J. (1999) <u>Clinical handbook of psychotropic</u> <u>drugs; 9th Edition</u>. Seattle: Hogrefe & Huber Publishers.

Blake, R., Turner, L.M., Smoski, M.J., Pozdol, S.L., & Stone, W.L. (2003). Visual recognition of biological motion is impaired in children with autism. <u>Psychological</u> <u>Science, 14</u>, 151-157.

Bonda, E., Petrides, M., Ostry, D., & Evans, A. (1996). Specific involvement of human parietal systems and the amygdala in the perception of biological motion. <u>The Journal of Neuroscience, 16</u>, 3737-3744.

Born, R.T., & Tootell, R.B. (1992). Segregation of global and local motion processing in primate middle temporal visual area. <u>Nature, 357</u>, 497-499.

Brainard, D.H. (1997). The Psychophysics Toolbox. Spatial Vision, 10, 443-446.

Bunney, W.E. Jr., Hetrick, W.P., Bunney, B.G., Patterson, J.V., Jin, Y., Potkin, S.G., Sandmai, C.A (1999). Structured interview for assessing perceptual anomalies (SIAPA). <u>Schizophrenia Bulletin, 25</u>, 577-592.

Cadenhead, K.S., Serper, Y., and Braff, D.L. (1998) Transient versus sustained visual channels in the visual backward masking deficits of schizophrenia patients. <u>Biological Psychiatry, 43</u>, 132-138.

Chen, Y., Nakayama, K., Levy, D.L., Matthysse, S., & Holzman, P.S. (1999a) Psychophysical isolation of a motion-processing deficit in schizophrenics and their relatives and its association with impaired smooth pursuit. <u>Proceedings of the</u> <u>National Academy of Sciences of the United States of America, 96</u>, 4724-4729.

Chen, Y, Palafox, G.P., Nakayama, K., Levy, D.L., Matthysse, S., & Holzman, P.S. (1999b). Motion perception in schizophrenia. <u>Archives of General Psychiatry. 56</u>, 149-154.

Chen, Y., Nakayama, K., Levy, D., Matthysse, S., & Holzmann, P. (2003). Processing of global, but not local, motion perception is deficient in schizophrenia. <u>Schizophrenia Research, 61</u>, 215-227.

Chen Y, Levy, D.L, Sheremata, S.,. Nakayama, K., Matthysse, S., & Holzman, P.S (2003). Effects of typical, atypical, and no antipsychotic drugs on visual contrast detection in schizophrenia. <u>Am J Psychiatry</u> 160: 1795-1801.

Corcoran, R., Mercer, G., & Frith, C.D. (1995). Schizophrenia, symptomatology and

social inference: Investigating "theory of mind" in people with schizophrenia. <u>Schizophrenia Research, 17</u>, 5-13.

Cutting, J., Dunne, F. (1986). The nature of the abnormal perceptual experiences at the onset of schizophrenia. <u>Psychopathology</u>, 19, 347-52.

Doody, G.A., Götz, M., Johnstone, E.C., Frith, C.D., & Cunningham Owens, D.G. (1998). Theory of mind and psychoses. <u>Psychological Medicine</u>, *28*, 397-405.

Field, D.J., Hayes, A., & Hess, R.F. (1993). Contour integrations by the human visual system: Evidence for a local "association" field. <u>Vision Research, 33</u>, 173-193.

Frith, C.D., Corcoran, R. (1996). Exploring 'theory of mind' in people with schizophrenia. <u>Psychological Medicine</u>, *26*, 521-30.

Gilbert, C. (1993). Circuitry, architecture and functional dynamics of visual cortex. <u>Cerebral Cortex, 3</u>, 373-386.

Grossman, E., & Blake, R. (1999). Perception of coherent motion, biological motion and form-from-motion under dim-light conditions. <u>Vision Research</u>, <u>39</u>, 3721-3727.

