Reading Impairments in Schizophrenia Relate to Individual Differences in Phonological Processing and Oculomotor Control: Evidence From a Gaze-Contingent Moving Window Paradigm

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Reading Impairments in Schizophrenia Relate to Individual Differences in Phonological Processing and Oculomotor Control: Evidence From a Gaze-Contingent Moving Window Paradigm

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Language and oculomotor disturbances are 2 of the best replicated findings in schizophrenia. However, few studies have examined skilled reading in schizophrenia (e.g., Arnott, Sali, Copland, 2011; Hayes & O’Grady, 2003; Revheim et al., 2006; E. O. Roberts et al., 2012), and none have examined the contribution of cognitive and motor processes that underlie reading performance. Thus, to evaluate the relationship of linguistic processes and oculomotor control to skilled reading in schizophrenia, 20 individuals with schizophrenia and 16 demographically matched controls were tested using a moving window paradigm (McConkie & Rayner, 1975). Linguistic skills supporting reading (phonological awareness) were assessed with the Comprehensive Test of Phonological Processing (R. K. Wagner, Torgesen, & Rashotte, 1999). Eye movements were assessed during reading tasks and during nonlinguistic tasks tapping basic oculomotor control (prosaccades, smooth pursuit) and executive functions (predictive saccades, antisaccades). Compared with controls, schizophrenia patients exhibited robust oculomotor markers of reading difficulty (e.g., reduced forward saccade amplitude) and were less affected by reductions in window size, indicative of reduced perceptual span. Reduced perceptual span in schizophrenia was associated with deficits in phonological processing and reduced saccade amplitudes. Executive functioning (antisaccade errors) was not related to perceptual span but was related to reading comprehension. These findings suggest that deficits in language, oculomotor control, and cognitive control contribute to skilled reading deficits in schizophrenia. Given that both language and oculomotor dysfunction precede illness onset, reading may provide a sensitive window onto cognitive dysfunction in schizophrenia vulnerability and be an important target for cognitive remediation.

Keywords: reading, eye movements, schizophrenia, perceptual span
Pugh & McCardle, 2009) and the maintenance of skilled reading in adults (e.g., Jared & Seidenberg, 1991; Perfetti & Bell, 1991; Pollatsek, Lesch, Morris, & Rayner, 1992; Pollatsek, Reichle, & Rayner, 2006; Rayner, Sereno, Lesch, & Pollatsek, 1995; Reichle, Pollatsek, Fisher, & Rayner, 1998; Reichle, Rayner, & Pollatsek, 2003). Not surprisingly, impaired phonological skills are thought to play a role in reading disorders such as dyslexia (Hatcher, Hulme, & Ellis, 1994; Pugh & McCardle, 2009; Rack, 1994).

Skilled reading also involves the programming and execution of spatially and temporally precise eye movements (i.e., saccades), which bring printed material into foveal view at a self-driven pace (reviewed in Livermore, Gilchrist, & Everling, 2011; Rayner, 1998, 2009; Rayner et al., 2012). Pauses between saccades (i.e., fixations) allow for the extraction of linguistic information; the frequency and duration of these fixations are modulated by linguistic variables such as word length, word frequency, and contextual predictability (reviewed in Rayner, 1998, 2009). Linguistic variables, such as word length, also interact with oculomotor programming by affecting where the eyes first land on words (Pollatsek et al., 2006; Reichle et al., 1998, 2003). For example, in skilled readers, landing position on individual words is normally midway between the beginning and middle of a word (e.g., Dunn-Rankin, 1978; McConkie, Kerr, Reddix, & Zola, 1988; Rayner, 1979), which optimally capitalizes on word-initial information crucial for lexical processing.

Abnormalities in both language and oculomotor control are well documented in individuals with schizophrenia (e.g., Kuperberg, 2010a, 2010b; Levy et al., 2010; Li et al., 2009). Language abnormalities in schizophrenia that are relevant to reading include greater spreading activation or poor inhibitory control during lexical–semantic processing (e.g., Gouzoualis-Mayfrank et al., 2003; Kuperberg, 2010a, 2010b; Spitzer, 1997; Titone, Holzman, & Levy, 2002; Titone, Levy, & Holzman, 2000) and impaired phonological or speech-based processing (e.g., Angrilli et al., 2009; Baruch & Csermaksny, 2007; Cienfuegos, March, Shelley, & Javitt, 1999; Kasai et al., 2002; Revheim et al., 2006; Titone & Levy, 2004; Wexler, Stevens, Bowers, Sernyak, & Goldman-Rakie, 1998), which could impact grapheme-to-phoneme word decoding. Abnormalities in oculomotor control have also been well documented in schizophrenia (e.g., Clementz, McDowell, & Zisook, 1994; Gooding & Basso, 2008; O’Driscol & Callahan, 2008; Sereno & Holzman, 1995). These include reduced predictive saccade amplitudes (Clementz et al., 1994), increased antisaccade errors (Gooding & Basso, 2008; Sereno & Holzman, 1995), and low-velocity smooth pursuit with elevated saccade frequencies (O’Driscoll & Callahan, 2008).

It is striking that so few studies have investigated skilled reading in schizophrenia, given the deficits in language processing and oculomotor control and also the functional consequences of poor reading skills with respect to quality of life in both healthy populations and populations with chronic mental illness (Carpenter et al., 2000; Christensen & Grace, 1999; Christopher, Foti, Roy-Bujnowski, & Appelbaum, 2007; Gold, Goldberg, McNary, Dixon, & Lehman, 2002; Green & Riddell, 2007; McGurk & Meltzer, 2000; Revheim et al., 2006; Sentell & Skumway, 2003; Sticht, 1988). To this end, understanding the nature of any reading deficit in schizophrenia is crucial for developing remediation strategies that have the potential to directly improve quality of life.

Existing studies on reading in schizophrenia may be divided into those that have investigated single-word reading measures and those that have investigated more complex reading measures. The consensus of studies using measures of single-word reading, such as the National Adult Reading Test (NART; Nelson, 1982), is that reading is preserved in schizophrenia (e.g., Dalby & Williams, 1986; O’Carroll et al., 1992). However, retrospective studies using more complex reading measures show poor childhood (pre-illness) reading history in people with schizophrenia (Ambelas, 1992; Crow, Done, & Sacker, 1995; Fuller et al., 2002). For example, in male adolescents recruited by the Israeli Draft Board (n = 365,020), poor reading was associated with an increased incidence of schizophrenia later in life (Weiser et al., 2004; see also Reichenberg et al., 2002; Weiser et al., 2007).

More recent studies (Arnot, Sali, & Copland, 2011; Hayes & O’Grady, 2003; Revheim et al., 2006) have reported schizophrenia-related reading deficits using standardized assessment measures such as the Nelson-Denny Reading Test (NDRT; Brown, Fishco, & Hanna, 1993) or the Comprehensive Test of Phonological Processing (CTOPP; R. K. Wagner, Torgesen, & Rashotte, 1999). Compared with controls, people with schizophrenia exhibit reduced reading rates (Arnot et al., 2011; Hayes & O’Grady, 2003; Revheim et al., 2006), poorer reading comprehension (Arnot et al., 2011; Hayes & O’Grady, 2003; Revheim et al., 2006), and impaired phonological awareness (Revheim et al., 2006). Of note, one study found that between 20% and 60% of their participants with schizophrenia met diagnostic criteria for dyslexia, depending on the threshold used (Revheim et al., 2006).

