Reading in Schizophrenic Subjects and Their Nonsymptomatic First-Degree Relatives

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Previous studies have demonstrated eye movement abnormalities during smooth pursuit and antisaccadic tasks in schizophrenia. However, eye movements have not been investigated during reading. The purpose of this study was to determine whether schizophrenic subjects and their nonsymptomatic first-degree relatives show eye movement abnormalities during reading. Reading rate, number of saccades per line, amplitudes of saccades, percentage regressions (reverse saccades), and fixation durations were measured using an eye tracker (EyeLink, SensoMotoric Instruments, Germany) in 38 schizophrenic volunteers, 14 nonaffected first-degree relatives, and 57 control volunteers matched for age and National Adult Reading Test scores. Parameters were examined when volunteers read full pages of text and text was limited to progressively smaller viewing areas around the point of fixation using a gaze-contingent window. Schizophrenic volunteers showed significantly slower reading rates ($P = .004$), increase in total number of saccades ($P \leq .001$), and a decrease in saccadic amplitude ($P = .025$) while reading. Relatives showed a significant increase in total number of saccades ($P = .013$) and decrease in saccadic amplitude ($P = .020$). Limitation of parafoveal information by reducing the amount of visible characters did not change the reading rate of schizophrenics but controls showed a significant decrease in reading rate with reduced parafoveal information ($P < .001$). Eye movement abnormalities during reading of schizophrenic volunteers and their first-degree relatives suggest that visual integration of foveal and parafoveal information may be reduced in schizophrenia. Reading abnormalities in relatives suggest a genetic influence in reading ability in schizophrenia and rule out confounding effects of medication.

**Key words:** schizophrenia/reading/visual integration/ saccades/fixations/relatives

Introduction

Reduced scholastic performance has been reported in schizophrenia, which usually occurs long before diagnosis.1 However, reading performance has been poorly investigated in schizophrenia. Reading deficits could coalesce with other impairments associated with schizophrenia leading to poor integration into society.

Several studies have emerged describing reading deficits in schizophrenia although the mechanisms behind these impairments are not fully understood.2–5 Previous studies have linked reading deficits to poor comprehension,4 working memory deficits,3 difficulties with phonological processing,2 and dysfunctional visual processing through the magnocellular pathway.5 Both phonological processing deficits and magnocellular pathway dysfunction have also been implicated as causes of developmental dyslexia, which has led to comparisons between these 2 diseases.2,5 In general, previous studies have assessed reading using psychoeducational tools comparing other measures of cognitive or visual function. We have employed an alternative approach using eye movement recordings to investigate ocular motor performance in schizophrenia. Reduced ocular motor function in schizophrenia has been well documented for tasks such as smooth pursuit and antisaccades where the abnormalities are a reflection of high-level deficits in cortical processing.6–9

From the ocular motor perspective, reading consists of a series of fixations and saccades to acquire visual information, which is integrated from foveal and parafoveal (the area of the retina immediately surrounding the fovea retina).10,11 Semantic information about words is processed in foveal vision, while lower level information (like word length and orthographic information) is processed in parafoveal vision.12 The ocular motor reading pattern is the result of the coordinated contributions of
multiple cognitive, sensory, and motor neural processes many of which can be affected by schizophrenia. Consequently, abnormalities in eye movement reading patterns cannot be easily used to infer specific deficits in neural substrates. They do, however, reflect the ongoing moment by moment processing mechanisms taking place during the reading task. For example, the fixation duration is an indication of the difficulty an individual has in processing fixated words. Saccades made to reread text (ie, in 200897)Similar changes in reading observed in relatives 17.5 3 grid (width 0.01 During reading, the perceptual span of effective integration of parafoveal vis...ability to process nonfoveated information, either through integration of parafoveal visual information or from guessing words from their context.11,13