Grossman, E., Donnelly, M., Price, R., Morgan, V., Pickens, D., Neighbor, G., & Blake, R. (2000). Brain areas involved in perception of biological motion. <u>Journal of Cognitive Neuroscience</u>, 12, 711-720.

Hoffman, E.A., & Haxby, J.V. (2000). Distinct representation of eye gaze and identity in the distributed human neural system for face perception. <u>Nature</u> <u>Neuroscience, 3</u>, 80-84.

Howard, R.J., Brammer, M., Wright, I., Woodruff, P.W., Bullmore, E.T., & Zeki, S. (1996). A direct demonstration of functional specialization within motion-related visual and auditory cortex of the human brain. <u>Current Bioloogy</u>, 6, 1015-1019.

Jellema, T., Baker, C.I., Wicker, B., & Perrett, D.I. (2000). Neural representation for the perception of the intentionality of actions. <u>Brain and Cognition</u>, 44, 280-302.

Jellema, T., & Perrett, D.I. (2003). Cells in monkey STS responsive to articulated body motions and consequent static posture: a case of implied motion? <u>Neuropsychologia</u>, 41, 1728-1737.

Johansson, G. (1973). Visual perception of biological motion and a model for its analysis. <u>Perception & Psychophysics</u>, 14, 201-211.

Joober, R., Rouleau, G.A., Lal, S., Dixon, M., O'Driscoll, G., Palmour, R., Annable, L., Bloom, D., Lalonde, P., Labelle, A., & Benkelfat, C., 2002. Neuropsychological impairments in neuroleptic-responder vs. -nonresponder schizophrenic patients and healthy volunteers. <u>Schizophrenia Research</u> 53:229-238.

Li, C.S. (2002). Impaired detection of visual motion in schizophrenia patients. <u>Progress in Neuro-Psychopharmacology and Biological Psychiatry, 26</u>, 929-934.

O'Donnell, B.F., Swearer, J.M, Smith, L.T., Nestor, P.G., Shenton, M.E., & McCarley, R.W. (1996). Selective deficits in visual perception and recognition in schizophrenia. <u>American journal of Psychiatry, 153</u>, 687-692.

O'Donnell, B.F., Potts, G.F., Nestor, P.G., & Stylianopoulos, K.C., Shenton M.E., & McCarley, R.W. (2002) Spatial frequency discrimination in schizophrenia. <u>Journal</u> <u>of Abnormal Psychology. 111</u>, 620-625.

Overall J.E.,& Gorham D.R. (1962) The Brief Psychiatric Rating Scale. <u>Psychological Reports</u>, 10,799-812.

Pickup, G.J., & Frith, C.D. (2001). Theory of mind impairments in schizophrenia : symptomatology, severity and specificity. <u>Psychological Medicine</u>, <u>31</u>, 207-220.

Pilowsky, T., Yirmiya, N., Arbelle, S., & Mozes, T. (2000). Theory of mind abilities of children with schizophrenia, children with autism, and normally developing children. <u>Schizophrenia Research, 42</u>, 145-155.

Puce, A., Allison, T., Bentin S., Gore J.C., & McCarthy, G. (1998). Temporal cortex activation in humans viewing eye and mouth movements. <u>The Journal of Neuroscience, 18</u>, 2188-2199.

Purdon, S.E., Jones, B.D., Stip, E., Labelle, A., Addington, D., David, S.R., Breier, A., & Tollefson, G.D., 2000. Neuropsychological change in early phase schizophrenia during 12 months of treatment with olanzapine, risperidone, or haloperidol. <u>Archives of General Psychiatry, 57</u>, 249-258.

Raine, A. (1991). The SPQ: A scale for the assessment of schizotypal personality based on DSM III-R criteria. <u>Schizophrenia Bulletin.17</u>, 556-564.