Schizophrenia and dyslexia are fundamentally distinct in their clinical presentation and functional outcome; however, there are several commonalities between the disorders in terms of etiology and cognitive or perceptual deficits (Condray, 2005). For example, genes implicated in reading disorder account for significant variation in brain volumes in schizophrenia (Jamadar et al., 2011). Similarly, volumes of brain areas implicated in dyslexia are associated with reading comprehension in schizophrenia (Leonard et al., 2008). Moreover, schizophrenia and dyslexia both involve impaired performance on measures of magnocellular function, such as contrast sensitivity and motion perception (e.g., Chen, Nakayama, Levy, Matthews, & Holzman, 1999; Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Livingstone, Rosen, Drislane, & Galaburda, 1991; Martín et al., 2008; Revheim et al., 2006; Talcott et al., 1998). Such deficits are associated with reduced reading proficiency in schizophrenia (Revheim et al., 2006). Moreover, impairments in smooth pursuit eye movements (e.g., Adler-Grinberg & Stark, 1978; Eden, Stein, Wood, & Wood, 1994; O’Driscol & Callahan, 2008; Pavlidis, 1981) and in antisaccades, an oculomotor measure of cognitive control (e.g., Bisaldi, Fischer, & Hartnegg, 2000; Gooding & Basso, 2008; Sereno & Holzman, 1995), have been widely reported in both groups. Thus, schizophrenia and dyslexia are associated with similar impairments in linguistic, phonological, visual, and oculomotor processes (Fuller et al., 2002; Leonard et al., 2008; Revheim et al., 2006).

In this study, we are particularly interested in the perceptual span during reading, which is the amount of parafoveal information that can be extracted at a single fixation. Perceptual span is optimally quantified using a gaze-contingent moving window paradigm, where text is presented normally in the foveal region but is
obscured by a pattern mask in the parafoveal region (McConkie & Rayner, 1975; Rayner & Bertera, 1979). The size of the window of normal text is manipulated: When it is smaller than the perceptual span of the reader, saccade lengths and reading speed are reduced, presumably because the missing information is normally used in natural reading. In skilled readers of left-to-right orthographies, the perceptual span is asymmetric, extending 3–4 characters to the left of fixation and 14–15 characters to the right of fixation (McConkie & Rayner, 1975, 1976; Rayner & Bertera, 1979; Rayner, Well, & Pollatsek, 1980). In unskilled readers (e.g., beginner readers, poor readers, and readers with dyslexia), the perceptual span to the right of fixation is generally smaller, presumably because they allocate more resources to foveal processing and less to parafoveal processing (Henderson & Ferreira, 1990). Thus, unskilled readers are less affected by larger reductions in window size, indicative of reduced perceptual span (Bélanger, Slattery, Mayberry, & Rayner, in press; Häikö, Bertram, Hyönä, & Niemi, 2009; Rayner, 1986; Rayner, Murphy, Henderson, & Pollatsek, 1989).

Although eye movement measures are a major component of reading research, only one study has assessed eye movements during reading in schizophrenia (E. O. Roberts et al., 2012). Thus, the aims of this report are threefold. The first is to determine whether eye movement measures of skilled reading differentiate people with schizophrenia from a sample of matched healthy controls. We hypothesized that people with schizophrenia exhibit eye movement behaviors that are well-established markers of reading difficulty. These include reduced forward saccade amplitudes, longer fixation durations, and more regressive saccades than in controls (Adler-Grinberg & Stark, 1978; Hutzler & Wimmer, 2004; Jones, Kelly, & Corley, 2007; Pavlidis, 1978; Rayner, 1985, 1986, 1998, 2009; E. O. Roberts et al., 2012).

The second aim is to determine whether people with schizophrenia have reduced perceptual spans compared with controls for sentence-level text. We hypothesized that low-level difficulties in skilled reading in schizophrenia (e.g., difficulties with phonological awareness and grapheme-to-phoneme conversion) increase foveal processing load, thereby reducing parafoveal information processing during fixation (Henderson & Ferreira, 1990; Rayner, 1985, 2009). Further, reduced parafoveal processing during normal reading, in turn, translates to a smaller perceptual span during reading. Thus, experimentally reducing the amount of parafoveal text available during reading should have a reduced impact on individuals with schizophrenia compared with controls. A recent study by E. O. Roberts et al. (2012) provided some evidence for this hypothesis: Perceptual span reductions in schizophrenia were found when people read paragraphs that extended over several pages of text. However, given that individuals with schizophrenia have impaired discourse processing (e.g., Ditman & Kuperberg, 2007, 2010), part of the reduction could relate to a reduced ability to integrate information across sentences. Here, we assess perceptual span in schizophrenia under conditions that are relatively undemanding at the discourse level, that is, during syntactically simple, single-sentence reading.

Our third aim is to examine measures of reading performance (perceptual span, comprehension) in relation to the cognitive and motor processes that support reading, a hypothesis that has not been investigated previously. One hypothesis was that perceptual span during reading in schizophrenia is linked to measures of phonological processing, which is a primary source of reading impairment in people with dyslexia (Hulme, Snowling, Caravolas, & Carroll, 2005; Pugh & McCardle, 2009; Rack, 1994). A second hypothesis was that reading measures in schizophrenia are related to measures of basic oculomotor control that are impaired in schizophrenia. A third hypothesis was that reading measures in schizophrenia are related to measures tapping the strategic control of eye movements. The strategic control of eye movements would seem to be critical to allow saccade length and frequency to be modulated by linguistic variables during reading. Thus, we hypothesized that oculomotor tasks that tap the ability to voluntarily initiate and withhold initiation of saccades (predictive saccades, antisaccades) relate to reading performance.

Method

Participants

Twenty outpatients (16 male, 4 female) who met criteria for schizophrenia according to the Diagnostic and Statistical Manual of Mental Disorders (4th ed.; DSM–IV; American Psychiatric Association, 1994) were tested. Diagnosis of schizophrenia was confirmed through the patient version of the Structured Clinical Interview for DSM–IV (SCID) Axis I disorders (First, Spitzer, Gibbon, & Williams, 1996) and through chart review. Patients were clinically stable, with no change in medication dose for at least 4 weeks prior to testing. Seventeen patients were receiving atypical neuroleptic treatment, and three patients were not receiving neuroleptic treatment at the time of testing. Medicated patients were receiving an average chlorpromazine equivalent dose of 443.57 mg/day (±277.55 mg/day). Current symptoms were rated using the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962), with an average total score of 53.05 (±11.78). Patients’ average illness duration was 10.85 years (±9.43 years). Ten patients were native English speakers, and 10 patients were native French speakers.

Inclusion criteria included estimated verbal IQ greater than 80 (based on the Vocabulary subtest of the Wechsler Adult Intelligence Scale—Revised [WAIS–R]; Wechsler, 1981), having English or French as the first acquired and currently dominant language (based on a language questionnaire modeled after the Language Proficiency and Experience Questionnaire (Marian, Blumenfeld, & Kaushanskaya, 2007), and being within ages 18–50 years. Exclusion criteria included history of neurological impairment (other than schizophrenia), current substance abuse or history of substance dependence within 4 weeks prior to testing, current use of drugs that affect saccade velocities (e.g., benzodiazepines, chloral hydrate), and visual deficiencies (e.g., uncorrected deficits in visual acuity). Visual acuity was assessed using the Snellen chart, with a minimum criterion of 20/40 vision at a viewing distance of 20 feet (6 m).