There has been much recent interest in visual deficits associated with schizophrenia. Perceptual abnormalities, such as reduced gain control (adaptation and optimization of visual responses to their immediate context) and poor visual integration (assimilation of simpler components of vision such as color, motion, and orientation into a more global representation), have recently been detected.14,15 Recently, Knebel et al used visual evoked potentials (VEPs) to show that schizophrenia leads to deficits in the early visual processing of parafoveal vision.16 During reading, the perceptual span of effective vision extends into the parafoveal vision, from 3 to 4 character spaces to the left of fixation to 14–15 character spaces to the right.17,18 Much of this knowledge has been attained using the gaze-contingent window technique (GCWT), where text is replaced outside of a window locked to the point of fixation with a mask, such as letter x’s. This technique has been used in the current study to investigate of foveal and parafoveal integration during reading 10,11,17

Our aim was to investigate reading performance in schizophrenia using eye movement recordings to characterize ocular motor performance for the first time comparing ocular motor characteristics previously described for development dyslexia. In addition, we have used the GCWT to investigate foveal and parafoveal integration. We compared reading performance in first-degree unaffected relatives of schizophrenic subjects because they demonstrate ocular motor deficits that could be possible genetic markers for schizophrenia.19-21 Similar changes in reading observed in relatives would also rule out confounding effects of medication.

Our underlying hypothesis is that schizophrenic volunteers have deficits in the integration of foveal and parafoveal information, leading to a reduction in the amplitude of saccades to move onto subsequent words, which results in slower reading. Our a priori hypothesis is that reducing parafoveal information using the GCWC has significantly less effect on reading in schizophrenic volunteers compared with controls. Because there is evidence of a genetic component to schizophrenia, we expected changes in first-degree relatives.

Methods

Experimental Setup and Protocol

For all experiments, an infrared video pupil tracker (Eyelink eye tracker, SensoMotoric Instruments GmbH, Berlin, Germany; resolution 0.005°, range ± 30° noise level < 0.01° root mean square, sample rate 250 Hz) was used to record horizontal and vertical eye positions. During the eye movement–recording mode, the EyeLink system operates under a MS-DOS (Microsoft Corporation) environment recording all data directly to hard disk. After completion of an experiment, the data are transferred over a network link into a Windows environment where the data are converted into Spike2 neurophysiological software files (Cambridge Electronic Design, UK) using a batch process, which employs custom-written C++ programs. All files were subsequently backed up using recordable media.

Stimuli were projected onto a rear projection screen of 1.75 m width and 1.17 m height (VisLab projection system; SensoMotoric Instruments, GmbH) and Hitachi CP-X958 LCD video projector (resolution 1024 x 768). Volunteers sat at a distance of 1.2 m from the screen with their head on a chin rest. Before each experiment, eye data were calibrated using 9 fixation points projected in a 3 x 3 grid (width ± 20°; height ± 17.5°). Calibrations of the eye data were repeated until validations of the calibration data demonstrated an average error in both the horizontal and vertical dimensions of <0.5° visual angle. Drift corrections were performed before each task.

Reading text was then projected on to the screen using texts from a modern English translation of the Brother Grimm fairy tales (allowing comparison with the Moorfields reading chart). The size of the text was equivalent to a LogMAR visual acuity (VA) of 0.76 (approximately 6/36 Snellen VA).

Distance VA was tested with logMAR ETDRS crowded letters (Keeler Ltd) at 4m, near VA with Moorfields Bar Reading Book at 40cm (Clement Clarke Ltd), and stereo...
window sizes (2, 4, 8, 16 visible characters, and a full page of text) presented in random order. Three characters were visible to the left of fixation for the first 4 conditions (see online supplementary figure 1). Each subject was asked to silently read 2 pages for each window size.

The update of the display took a maximum of 2 full screen (FS) refreshes (ie, 33 ms or less) relative to a change in eye position which was sampled every 4 ms. This delay was not long enough to be perceived by the participants. A small amount of vertical drift of gaze data can occur in the EyeLink system due to headband slippage with time. To accommodate for this, the vertical extent of the moving window was set to 1.7° (the text height was a maximum of 0.5°) and a text spaced was used with a line width of 1.5 lines (equivalent to 1.5°). In addition, drift corrections were performed to correct any horizontal or vertical error prior to the presentation of every text paragraph.