Ransil, B.J., & Schachter, S.C. (1994). Test-retest reliability of the Edinburgh Handedness Inventory and Global Handedness preference measurements, and their correlation. <u>Perceptual and motor skills, 79</u>, 1355-72

Schenk, T., & Zihl, J. (1997a). Visual motion perception after brain damage: I. Deficits in global motion perception. <u>Neuropsychologia</u>, <u>35</u>, 1285-1297.

Schenk, T., & Zihl, J. (1997b). Visual motion perception after brain damage: II. Deficits in form-from-motion perception. <u>Neuropsychologia</u>, <u>35</u>, 1299-1310.

Shenton, M.E., Kikinis, R., Jolesz, F.A., Pollak, S.D., LeMay, M., Wible, C.G., Hokama, H., Martin, B.S., Metcalf, D., Coleman, M., & McCarley, R.W. (1992). Abnormalities of the left temporal lobe and thought disorder in schizophrenia. A quantitative magnetic resonance imaging study. <u>The New England Journal of</u> <u>Medicine, 327</u>, 604-12.

Spitzer, R.L., & Williams, J.D.W. (1985) <u>Structured Clinical Interview for DSM III-R</u>. New York State Psychiatric Institute Biomedical Research Division, New York.

Vaina, L.M., Solomon, J., Chowdhury, S., Sinha, P., & Bellivieau, J.W. (2001). Functional neuroanatomy of biological motion perception in humans. <u>Proceedings</u> of the National Academy of Sciences of the United States of America, 98, 11656-11661.

Wechsler Abbreviated Scale of Intelligence[™] (WASI[™]). (1999) <u>The Psychological</u> <u>Corporation.</u>

Zigler, E., & Levine, J. (1981). Premorbid competence in schizophrenia : What is being measured? <u>Journal of Consulting and Clinical Psychology</u>, 49, 96-105.

	Control Subjects	Schizophrenic Subjects
	(n = 15)	(n = 14)
Age	36.6 (11.8) ^A	38.3 (7.8)
Education (years)	13.5 (2.3)	12.6 (2.0)
WASI IQ Score ^B	95.7 (15.0)	95.0 (12.6)
BPRS	N/A	28.3 (11.7)
SANS	N/A	30.9 (20.7)
SAPS	N/A	30.8 (20.2)
SPQ	18.1 (11.1)	N/A
Handedness (L/R/Ambi)	1/14/0	0/13/1
CPZ Equivalent ^D	N/A	290.8(143.9)
Illness Duration (years)	N/A	15.0 (8.39)
Zigler Score ^E	5.9 (1.8)	2.5 (1.3)

Table 1. The demographic data

^A Mean (standard deviation)

^BWechsler Abbreviated Scale of Intelligence™ (WASI™).

^c Chlorpromazine dose equivalent (mg/day)

^E Zigler Social Competence Scale (Zigler, E., & Levine, J., 1981) ranges from minimum of zero to maximum of 8

FIGURE CAPTIONS

Fig. 1. Examples of stimuli used in the biological motion task. Frames on the left show normal biological activity (walking) from quasi-successive point-light animation sequences. Scrambled motion frames on the right side are the corresponding counterparts of the biological sequences on the left, containing the same dots undergoing the same local motions, only spatiotemporal phases were scrambled to generate meaningless, nonbiological motion.

Fig. 2. Examples of the global form task. The target was a quasi-circular shape formed by a small group of six lines. The difficulty of the target was adjusted by varying jitter, the angular deviations among target contours from the canonical values, according to a staircase procedure. In example A, the target is easily recognized at the upper-right quadrant (see arrow). Example B shows more difficult trial: the target is much less clear (lower-left quadrant, see arrow)

Fig.3. d' on the biological motion task (left) and angular jitter thresholds on the global-form task (right) for 70% accuracy. Each dot represents an individual d' or jitter threshold. Mean (SE) values were given in each panel.

Fig. 4. Correlation between <u>d</u>' and scores on Zigler social competence scale.





Figure 2

А



Figure 3