Sixteen nonpsychiatric controls (13 male, 3 female) were tested. Controls were matched to patients on gender, language background, age, WAIS–R Vocabulary subtest scores, and parental socioeconomic status (SES) based on parental occupation, ranked on an ordinal scale from 1 (major professional) to 9 (unemployed) using the Hollingshead Occupational Scale (Hollingshead, 1975). Controls did not significantly differ from patients on age (31.05 vs. 31.56 years, respectively; p = .87), scaled WAIS–R Vocabulary subtest scores (12.75 vs. 10.83; p = .10), or parental SES (3.95 vs. 3.10).
3.87, respectively; \( p = .74 \)). All controls were screened with the nonpatient version of the SCID (First et al., 1996) and were excluded for current history of Axis I disorders. Ten controls were native English speakers, and six controls were native French speakers. Characteristics of the participant groups are presented in Table 1.

Patients were recruited through McGill University-affiliated outpatient services (e.g., Douglas Hospital, Montreal, Quebec, Canada); thus, all patients were living independently in the community. Controls were recruited from the larger Montreal community. All participants provided informed consent after the study was fully explained to them, and they were compensated $18/hr.

Materials

Gaze-contingent moving window task. We used the classic moving window paradigm (McConkie & Rayner, 1975; Rayner & Bertera, 1979). This paradigm allowed us to examine both global aspects of reading performance (e.g., forward fixation duration, forward saccade duration) and perceptual span (i.e., the amount of textual material encoded at each fixation).

Text materials consisted of 90 short, syntactically simple sentences distributed across five moving window conditions. One set of materials was created in English and another in French, as the study included both native English and native French readers. Given that the sentences were syntactically simple, we ensured that the English and French versions were comparable by obtaining direct translations of each other (see Table 2 for sample sentences). The sentences were coded for several linguistic variables such as total number of words, word length, and frequency. The English sentences had an average of 10 words and an average word length of 4.38 characters. English word frequencies were obtained from the Kucˇera and Francis (1967) corpus of the English Lexicon Project (Balota et al., 2007), with an average word frequency of 4,572 parts per million. The French sentences had an average of 11 words and an average word length of 4.33 characters. French word frequencies were obtained from the LEXIQUE database (New, Pallier, Ferrand, & Matos, 2001), with an average word frequency of 5,538 parts per million. The English and French sentences were matched on word frequency and average word length (all \( ps > .36 \)). All materials were exclusively presented in participants’ native and most dominant language, either English or French.

The experimental sentences were divided into five moving window conditions (75 sentences in total; 15 sentences per condition) that manipulated the amount of parafoveal information available at each fixation in a gaze-contingent fashion: four conditions consisting of progressively wider windows to the right of fixation and one no-window (full text) condition (window size to the left of fixation was fixed at four characters). The narrowest window condition was 2 characters to the right of fixation, then 6 characters, 10 characters, 14 characters, and finally, the no-window (full text) condition. During fixation, text was presented normally within the window; however, beyond the window of normal text, characters and spaces were replaced by dashes. Sample sentences in each condition are presented in Table 3. There were 15 practice sentences in total (three sentences per window condition). All sentences were matched on total number of words, word length, and frequency across the five window conditions (all \( ps > .28 \)).

Based on prior work using this task with healthy readers (e.g., Rayner, 1986), we expected the 14-character window to be comparable to the full-text condition for controls; in smaller windows, reading performance (assessed by the number of words read per minute) was expected to systematically decrease as window size decreased. For patients, our hypothesis was that they would be less
affected by reductions in window size; thus, the results for the 14, 10, and possibly 6-character windows would be identical to that for the full-text condition.

**Standardized reading tests.** Following Revheim et al. (2006), we administered a battery of standardized reading tests, including the core subtests of the CTOPP (R. K. Wagner et al., 1999) and the Comprehension and Reading Rate subtests of the NDRRT (Brown et al., 1993).

The CTOPP assesses three fundamental components of phonological processing: phonological awareness (i.e., knowledge of the sound structure of words) via the Elision and Blending Words subtests; phonological memory (i.e., coding and storage of phonological information in short-term memory) via the Memory for Digits and Nonword Repetition subtests; and rapid naming (i.e., prompt, efficient retrieval of phonological information from long-term memory) via the Rapid Digit Naming and Rapid Letter Naming subtests. Raw subtest scores are converted to standard scores, which are then converted to three standard composite scores (i.e., the three components of phonological processing), with a mean of 100 and a standard deviation of 15. Although the CTOPP was developed using norms from American English speakers ages 7–24, a comparable French version suitable for French speakers in Quebec was used (Bélard & Hébert, 2009; unpublished, adapted version of the CTOPP).

The NDRRT Comprehension subtest consists of silent passage reading (seven in total), followed by comprehension questions. The NDRRT Reading Rate subtest assesses the number of words read during the first minute of passage reading. Raw scores are converted to scaled scores. Although the NDRRT was developed using norms from American English speakers, a comparable French version suitable for French speakers in Quebec was used (available upon request).

**Eye movement recording tests of basic oculomotor control and executive functions.** Two tasks were administered to assess basic oculomotor control: a prosaccade task and a smooth pursuit task. Two additional eye movement tasks were administered to assess executive functions: a predictive saccade task (a measure of oculomotor planning) and an antisaccade task (a measure of oculomotor inhibition/cognitive control).

In the prosaccade task, participants fixated a central target (0.5° by 0.5° of visual angle) on a computer screen. After 800 to 1,400 ms, a peripheral target (0.5° by 0.5° of visual angle) appeared 11° to the left or right of the central target. The direction of the peripheral target was pseudorandomized such that the target could not move in the same direction on more than three consecutive trials. Participants were instructed to look toward the peripheral target as quickly as possible when it appeared on the screen but to look in the opposite direction as quickly as possible (i.e., toward the mirror position on the opposite side of the computer screen). Nine practice trials were completed to ensure task comprehension, followed by 48 experimental trials. Each trial lasted a maximum of 2,000 ms. Percentage of errors (i.e., first saccades exceeding 2° in the direction of the target), saccade amplitude, and saccade latency were examined.

**Apparatus**

**Gaze-contingent moving window task.** Eye movements were recorded with an EyeLink 1000 desktop-mounted system, with a sampling rate of 1 kHz, spatial resolution of 0.01°, and mean accuracy of 0.25° (SR-Research, Mississauga, Ontario, Canada). Although viewing was binocular, eye movements were recorded from the right eye only. Eye movements were calibrated using a 5-point grid. The average fixation error on validation was less than 0.4° of visual angle. A drift correction point was presented before the onset of each sentence to ensure tracking accuracy. Head movements were minimized by padded chin- and head-rests. Sentences were presented on a 21-in. ViewSonic cathode ray tube (CRT) monitor with a screen resolution of 1024 × 768 pixels and a refresh rate of 144 Hz. The monitor was positioned 57 cm from participants. Sentences were presented in yellow 11-point Courier New font (due to equidistant character spacing) on a black background using Eyetack software (Version 0.7.10g) developed at the University of Massachusetts Amherst (http://www.psych.umass.edu/eyelab/software). All sentences were displayed on a single line, with a maximum of 75 characters and with 3.2 characters subtending 1° of visual angle. The display change delay following eye movements was 8.7 ms; thus, perception of
window movement was synchronized with participant eye movements.

**Eye movement recording tests of basic oculomotor control and executive functions.** Eye movements were recorded in a darkened room with an EyeLink II headband-mounted system, with a sampling rate of 250 Hz, spatial resolution of 0.01°, and mean accuracy of 0.25° (SR-Research, Ontario, Canada). Although viewing was binocular, eye movements were recorded from the dominant eye only. Eye movements were calibrated using a 3-point horizontal line. The average fixation error on validation was less than 0.4° of visual angle. A drift correction point was presented between each trial to ensure tracking accuracy. The oculomotor tasks were presented on a 19-in. ViewSonic CRT monitor, with a screen resolution of 1024 × 768 pixels and a refresh rate of 120 Hz. The monitor was positioned 57 cm from participants.