For both experiments, multiple-choice questions (MCQ) based on the text were asked after each page to determine comprehension. All groups answered 98.6% or more of the questions correctly (experiment 1 schizophrenics and controls, relatives and controls, 98.6%, 99.6%, 100%, and 100%, respectively; experiment 2 schizophrenics 99.0% and controls 99.2%).

Subjects
All subjects were native English speakers and had best-corrected distance VA of 6/9.5 or better, corrected near VA of N6 or better, and stereoacuity of 240 minutes arc or better. Premorbid IQ was assessed using the National Adult Reading Test (NART). NART scores and age were matched between control groups and patient groups. All schizophrenic subjects met Diagnostic and Statistical Manual for Mental Disorders, Fourth edition, criteria for schizophrenia. The symptom severity of schizophrenic subjects was measured using a positive and negative syndrome scale.

The study received local ethical approval and conformed to the Declaration of Helsinki. After complete description of the study to the subjects, written informed consent was obtained.

**Experiment 1** Four groups of subjects were included: (1) patients with schizophrenia (n = 22), (2) controls for patients (similar mean and SD for age and NART score, n = 25), (3) nonaffected first-degree relatives of schizophrenics (n = 14, including 11 who were related to the schizophrenic patients in this study) and, (4) controls for relatives (n = 17). Twenty-one patients were on atypical antipsychotic medication, olanzapine (n = 5), risperidone (n = 4), clozapine (n = 3), aripiprazole (n = 3), aripiprazole with risperidone (n = 1), aripiprazole with clozapine (n = 1), and amisulpride (n = 1) and 3 were on typical antipsychotic medication, pipotiazine (n = 1), sulpiride (n = 1), and phenothiazine (n = 1) and 1 subject was not on medication at time of testing. The schizotypal symptoms of all relatives and controls were recorded on the day of examination using the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE) scoring system. The O-LIFE consists of a series of questions that rates individuals along 4 scales of schizotypal traits: (1) unusual experiences, (2) cognitive disorganization, (3) introverted anhedonia, and (4) impulsive nonconformity.

**Experiment 2** A group of schizophrenic patients (n = 16) and a group of age- and NART-matched controls (n = 15) were included. Fourteen schizophrenic subjects were on atypical antipsychotic medication, olanzapine (n = 5), risperidone (n = 3), aripiprazole (n = 3), pipotiazine (n = 1), sulpiride (n = 1), and clozapine (n = 1).

Subject characteristics are shown in table 1. For both experiments, none of the relatives or controls were on antipsychotic medication.

Table 1. Details of Volunteers Recruited for the 2 Experiments Showing Mean (and SD)

<table>
<thead>
<tr>
<th></th>
<th>Schizophrenia</th>
<th>Control</th>
<th>Relative</th>
<th>Control</th>
<th>Schizophrenia</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>22</td>
<td>25</td>
<td>14</td>
<td>17</td>
<td>16</td>
<td>15</td>
</tr>
<tr>
<td>Age (y)</td>
<td>38 (8.9)</td>
<td>38 (13.9)</td>
<td>53 (16.0)</td>
<td>52 (14.3)</td>
<td>39 (10.7)</td>
<td>41 (12.1)</td>
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<tr>
<td>NART score</td>
<td>110 (7.9)</td>
<td>111 (6.4)</td>
<td>114 (5.5)</td>
<td>113 (5.6)</td>
<td>107 (9.6)</td>
<td>113 (6.0)</td>
</tr>
<tr>
<td>Visual acuity (logMAR)</td>
<td>−0.07 (0.12)</td>
<td>−0.03 (0.13)</td>
<td>−0.03 (0.13)</td>
<td>−0.01 (0.14)</td>
<td>−0.07 (0.12)</td>
<td>−0.04 (0.10)</td>
</tr>
<tr>
<td>Disease duration (y)</td>
<td>7.1 (4.5)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>7.9 (4.6)</td>
<td>—</td>
</tr>
<tr>
<td>Age at onset (y)</td>
<td>27 (7.8)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>27 (7.7)</td>
<td>—</td>
</tr>
<tr>
<td>PANSS-P</td>
<td>15.3 (5.9)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>15.7 (5.9)</td>
<td>—</td>
</tr>
<tr>
<td>PANSS-N</td>
<td>14.9 (6.0)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>14.5 (5.6)</td>
<td>—</td>
</tr>
<tr>
<td>PANSS-G</td>
<td>28.1 (9.6)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>26.1 (10.1)</td>
<td>—</td>
</tr>
<tr>
<td>Daily chlorpromazine equivalent (mg)</td>
<td>262 (125)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>288 (141)</td>
<td>—</td>
</tr>
</tbody>
</table>