**Procedure**

All participants were tested in two 3-hr sessions separated by no more than 2 weeks. Participants were given breaks during each session. Clinical and demographic information were collected first, and then the experimental tasks (i.e., moving window task, standardized reading tests, and oculomotor tasks) were presented in a random order.

Regarding the moving window task, the 15 experimental sentences per window condition were presented in a single block across the five window conditions. To avoid practice effects, the order of the blocks was randomized using three separate lists. The order of the lists was counterbalanced across participants. Participants were informed that most sentences would be partially masked by dashes and that they should read the sentences silently and at their normal pace for comprehension despite the dashes. Following Rayner (1986), participants were also instructed that they should try to read each sentence only once (i.e., they should avoid rereading sentences), unless they had difficulty understanding the content of a sentence.

The onset of each trial was initiated by fixing on a yellow, gaze-contingent box located before the first word of each sentence. To become familiarized with the task, participants first read the 15 practice sentences, starting with the no-window (full text) condition, followed by conditions of progressively narrower window sizes. Participants then read the 75 experimental sentences. Participants pressed a button on a controller pad after reading each sentence. To ensure that participants maintained attention while reading, simple yes/no comprehension questions appeared on 20% of the experimental trials. The comprehension questions were evenly distributed across the five moving window conditions.

Regarding the oculomotor tasks, order of presentation was pseudorandomized across participants, and order of completion of antisaccade and prosaccade tasks was counterbalanced across participants.

**Data Analytic Procedure**

Eye movement measures examined included reading rate (number of words read per minute, starting with initial sentence presentation and ending with sentence completion via button press), mean forward saccade length (number of characters), mean forward fixation duration (ms), and number of regressive saccades (backward saccades).

The eye movement data were analyzed using linear mixed effects (LME) models within the lme4 package (Bates & Sarkar, 2007) of R (Version 2.13.1; Baayen, Davidson, & Bates, 2008; R Development Core Team, 2010). LME models offer several analytical advantages over standard analyses of variance: Trial-by-trial data are used as input, so there is no loss of information by averaging over participants and items; statistical outliers are less influential; and statistical power is increased while simultaneously accounting for heterogeneity of variance from multiple random effects variables (e.g., participants, items; Baayen et al., 2008; Quéné & van den Bergh, 2008). Markov chain Monte Carlo (MCMC) sampling tests (n = 10,000) were used to obtain p values for fixed factors in all models.

**Results**

Each participant’s sentence data were first examined for track loss (e.g., blinks) using EyeDoctor software developed at the University of Massachusetts Amherst (http://www.psych.umass.edu/eyelab/software). All blinks were excluded, resulting in 1.2% data loss. Fixations less than 80 ms in duration and within one character of another fixation were combined (0.6% of fixations). All other fixations less than 80 ms in duration were excluded, resulting in 2.3% data loss. Saccades were identified using the SR-Research saccade detection algorithm: minimum velocity of 30°/sec, minimum acceleration of 8000°/sec², and minimum change in eye position of 0.15°.

**Eye Movement Differences Between Groups for Full-Text Reading**

To address the first aim of this report, that is, assessing whether there are differences between the participant groups in eye movement measures of normal reading, the same LME model was applied to each eye movement measure, drawing from the no-window (full text) condition. Participants and items were random factors (random intercepts only) and clinical status (treatment coded: controls vs. schizophrenia patients; controls = baseline) was a fixed factor. Several control predictors were also included to statistically control for variance due to potential effects of age (continuous), participant native language (treatment coded: English vs. French; English = baseline), years of education (continuous), chlorpromazine equivalent dose (continuous), and trial number (continuous). Maximum correlations among main effects were under 0.34 for each model, suggesting minimal influence of colinearity. The data, averaged over sentences for each group, are presented in Table 4.

An effect of clinical status was found for all eye movement measures (see Table 5). Relative to controls, schizophrenia patients read fewer words/minute (b = -45.56, SE = 17.44, p_{MCMC} = .0012; 138.84 vs. 208.02 words/minute, respectively), made shorter forward saccades (b = -1.27, SE = 0.76, p_{MCMC} = .0180; 6.89 vs. 8.71 characters, respectively), made longer forward fixation durations (b = 36.68, SE = 13.33, p_{MCMC} = .0002; 240.93 vs. 201.59 ms, respectively), and made more regressive saccades than did controls (b = 0.83, SE = 0.44, p_{MCMC} = .0158; 2.45 vs. 1.36 saccades, respectively). Moreover, patients had significantly lower
sentence comprehension performance than did controls (84% vs. 88% accuracy, respectively; \( p \leq .05 \)). Thus, during normal reading, individuals with schizophrenia exhibited eye movement behaviors that are robust markers of reading difficulty. Importantly, these effects were not driven by individual differences in medication status, as chlorpromazine equivalent dose never significantly contributed to the models (all \( p \geq .11 \)).

### Perceptual Span in Controls and Patients

To address the second aim of this report, that is, assessing whether people with schizophrenia have reduced perceptual spans compared with controls, separate LME models were first created for each participant group. In prior work (e.g., Rayner, 1986), perceptual span was estimated as the window size where reading rates decreased relative to the full-text condition. Accordingly, reading rate was our primary dependent variable. Participants and items were random factors (random intercepts only), and window size (treatment coded: full-text condition vs. 2-, 6-, 10-, and 14-character windows; full-text condition = baseline) was a fixed factor. The same control predictors included in previous models were included, except that chlorpromazine equivalent dose (continuous) was not included in the separate model for controls. The maximum correlation among main effects was under 0.32 for each model. Reading rate data as a function of window size for both participant groups are plotted in Figure 1.

#### Perceptual span in controls.

Relative to the no-window (full text) condition, reading rates decreased for all smaller window sizes, except the 14-character window (see Table 6). Reading rates were significantly slower in the 2-character versus no-window condition (\( b = -84.59, SE = 4.61, p_{MCMC} = .0001; 121.87 \) vs. 208.02 words/min, respectively), 6-character versus no-window condition (\( b = -12.96, SE = 4.83, p_{MCMC} = .0062; 187.93 \) vs. 208.02 words/min, respectively), and 10-character versus no-window condition (\( b = -12.06, SE = 4.60, p_{MCMC} < .0049; 200.30 \) vs. 208.02 words/min, respectively). In contrast, reading rates were faster in the 14-character versus no-window condition (\( b = 12.77, SE = 4.79, p_{MCMC} = .0064; 214.53 \) vs. 208.02 words/min, respectively). Consistent with prior work involving skilled readers, these findings suggest that the perceptual span in controls was roughly 14 characters to the right of fixation, as perceptual span did not decrease at this window size.

#### Perceptual span in patients.

Relative to the no-window (full text) condition, reading rates significantly decreased for the smallest window size only: 2-character versus no-window condition (\( b = -45.93, SE = 3.40, p_{MCMC} = .0001; 96.27 \) vs. 138.84 words/min, respectively; see Table 6). Reading rates did not decrease across the larger window sizes: 6-character versus no-window condition (\( b = 0.45, SE = 3.29, p_{MCMC} = .8942; 140.18 \) vs. 138.84 words/min, respectively), 10-character versus no-window condition (\( b = 5.89, SE = 3.32, p_{MCMC} = .0744; 146.68 \) vs. 138.84 words/min, respectively), and 14-character versus no-window condition (\( b = 6.64, SE = 3.31, p_{MCMC} = .0832; 148.07 \) vs. 138.84 words/min, respectively). Again, these effects were not driven by individual differences in medication status, as chlor-
promazine equivalent dose did not significantly contribute to the model \((p > .07)\). These results suggest that the perceptual span is smaller in patients than controls, with an estimated magnitude of less than 6 characters to the right of fixation.

**Group differences in perceptual span.** To verify group differences in perceptual span statistically, the same LME model was used to predict reading rates across participants with clinical status (treatment coded: controls vs. schizophrenia patients; controls = baseline) included as an additional fixed factor. The maximum correlation among main effects was under 0.40.

A main effect of clinical status was found \((b = -54.47, SE = 15.28, p_{MCMC} = .0001)\), where reading rates were slower for schizophrenia patients versus controls across all window sizes (134.62 vs. 187.21 words/min, respectively). Main effects of win-

**Figure 1.** Reading rate data (mean values) as a function of window size for controls and schizophrenia patients. Error bars represent standard error of the mean.

**Table 6**

<table>
<thead>
<tr>
<th>Linear mixed effects of reading rate</th>
<th>Controls ((n = 16))</th>
<th>Patients ((n = 20))</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fixed effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4L/14R condition</td>
<td>12.77, 4.79, .0064**</td>
<td>6.64, 3.31, .0832</td>
</tr>
<tr>
<td>4L/10R condition</td>
<td>-12.06, 4.60, .0094**</td>
<td>5.89, 3.32, .0744</td>
</tr>
<tr>
<td>4L/6R condition</td>
<td>-12.96, 4.83, .0062**</td>
<td>0.45, 3.29, .8942</td>
</tr>
<tr>
<td>4L/2R condition</td>
<td>-84.59, 4.61, .0001***</td>
<td>-45.93, 3.40, .0001***</td>
</tr>
<tr>
<td><strong>Control predictors</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (in years)</td>
<td>0.71, 8.51, .9052</td>
<td>-7.67, 7.36, .9331</td>
</tr>
<tr>
<td>Native language (b)</td>
<td>8.31, 18.11, .5914</td>
<td>29.70, 19.02, .0852</td>
</tr>
<tr>
<td>Education (in years)</td>
<td>4.94, 8.96, .5122</td>
<td>0.83, 7.37, .8798</td>
</tr>
<tr>
<td>Chlorpromazine dose (mg/day)</td>
<td>-0.05, 0.03, .0702</td>
<td></td>
</tr>
<tr>
<td>Trial order</td>
<td>0.37, 0.48, .1342</td>
<td>0.20, 0.15, .1274</td>
</tr>
<tr>
<td>(Intercept)</td>
<td>187.82, 11.97, .0001***</td>
<td>133.07, 11.87, .0001***</td>
</tr>
<tr>
<td><strong>Random effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subject</td>
<td>1,030.76</td>
<td>1,025.30</td>
</tr>
<tr>
<td>Item</td>
<td>149.72</td>
<td>121.70</td>
</tr>
<tr>
<td>Residual</td>
<td>2,486.82</td>
<td>1,612.80</td>
</tr>
</tbody>
</table>

**Note.** MCMC = Markov chain Monte Carlo; L = characters to the left of fixation; R = characters to the right of fixation.

\(a\) Contrasts were treatment-coded; model assumes no-window (full text) condition as the baseline across conditions. \(b\) Contrasts were treatment-coded (English vs. French); model assumes English as the baseline across conditions.

\(** p_{MCMC} < .01. \) \(*** p_{MCMC} < .001. \)
dow size were found where, relative to the no-window condition, reading rates were slower across participant groups in the 2-character window condition \((b = -85.09, SE = 4.13, p_{MCMC} = .0001; 107.62 \text{ vs. } 169.25 \text{ words/min, respectively})\), the 6-character window condition \((b = -15.01, SE = 4.21, p_{MCMC} = .0008; 161.34 \text{ vs. } 169.25 \text{ words/min, respectively})\), and the 10-character window condition \((b = -13.32, SE = 4.12, p_{MCMC} = .0009; 162.31 \text{ vs. } 169.25 \text{ words/min, respectively})\). Reading rates were not significantly slower in the 14-character window condition versus no-window condition.

Further, clinical status interacted significantly with all but the 14-character window condition, suggesting that patients were less affected by decreasing window sizes than were controls. Relative to the no-window condition, differences in reading rates were smaller in patients versus controls for the 2-character window condition \((b = 37.96, SE = 5.59, p_{MCMC} = .0001; 42.57 \text{ vs. } 86.16 \text{ words/min, respectively})\), the 6-character window condition \((b = 15.25, SE = 5.60, p_{MCMC} = .0050; -1.34 \text{ vs. } 20.09 \text{ words/min, respectively})\), and the 10-character window condition \((b = 8.60, SE = 5.54, p_{MCMC} < .0096; -7.83 \text{ vs. } 7.72 \text{ words/min, respectively})\). No differences between patients and controls were found for the 14-character window condition \((b = 2.12, SE = 5.55, p_{MCMC} = .7080; -12.23 \text{ vs. } -6.51 \text{ words/min, respectively})\).

As convergent evidence of these group differences in perceptual span, an alternate estimate of perceptual span used in prior work was considered: average forward saccade length (Rayner, 1986, 2009). Forward saccade length for the largest window sizes only (i.e., the no-window and 14-character conditions) was used as a proxy for perceptual span, as it provided a more informative estimate of individual differences that reflected the overall group effects for reading rate (words per minute) as a function of window size.\(^1\) Using this estimate, perceptual span was, again, larger for controls than for schizophrenia patients \((p = .0001; 8.58 \text{ vs. } 6.82 \text{ characters})\). Patients had approximately 69% of the perceptual span of controls for the reading rate estimate and 79% of the perceptual span of controls using the forward saccade length estimate.

### Relation of Perceptual Span Among Patients to Phonological Processing, Basic Oculomotor Control, and Executive Functions

To address the third aim of this report, that is, assessing the underlying bases for any perceptual span reductions in schizophrenia, associations between the forward saccade length estimate of perceptual span and measures of phonological processing, basic oculomotor control, and executive functions were examined in patients only. Thus, all models excluded controls.

**Perceptual span and phonological processing.** Performance on the standardized reading tests was significantly poorer in schizophrenia patients than in controls for all measures (see Table 7). Consistent with prior work (e.g., Revheim et al., 2006), patients had significantly lower CTOPP Phonological Awareness composite scores \((p = .0001)\), Phonological Memory composite scores \((p = .0001)\), and Rapid Naming composite scores \((p = .0015)\) than did controls. Patients also had significantly lower NDRT Comprehension and Reading Rate subtest scores than did controls \((p = .0001 \text{ and } .0002, \text{ respectively})\).

To examine the relation between perceptual span and phonological processing in schizophrenia patients only, separate LME models were created for the three CTOPP composite scores: Phonological Awareness, Phonological Memory, and Rapid Naming, as significant between-groups differences were found for these measures. Participants and items were random factors (random intercepts only), and the CTOPP composite scores were fixed factors. The same control predictors included in previous models were included. Perceptual span, hereafter quantified using the forward saccade length estimate (drawn from the no-window and 14-character window conditions), was the primary dependent variable. The maximum correlation among main effects was under 0.35 for all models.

Forward saccade length was significantly predicted by Phonological Awareness composite scores \((b = 0.07, SE = 0.04, p_{MCMC} = .0114)\), such that lower phonological awareness was associated with shorter forward saccade lengths (see Figure 2). Phonological Memory \((b = 0.02, SE = 0.02, p_{MCMC} = .3358)\) and Rapid Naming \((b = 0.01, SE = 0.08, p_{MCMC} = .7218)\) composite scores did not significantly predict forward saccade length.