Note: Positive and Negative Syndrome Scale including positive (PANSS-P), negative (PANSS-N), and general scales (PANSS-G) are shown. NART, National Adult Reading Test.
Analysis and Statistics

For both experiments, reading rate (words/min), total number of saccades per line (forward and reverse saccades), amplitude of saccades (of forward saccades only), fixation duration, and percentage regressions (ie, mean number of reverse saccades × 100%/mean of total number of saccades during line reading) were analyzed (see figure 1). Saccadic disconjugacy was also estimated from absolute differences in saccadic amplitudes for left and right eyes.

Since ocular motor data were normally distributed, the general linear model was used for statistical analysis. Univariate analysis was used for experiment 1 including group (eg, schizophrenic or control) as fixed factor and ocular motor parameters as the outcome measure (eg, total number of saccades). In addition, linear mixed models were used to estimate heritability of ocular motor parameters between schizophrenic volunteers and their relatives. Schizotypal traits (O-LIFE scores) were compared between schizophrenic relatives and controls using the Mann-Whitney U test.

Repeated measures ANOVA and regression analysis were used for second experiment (GCWT). The effect of reducing parafoveal information on reading parameters in schizophrenics compared with controls was tested using the significance of the interaction term for window size × group.

Results

Experiment 1: Text Reading

Examples of the reading pattern observed for a schizophrenic volunteer, a relative, and age-matched controls are shown in figure 1. The typical staircase pattern of saccades and fixations from left to right (when reading a line) with large return saccade to the left (to a new line) was observed for all individuals. The schizophrenic subject made more saccades of smaller amplitudes resulting in a slower reading rate per line compared with the control subject. The relative of the schizophrenic volunteer also made more saccades with a slower reading rate compared with the control.

Mean values (with SEs) of ocular motor characteristics analyzed from eye movement recordings are shown in figure 2. Schizophrenic volunteers read significantly slower compared with controls ($F = 9.09, P = .004$) with a mean reading rate of 227 words/minutes compared with 297 words/minutes in controls (figure 2A). Although relatives of schizophrenic showed a similar trend (mean reading rate of 274 words/minutes, falling between that of...
schizophrenic patients and controls), they were not significantly different to age-matched controls ($F = 1.13$, $P = .300$, reading rate = 303 words/min) (figure 2A).

Of the ocular motor characteristics contributing to reading rate (figure 2B–E), the parameter with the clearest differences was the total number of saccades per line (figure 2B), which was significantly greater in both the schizophrenic group ($F = 17.73$, $P = .00014$) and relatives ($F = 6.94$, $P = .013$) in comparison with age-matched control groups. This difference was not due to greater numbers of regressions in schizophrenics and relatives, which were similar in proportion to

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**Fig. 2.** Means and standard error of means of all the subjects for the analyzed parameter: (A) reading rate (words/min), (B) total number of saccades per line, (C) percentage regressions (%), (D) saccadic amplitude (°), (E) fixation duration (ms). Note: Statistically significant differences between schizophrenic patients or relatives and respective control groups were (A) $F = 9.09$, $P = .004$; (B) $F = 17.73$, $P = .00014$; (C) $F = 6.94$, $P = .013$; (D) $F = 5.39$, $P = .025$, and (E) $F = 6.07$, $P = .020$. 