---

\(^1\) The reading rate estimate of perceptual span was less stable at the individual level than was the forward saccade length estimate, since for each window size, it was based on 15 sentences with a single value from each sentence. As a result of the variability, the window size at which reading rate declined was not clear for several participants. In contrast, perceptual span based on forward saccade length data was based on an average of eight saccades per sentence for controls and 11 for patients, with 15 sentences for the two window sizes (i.e., the no-window and 14-character conditions). Thus, this measure of perceptual span was inherently more stable, being based on more data, and yielded for all participants a clear point at which window size affected performance.

### Table 7

<table>
<thead>
<tr>
<th>Measure</th>
<th>Controls (n = 16)</th>
<th>Patients (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CTOPP Phonological Awareness composite score</td>
<td>113.88 (3.07)**</td>
<td>97.00 (10.75)**</td>
</tr>
<tr>
<td>Elision Subtest standard score</td>
<td>11.25 (0.58)**</td>
<td>9.30 (1.69)**</td>
</tr>
<tr>
<td>Blending Words standard score</td>
<td>13.38 (0.72)**</td>
<td>9.85 (2.39)**</td>
</tr>
<tr>
<td>CTOPP Phonological Memory composite score</td>
<td>120.63 (4.50)**</td>
<td>103.60 (14.49)**</td>
</tr>
<tr>
<td>Memory for Digits standard score</td>
<td>13.38 (1.15)**</td>
<td>10.45 (3.43)**</td>
</tr>
<tr>
<td>Nonword Repetition standard score</td>
<td>13.38 (0.72)**</td>
<td>11.10 (1.65)**</td>
</tr>
<tr>
<td>CTOPP Rapid Naming composite score</td>
<td>113.06 (4.40)**</td>
<td>94.75 (16.77)**</td>
</tr>
<tr>
<td>Rapid Digit Naming standard score</td>
<td>12.56 (2.28)**</td>
<td>9.25 (2.49)**</td>
</tr>
<tr>
<td>Rapid Letter Naming standard score</td>
<td>11.88 (3.12)*</td>
<td>8.90 (3.67)*</td>
</tr>
<tr>
<td>NDRT Comprehension scaled score</td>
<td>230.88 (11.19)**</td>
<td>191.00 (25.14)**</td>
</tr>
<tr>
<td>NDRT Reading Rate scaled score***</td>
<td>220.19 (26.64)**</td>
<td>189.20 (17.29)**</td>
</tr>
</tbody>
</table>

*Note. CTOPP = Comprehensive Test of Phonological Processing; NDRT = Nelson-Denny Reading Test; MCMC = Markov chain Monte Carlo.

\(P_{MCMC} < .05. \quad \ast P_{MCMC} < .01. \quad \ast\ast P_{MCMC} < .001.\)
Thus, assuming that forward saccade length is a good proxy for perceptual span, reduced perceptual span in schizophrenia may be related to language-related deficits, primarily phonological awareness.

**Perceptual span and basic oculomotor control.** No group differences in basic oculomotor control (prosaccades, smooth pursuit) were found between patients and controls (all ps > .36; see Table 8). Prior work has shown that negative symptoms in schizophrenia are associated with anomalous oculomotor control (e.g., Katsanis & Iacono, 1991). Thus, the absence of group differences in smooth pursuit may be due to the lack of negative symptoms in the patient sample (average of BPRS negative subscales = 1.69 ± 0.54). The lack of between-groups differences in basic oculomotor control suggests that reduced forward saccade lengths in schizophrenia are not attributable to deficits in basic oculomotor control. Consequently, we did not examine the influence of basic oculomotor control on perceptual span reductions in schizophrenia.

**Perceptual span and executive functions.** Group differences were found in amplitudes of predictive saccades (p = .0003), with schizophrenia patients having smaller amplitudes than did controls (9.11° vs. 10.61° of visual angle, respectively; see Table 8). Group differences were also found on percentage of antisaccade errors (p = .0399), with schizophrenia patients making more errors than did controls (17.91% vs. 10.06%, respectively).

To assess the influence of individual variation in higher order oculomotor measures on forward saccade length in schizophrenia, separate LME models were created for the oculomotor variables with significant between-groups differences (i.e., amplitudes of predictive saccades and percentage of antisaccade errors). Again, the forward saccade length estimate was the primary dependent variable, drawing from the no-window and 14-character conditions. The control predictors were the same as those included in previous models. The maximum correlation among main effects was under 0.38 for each model.

**Predictive saccade task.** Amplitudes of predictive saccades were related to forward saccade length (b = 0.69, SE = 0.24, p_{MCMC} = .0001), with smaller predictive saccade amplitudes associated with smaller forward saccade lengths during reading (see Figure 3).

**Antisaccade task.** Percentage of antisaccade errors was not related to forward saccade length during reading (b = -0.02, SE = 0.02, p_{MCMC} = .0976).

### Table 8

<table>
<thead>
<tr>
<th>Control and measure</th>
<th>Controls (n = 16)</th>
<th>Patients (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prosaccade task</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Errors (%)</td>
<td>0.00 (0.00)</td>
<td>12.00 (55.00)</td>
</tr>
<tr>
<td>Amplitude (° of visual angle)</td>
<td>10.89 (0.55)</td>
<td>10.91 (1.15)</td>
</tr>
<tr>
<td>Latency (ms)</td>
<td>169.29 (17.97)</td>
<td>169.37 (20.31)</td>
</tr>
<tr>
<td>Smooth pursuit task</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pursuit gain (eye velocity/target velocity)</td>
<td>0.91 (0.10)</td>
<td>0.94 (0.04)</td>
</tr>
<tr>
<td>Total saccade rate</td>
<td>2.11 (0.76)</td>
<td>2.14 (0.65)</td>
</tr>
<tr>
<td>Predictive saccade task</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amplitude of visually guided saccades (° of visual angle)</td>
<td>10.80 (0.81)</td>
<td>10.39 (0.99)</td>
</tr>
<tr>
<td>Latency of visually guided saccades (ms)</td>
<td>118.78 (10.15)</td>
<td>124.80 (28.08)</td>
</tr>
<tr>
<td>Predictive saccades (%)</td>
<td>49 (25)</td>
<td>42 (26)</td>
</tr>
<tr>
<td>Amplitude of predictive saccades (° of visual angle)</td>
<td>10.61 (0.91)***</td>
<td>9.11 (1.23)***</td>
</tr>
<tr>
<td>Latency of predictive saccades (ms)</td>
<td>-44.26 (69.16)</td>
<td>-36.79 (39.83)</td>
</tr>
<tr>
<td>Antisaccade task</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Errors (%)</td>
<td>10.06 (15.44)*</td>
<td>17.91 (17.68)*</td>
</tr>
<tr>
<td>Amplitude (° of visual angle)</td>
<td>12.18 (2.23)</td>
<td>11.45 (3.34)</td>
</tr>
<tr>
<td>Latency (ms)</td>
<td>234.98 (33.90)</td>
<td>263.21 (56.85)</td>
</tr>
</tbody>
</table>

*Note.* MCMC = Markov chain Monte Carlo.