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E. O. Roberts et al
controls (~18% of the total number of saccades) (figure 2C). Schizophrenics and relatives also made significantly smaller saccades compared with controls ($F = 5.39, P = .025$ and $F = 6.07, P = .020$, respectively) (figure 2D). Fixation duration was not significantly different between the groups. Mean values fell between 200 and 250 ms (figure 2E).

There was no significant differences in the degree of saccadic disconjugacy between schizophrenics and controls or relatives and controls ($P > .05$).

Linear mixed models did not reveal any patterns of heritability of ocular motor traits between schizophrenic volunteers and their relatives (reading rate: $P = .642$; number of saccades per line: $P = .325$; percentage regressions: $P = .658$; amplitude of saccades: $P = .202$; fixation duration: $P = .276$). In addition, there were no significant differences between schizophrenic relatives and controls for any of the 4 schizotypy scales: unusual experiences ($P = .857$), cognitive disorganization ($P = .424$), introverted anhedonia ($P = .679$), and impulsive nonconformity ($P = .228$). There were no significant correlations between any of the ocular motor parameters and daily equivalent chlorpromazine dose ($P > .05$ for all parameters).

We compared the relative sensitivities, specificities, and positive predictive values of ocular motor parameters for detection of schizophrenia. We used thresholds that set the sensitivity at 72.7% for all parameters, which allows relative comparison of specificity. The resultant specificities and positive predictive values, respectively, for each parameter were: 56.0% and 59.3% for reading rate, 60.0% and 61.5% for total number of saccades per line, 44.0% and 53.3% for proportion of regressions, 52.0% and 57.1% for amplitude of saccades, 28.0% and 47.1% for fixation duration.

**Experiment 2: GCWT**

Schizophrenic volunteers showed little change in reading patterns when parafoveal viewing was disrupted by reducing the window size. They used similar number of saccades per line with similar reading speeds irrespective of window size (figure 3A). In contrast, in a representative control volunteer increasing window size and parafoveal information decreased the number of saccades per line and increased the reading rate (figure 3B).

Figure 4A shows the mean effect of changing window size on reading rate. Repeated measures ANOVA demonstrated that window size had a significant effect on the reading rate ($F = 21.24, P < .001$) with a significant interaction between window size and group (ie, schizophrenics or controls, $F = 5.60, P = .026$). This was because of the greater change in reading rate with window size in the control group compared with the schizophrenic group evident from the difference in slopes between the groups (slope = 0.068 for schizophrenics and 0.396 for controls; $P = .126$ for schizophrenics and $P < .0001$ for controls).

Window size had a significant effect on total number of saccades (figure 4B) ($F = 7.6, P = .01$), mainly due to the effect of the control group ($F = 7.10, P < .001$ for control group and slope = -0.873, $P < .001$ for schizophrenic group, when treated independently), although the interaction between window size and group was not significant ($P = .94$). In contrast to the first experiment, there was a significant difference in the number of regressions made by schizophrenics and controls ($F = 7.10, P = .012$, figure 4C), with the schizophrenic group making more regressions overall. The window size had a significant effect on percentage regressions in the group as a whole ($F = 8.03, P = .008$).
Amplitude of saccades (figure 4D) and fixation duration (figure 4E) showed opposite patterns with significantly increasing amplitude of saccades \((F = 51.0, P < .0001)\) and decreasing fixation durations with increasing window size \((F = 26.7, P < .0001)\) overall. There were no significant differences between controls and schizophrenics for either parameter (saccadic amplitudes: \(F = 0.024, P = .878\) and fixation duration: \(F = 0.3254, P = .573\)). However, there was a strong interaction between window size and group for amplitude of saccades \((F = 8.33, P = .007)\) because of the greater change in amplitude of saccades with window size in the control group (the interaction between window size and group for fixation duration was near to significance; \(F = 3.70, P = .064\)). Closer observation shows that this is mainly due to a flattening of the curve in the schizophrenic group (filled squares, figure 4D) when the window changes from 8 letters to the right of fixation visible to 16 letters and FS. This pattern of less change occurring at larger window size than 8 letters to the right can also be clearly observed for fixation duration and number of regressions.

Discussion

Experiment 1: Text Reading

We found that during reading of text paragraphs schizophrenics made significantly smaller and more frequent saccades than controls, resulting in significantly slower reading rates. A similar saccadic pattern was seen in relatives, although the reading rate in relatives was not significantly different from either the control or schizophrenic groups. The number and amplitude of saccades of relatives fell between the schizophrenics and controls.
In contrast, mean fixation duration, which reflects the difficulty an individual has in processing words directly under fixation, was not significantly different between schizophrenic and controls. Previous studies manipulating the disappearance of text immediately after fixation show that most of the processing time during fixations is due to cognitive rather than visual processing of the text. Our results imply that individuals with schizophrenia do not have significant difficulties in visual and cognitive processing of words currently being fixated. This is also substantiated by the observation that schizophrenics did not make significantly more regressions to reread words in experiment 1. Regressions indicate difficulties in linguistic processing and ocular motor errors. In contrast, the significantly smaller and more frequent saccades made by schizophrenic volunteers suggest that they are less able to incorporate textual information not currently being fixated. This could imply reduced sensitivity to parafoveal information, which was explored further in experiment 2 using the GCWT.

Ocular motor deficits have been previously described in schizophrenia particularly in relation to smooth pursuit and antisaccadic eye movements. One might postulate that the increase in number of saccades and decrease in saccadic amplitude may be a result of an inability to make normal saccades and correct fixation during text reading. However, forward saccades have been found to be normal in schizophrenia and we found no significant differences in fixation duration parameters. It is possible that the schizophrenic subjects have difficulty in suppressing irrelevant or small words usually skipped during normal reading, similar to the difficulty they have suppressing a reflexive saccade during antisaccadic tasks. This suppression requires input from the frontal eye fields and dorsolateral prefrontal cortex, which have been implicated in schizophrenia.

**Experiment 2: GCWT**

Using the GCWT, we demonstrated that disruption in the amount of parafoveal text available to the right of fixation had a significant effect on reading rates in the control group but not in the schizophrenic group in accordance with our a priori hypothesis. Based upon these results, we postulate that reading deficits in schizophrenia are due to impaired sensitivity to parafoveal visual information leading to reduced reading speed and abnormal ocular motor patterns. This hypothesis is corroborated by the recent findings of Knebel et al who showed deficits in early parafoveal visual processing in schizophrenia using VEPs. They observed that the P1 VEP response is strongly modulated in controls with the extent of parafoveal stimulation but not in schizophrenia. Schizophrenic patients respond to wider stimulation of the visual field as if a narrow visual field stimulus is being applied. In addition using source estimation of the VEP data, Knebel et al also showed that this was associated with differences in activity in the left precuneus and middle inferior parietal cortex, areas which correspond to the dorsal visual pathway. These pathways could also underlie the reading deficits observed in schizophrenia.

Previous investigations using GCWT experiments have effectively demonstrated the preview benefit of seeing text in the parafoveal vision before processing the word with foveal vision. Not only does parafoveal information guide the eyes where to move to next in the text but experiments reveal that orthographic, phonological, and morphological information are all derived from parafoveal vision (recently reviewed in Schotter et al). Consequently, reduced sensitivity to parafoveal information in schizophrenia could indirectly contribute to other reading deficits described such as phonological processing and comprehension difficulties. These areas require further investigation.

Reduced perceptual spans have also been reported in dyslexic readers, in the elderly, in children, and also in normal slow readers and have been attributed to greater processing resources being allocated to the currently fixed word leaving less attentional resources available for visual processing of text to the right of fixation. Ocular motor patterns associated with these conditions are quite different to those observed in schizophrenia, however. For example, normal older readers appear to adopt a riskier strategy to reading compared with young readers resulting in longer saccades to new words and more frequent regressions. Normal slow readers in contrast make shorter saccades, longer fixations, with more regressions compared with fast readers. The normal fixation durations observed in schizophrenia argues against greater attentional resources being allocated to currently fixated words. Indeed, for the smaller window size in experiment 2, fixation duration was shorter in schizophrenia compared with controls implying that schizophrenics were less distracted by the visual limitation imposed by the task.