*p_{MCMC} < .05.*  *** p_{MCMC} < .001.
We also assessed whether any of our predictor variables were associated with reading comprehension of the sentences. We used simple linear regression analyses to evaluate the impact of predictor variables with significant between-groups differences (CTOPP composite scores and executive functions) on reading comprehension for the no-window and 14-character window conditions combined (i.e., the two conditions most likely to reflect normal reading processes). Only models that included percentage of errors on the antisaccade task, $F(1, 18) = 11.57, p = .0032$, adjusted $R^2 = 0.36$, significantly predicted sentence comprehension performance for the combined no-window and 14-character window conditions. No other measures (CTOPP composite scores and predictive saccade amplitudes) significantly predicted sentence comprehension performance. Thus, increased errors on the antisaccade task were associated with decreased reading comprehension (see left panel of Figure 4). Antisaccade errors also predicted reading comprehension on the NDRT (scaled scores): percentage of errors, $F(1, 18) = 11.17, p = .0036$, adjusted $R^2 = 0.35$ (see right panel of Figure 4).

**Discussion**

The purpose of this study was to investigate skilled reading in people with schizophrenia using a battery of tests that included a moving window paradigm (McConkie & Rayner, 1975), which manipulated parafoveal information in a gaze-contingent manner; standardized linguistic tests of phonological processing (CTOPP); and tasks that assess basic oculomotor control (prosaccades, smooth pursuit) and executive functions (predictive saccades, antisaccades).

There were several key findings. First, individuals with schizophrenia exhibited robust eye movement markers of reading difficulty relative to controls, even after controlling for demographic variables and medication. These included slower reading rates, shorter forward saccade lengths, longer forward fixation durations, and more regressive saccades during full-text reading. Second,
individuals with schizophrenia exhibited reduced perceptual spans and were less affected by reductions in parafoveal window size than were controls, presumably due to increased foveal processing load (Henderson & Ferreira, 1990; Rayner, 1986). Third, individuals with schizophrenia exhibited significant impairments on both standardized reading measures (reduced reading speed, reading comprehension, and phonological processing) and oculomotor measures of cognitive control (decreased predictive saccade amplitudes, increased antisaccade errors) relative to controls. Finally, there were significant associations between perceptual span reductions (as inferred from using forward saccade length in the no-window and 14-character window conditions) in schizophrenia and deficits in phonological awareness and reduced predictive saccade amplitudes. Deficits in oculomotor inhibition/cognitive control (antisaccade errors) were not associated with perceptual span reductions but were associated with poorer reading comprehension (sentences, NDRT). We now discuss these findings more fully with respect to the prior literature.

The first key finding was that individuals with schizophrenia exhibited differences in their eye movement record that are hallmark of reading difficulty (Adler-Grinberg & Stark, 1978; Hutzler & Wimmer, 2004; Jones, Kelly, & Corley, 2007; Pavlidis, 1978; Rayner, 1985, 1986, 1998, 2009). These included slower reading rates, shorter forward saccade lengths, longer forward fixation durations, and more regressive saccades than in controls, all of which have been reported in beginner readers, less skilled readers, and readers with dyslexia (Adler-Grinberg & Stark, 1978; Bélanger et al., in press; Hutzler & Wimmer, 2004; Jones et al., 2007; Pavlidis, 1978; Rayner, 1985, 1986, 1998, 2009). Of note, all results held when controlling for medication dose. Evidence of reading difficulty in schizophrenia is consistent with results of previous studies of naturalistic reading in schizophrenia that used standardized reading tests (Arnott et al., 2011; Hayes & O’Grady, 2003; Revheim et al., 2006) and a recent study that examined eye movements during paragraph reading in schizophrenia (E. O. Roberts et al., 2012).

The second key finding was that people with schizophrenia exhibited smaller perceptual spans (i.e., the span of effective vision during reading) than did controls. Prior work involving beginner readers (Häkkiö et al., 2009) and readers with dyslexia (Rayner, 1986; Rayner et al., 1989) has shown that perceptual span is sensitive to variations in reading skill, such that lower reading proficiency is associated with smaller perceptual spans. In the present study, controls’ reading rates decreased (relative to the no-window condition) for all window sizes smaller than the 14-character window, using the reading rate estimate of perceptual span. This finding suggests that perceptual span was roughly 14 characters to the right of fixation in controls, consistent with prior work involving skilled readers (McConkie & Rayner, 1975, 1976; Rayner & Bertera, 1979). In contrast, schizophrenia patients’ reading rates decreased (relative to the no-window condition) only for the window size smaller than the 6-character window (2-character window). This finding suggests that perceptual span was less than 6 characters to the right of fixation in schizophrenia patients but more than 3 characters, since the most restrictive window condition (2-character window) was also detrimental to reading in patients with schizophrenia. Thus, the most restrictive window condition (2-character window) negatively impacted reading regardless of participant group. Perceptual span reductions in schizophrenia for sentence-level text are consistent with reduced performance on other measures of reading skill (e.g., NDRT comprehension and reading rate). Although several details differ, our results are also consistent with previous work demonstrating perceptual span reductions in schizophrenia during paragraph reading (E. O. Roberts et al., 2012).

Perceptual span reductions are potentially attributable to at least two aberrant processes. First, smaller perceptual spans could be related to heightened word encoding difficulty, resulting in increased foveal processing load, which thereby reduces parafoveal processing (Henderson & Ferreira, 1990; Rayner, 1986). This is true of readers with dyslexia (Rayner, Pollatsek, & Bilsky, 1995). Second, smaller perceptual spans could be related to a reduced visual ability to extract parafoveal information. For example, skilled older adult readers have smaller and less asymmetric perceptual spans than do skilled younger adult readers (Rayner, Castellano, & Yang, 2009). However, these differences are not due to increased foveal processing load but rather to a reduced ability to extract parafoveal information due to age-related reductions in the field of view (Sekuler, Bennett, & Mamlok, 2000). As schizophrenia patients have recently been reported to have perceptual span reductions in (nonreading) visual search tasks (Elahipanah, Christensen, & Reingold, 2011), both sources of impairment may exist in individuals with schizophrenia. However, the relationship between perceptual span and measures of phonological processing suggest that increased foveal processing load is likely an important contributor.

Perceptual span can be dissociated from other aspects of the eye movement reading record. For example, while both individuals with dyslexia and elderly adults have reduced perceptual spans, individuals with dyslexia have shorter forward saccade amplitudes, while elderly adults have longer forward saccade amplitudes (Kemper & Liu, 2007; Rayner, Reichle, Stroud, Williams, & Pollatsek, 2006). Based on the data reported here, the reading profile in schizophrenia is more comparable to that of less skilled readers and readers with dyslexia (Bélanger et al., in press; Rayner, 1986; Rayner et al., 1989) than to that of other special populations (e.g., older adults; Rayner et al., 2009).

The third key finding was that deficits in phonological awareness (assessed by the CTOPP) and higher order oculomotor performance were associated with perceptual span reductions among individuals with schizophrenia. We first discuss the relevance of phonological awareness results. This significant association is consistent with the notion that difficulty in processing letter–sound correspondences between words compressed the perceptual span of natural reading in schizophrenia by increasing foveal load at each fixation (Henderson & Ferreira, 1990). The observation of phonological awareness impairments in individuals with schizophrenia is consistent with prior work. For example, Revheim et al. (2006) found that individuals with schizophrenia performed more poorly than did controls on the same standardized measures of phonological awareness used here, although of note, their patient sample was generally lower functioning than were the patients we tested. Interestingly, Arnott et al. (2011), who also tested a higher functioning patient sample than did Revheim and colleagues, found no group differences between patients and controls on standardized measures of phonological awareness. Thus, our findings are more consistent with those of Revheim and colleagues.
despite the fact that our patient sample was more comparable to that of Arnott and colleagues.