**Comparison of Schizophrenia and Developmental Dyslexia**

Using a series of standardized psychoeducational reading tests, Revheim et al have shown that schizophrenia results specifically in deficits during “real-world” reading tasks (ie, reading of text passages) rather than single-word reading. They also demonstrated that these reading deficits were strongly correlated to magnocellular pathway dysfunction (the pathway processing visual motion and spatial location rather than fine detail and form), a feature which is shared with dyslexics. Accordingly, Revheim et al found that 21%–63% of schizophrenic patients met the criteria for dyslexia depending on which diagnostic model was used. Arnott et al have also
Table 2. Comparison of Ocular Motor Characteristics in Schizophrenia (ie, Findings From Experiment 1) and in Developmental Dyslexia as Described in 6 Previous Studies,

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Schizophrenia % Difference</th>
<th>Developmental Dyslexia % Difference (Minimum, Maximum)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reading rate</td>
<td>-23.33</td>
<td>-41.38 (-64.4, -23.8)</td>
</tr>
<tr>
<td>Fixation duration</td>
<td>1.89</td>
<td>20.89 (5.63, 42.9)</td>
</tr>
<tr>
<td>Number of saccades/fixations per sequence</td>
<td>36.02</td>
<td>62.59 (3.74, 150.0)</td>
</tr>
<tr>
<td>Frequency of regressions</td>
<td>3.50</td>
<td>19.59 (-3.12, 55.6)</td>
</tr>
<tr>
<td>Saccadic amplitude</td>
<td>-14.21</td>
<td>-32.97 (-49.0, -14.1)</td>
</tr>
</tbody>
</table>

*Note:* Mean percentage difference from control values are shown where negative values indicate the parameter is less in schizophrenia compared with controls. The range of outcomes (maximum and minimum) described by the 6 studies are also shown for each parameter.

recently linked phonological processing deficits in schizophrenia to reading impairments, especially the ability to retrieve phonological information from the long-term memory (also called rapid naming). Phonological processing impairments are also strongly implicated as causing dyslexia.

Table 2 summarizes the ocular motor characteristics in schizophrenics recorded in experiment 1 and the data from 6 previous studies investigating developmental dyslexia. The table shows the percentage difference in each parameter in relation to their respective control groups. In general, reading deficits are more severe in developmental dyslexia leading to greater differences to controls for all parameters compared with schizophrenia. There are, however, specific differences between the ocular motor patterns in schizophrenia and dyslexia, which might imply that these are 2 distinct disorders. In particular, schizophrenic readers do not show abnormally long fixations or more regressions compared with dyslexic readers. This is in agreement with previous studies that schizophrenics do not have difficulty decoding printed material and show similar success at reading single words compared with controls. It should be noted, however, that a higher regression rate was seen in experiment 2 during FS viewing in schizophrenia. This could be due to a smaller sample size used for experiment 2 and perhaps the idiosyncratic nature of this parameter.

It is possible, however, that ocular motor patterns seen in schizophrenia are a mild form of that seen in developmental dyslexia because fixation duration and frequency of regressions are also the least affected parameters in developmental dyslexia. Maximum and minimum differences between dyslexia and controls for the 6 studies shown in table 2 illustrate the variability of findings from previous studies and imply that only tentative conclusions can be drawn from the comparing the ocular motor findings in schizophrenia and developmental dyslexia.

As far as we are aware the GCWT has not been performed on a cohort of patients with developmental dyslexia to allow comparison of findings from experiment 2. However, the method has been used to investigate individuals with specific forms of dyslexia, namely letter-by-letter acquired dyslexia and selective attentional dyslexia, which both show severe forms of disruption to the parafoveal visual processing.

Several studies have shown that developmental dyslexia is associated with poor binocular control evident from disconjugacy of saccades, leading to fixation disparity and fixational instability. No significant difference in disconjugacy of saccades was observed between the schizophrenia and the control group in this study.

**Possible Confounders**

Although schizophrenic volunteers in this study appeared to have no difficulty in understanding the text (all scored 98.6% or higher in the MCQ) and we used control groups that were equivalent in NART score (a measure of premorbid IQ), it would be difficult to rule out the possibility that the reading deficits we observed in the schizophrenic group could be due to cognitive deficits. Cognitive functions such as working memory, attention, and visual learning are all known to be impaired in schizophrenia. Determining the association between reading speed, visual integration, and cognitive function is outside the remit of this study but warrants further investigation.

Out of 38 subjects, 35 were on atypical medications. It has been suggested that antipsychotic medication might affect eye movements. The dopamine receptor antagonist properties of antipsychotic medication can cause Parkinson’s-like symptoms and disrupt ocular motor movements. Atypical antipsychotic medications can also have anti-dopaminergic effects. Reports have shown that drug withdrawn schizophrenic patients still show eye movement abnormalities suggesting the deficits are due to the underlying neuropathology of the condition rather than medication. The absence of any significant correlation between daily chlorpromazine dose equivalent and any of the ocular motor parameters argues against medication being a confounding factor as does the observation of schizophrenia-like ocular motor patterns in unmedicated relatives of the patients.
It is not likely that differences in reading were due to poor vision as all subjects had a VA of better than 6/9.5 which is more than sufficient to see the text which was equivalent to a VA of 6/36.

**Use of Reading as a Marker for Schizophrenia**

The clinically healthy relatives showed eye movement abnormalities during reading suggesting a genetic component and that reading could possibly be used as a genetic marker. Ho et al. assessed premorbid cognitive tests for early detection of schizophrenia in 217 adolescents and found standardized scholastic tests can enhance the detection of schizophrenia by 3- to 5-fold. This is complementary to similar suggestions in relation to smooth pursuit and antisaccades where relatives show increased errors and decreased gain during antisaccades and increased frequency of saccades and decreased gain during smooth pursuit. Interestingly, the reading performance of relatives was intermediate in relation to the schizophrenic and control subjects in the smooth pursuit and antisaccadic tasks. This may be due to genetic inhomogeneity, ie, inclusion of relatives who have no genetic predisposition. The statistical analysis revealed no heritability of ocular motor traits between schizophrenic volunteers and their relatives although the sample size was relatively small (n = 11). The small sample size may also explain the absence of significant differences in schizotypal symptoms between relatives and controls.

As well as overall reading performance, ocular motor reading parameters could also potentially be used for the early detection of schizophrenia. We found that the number of saccades made per line (sensitivity = 73%, specificity = 60%, and positive predictive value = 61.5%) was a small improvement over reading rate (sensitivity = 73%, specificity = 56%, and positive predictive value = 59.3%) for detection of schizophrenia. However, to objectively demonstrate the benefits of using an analysis of ocular motor reading parameters for the early detection of schizophrenia would require a study with a much larger sample size.

**Summary**

These findings suggest that deficits in early stages of visual processing in schizophrenia can work out into functional visual deficits in reading that could directly impact upon quality of life. Poor scholastic performance has been shown in premorbid schizophrenic adolescents. It is possible that these teenagers have abnormal reading patterns before the disease is diagnosed. It would be interesting to determine if adolescents at risk of the disease (relatives of schizophrenic patients) show similar reading problems. Reading support and increased reading times could be provided in schools to those at risk of the disease to improve poor scholastic performance associated with schizophrenia and possibly overall outcome of disease.

In this regard, it would be important to establish the link between schizophrenia and dyslexia through future investigations.

**Supplementary Material**

Supplementary material is available at http://schizophreniabulletin.oxfordjournals.org.

**Funding**

Ulverscroft Foundation.

**Acknowledgments**

The authors have declared that there are no conflicts of interest in relation to the subject of this study.

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