Schizophrenia-related impairments in phonological awareness may be part of a more general deficit in ability to process sound-based aspects of language. For example, Cienfuegos et al. (1999) found that individuals with schizophrenia exhibited deficits on a behavioral task assessing the categorical perception of speech sounds. Similarly, Kasai et al. (2002) found that individuals with schizophrenia exhibited electrophysiological markers of abnormalities in the preattentive perception of changes in speech sounds. Moreover, Wexler et al. (1998) found that difficulties discriminating pitch differences in tones were associated with deficits in working memory for words in a subset of their patients with schizophrenia. Finally, Titone and Levy (2004) found that people with schizophrenia had difficulty identifying spoken words that had many lexical competitors and that this impairment was associated with the propensity to experience auditory hallucinations. Indeed, several studies have linked spoken language processing impairments and auditory hallucinations in schizophrenia, which could suggest a common neurocognitive basis for both (Ford, Roach, Faustman, & Mathalon, 2007; Ford et al., 2009; Hoffman, Fernandez, Pittman, & Hampson, 2011; Hoffman, Rapaport, Mazure, & Quinlan, 1999; Kühn & Gallinat, 2010; Lee, Chung, Yang, Kim, & Suh, 2004; Vercammen, de Haan, & Aleman, 2008; Woodruff et al., 1997). In the present study, the correlation between phonological awareness scores and the degree to which people with schizophrenia experience auditory hallucinations was not significant. However, we tested only 20 patients and had an unbalanced distribution of scores on the BPRS. Thus, future studies with larger sample sizes would be in a better position to assess how particular symptoms of schizophrenia, such as auditory hallucinations, relate to both phonological awareness and reading deficits.

Turning to the findings for nonlinguistic oculomotor control, the association between reduced forward saccade amplitudes during reading (a proxy of perceptual span) and reduced amplitudes in a predictive saccade task suggests the possibility that both differences reflect abnormalities in the neural processes that control the metrics of voluntary saccades. Reduced saccade amplitudes, or hypometric saccades, are observed in patients with idiopathic reductions in dopamine, such as Parkinson’s disease, and these amplitude differences are greatest in cognitively loaded versus perceptually loaded saccades, such as predictive saccades (e.g., Crawford, Henderson, & Kennard, 1989; Lueck et al., 1992; Ventre, Papageorgiou, & Reich, 1992). Psychotic patients receiving dopamine antagonists have been reported to differ from comparable patients not receiving this medication in their tendency to generate hypometric saccades (Crawford, Haeger, Kennard, & Henderson, 1995; Crawford, Haeger, Kennard, Reveley, & Henderson, 1995), particularly for voluntary saccades (Crawford, Haeger, Kennard, & Henderson, 1995; Crawford, Haeger, Kennard, Reveley, & Henderson, 1995). Thus, hypometric voluntary saccades have been hypothesized to be attributable to decreased available dopamine. In the current study, differences in dopamine transmission could be either intrinsic to schizophrenia or attributable to the effects of medication. However, two findings in the current study argue against an interpretation of the data based on medication effects: First, neuroleptic dose (chlorpromazine equivalents) was not associated with forward saccade amplitude or predictive saccade amplitude; second, forward saccade amplitude during reading is less than three degrees of visual angle; however, dopamine antagonist effects are found for saccade amplitudes greater than 10 degrees of visual angle (Crawford, Haeger, Kennard, & Henderson, 1995; Crawford, Haeger, Kennard, Reveley, & Henderson, 1995).

Interestingly, the oculomotor measure of inhibition/cognitive control (antisaccade errors) was not related to perceptual span; however, it was the only measure associated with reading comprehension (sentences, NDRT). This suggests that necessary components of skilled reading are scaffolded on different capacities, including processes that regulate saccades under voluntary control, phonological awareness that affects core linguistic operations, and oculomotor inhibition/cognitive control that affects higher level aspects of psycholinguistic function (e.g., semantic integration or inferencing), which may improve text comprehension.

The relationship between inhibitory capacity and reading skill has been relatively underinvestigated, although numerous studies have suggested a link between inhibitory capacity and higher level aspects of language processing in healthy young readers (e.g., Bialystok & Craik, 2010; Germbacher & Faust, 1991; Linck, Hoshino, & Kroll, 2008; Miyake, Just, & Carpenter, 1994; S. Wagner & Gunter, 2004). A few studies have found significantly impaired Stroop performance (inhibition of the prepotent tendency to read a word and instead name its color) in dyslexia (Everatt, Warne, Miles, & Thoms, 1997; Helland & Asbjørnsen, 2000), although reading would be expected to be less automatic or prepotent in this group (Faccioli, Peru, Rubini, & Tassanari, 2008). Only one previous study investigated the relationship between antisaccade performance and reading skill, and this was in healthy young children (Huestegge, Radach, Corbic, & Huestegge, 2009). This study found that the relationship between antisaccade performance and eye movement measures of individual word reading in the second grade was not predictive of reading rate in the fourth grade.

An association between antisaccade errors and reading comprehension in the current study could relate to reading in at least two ways. First, the ability to attend to relevant stimuli and to screen out competing stimuli is likely a critical aspect of reading comprehension, which occurs at the lexical, sentence, and discourse levels (e.g., Daneman & Carpenter, 1983; Germbacher, 1990; Germbacher & Faust, 1991; Kintsch, 1988; May, Zacks, Hasher, & Multhaup, 1999; Miyake et al., 1994; S. Wagner & Gunter, 2004). Thus, a relationship between increased inhibitory capacity and increased reading skill would be expected. Second, working memory, a cognitive capacity associated with antisaccade performance (e.g., Crawford, Parker, Solis-Trapela, & Mayes, 2011; R. J. Roberts, Hagar, & Heron, 1994; Unsworth, Shrock, & Engle, 2004), is also presumably important in reading comprehension. Thus, the association between antisaccade errors and reading comprehension may arise from variations in working memory. However, our two direct measures of working memory (Memory For Digits and Nonword Repetition) were not associated with reading comprehension (sentences, NDRT). As these tasks may not be as taxing on working memory capacity as are other tasks normally used in the psycholinguistic literature (e.g., Daneman & Carpenter, 1980), future work involving a greater range of working memory span tasks may be better suited for investigating such hypotheses.

The findings observed here for individuals with schizophrenia bear some similarity to those previously found for individuals with...
dyslexia. For example, deficits in both phonological processing (e.g., Hatcher et al., 1994; Pugh & McCandless, 2009; Rack, 1994) and oculomotor control (e.g., Adler-Grinberg & Stark, 1978; Biscaldi, Fischer, & Aiple, 1994; Eden et al., 1994) have been extensively documented in individuals with dyslexia. One hypothesis is that a common neurodevelopmental etiology may underlie the types of reading deficits observed in schizophrenia and dyslexia (Condray, 2005). Evidence of a common neurodevelopmental etiology include (a) an association between reading impairments in schizophrenia and abnormal cortical structures related to genes involved in the manifestation of reading disorder (Jamadar et al., 2011); (b) anomalous brain areas implicated in dyslexia being predictive of cognitive functioning and reading comprehension in schizophrenia (Leonard et al., 2008); and (c) evidence of abnormal magnocellular function (Chen et al., 1999; Cornelissen et al., 1995; Livingstone et al., 1991; Martínez et al., 2008; Revheim et al., 2006; Talcott et al., 1998), anomalous smooth pursuit eye movements (e.g., Adler-Grinberg & Stark, 1978; Biscaldi et al., 1994; Eden et al., 1994; O’Driscoll & Callahan, 2008; Pavlidis, 1981), and impairments on saccadic measures of inhibition/cognitive control (e.g., Biscaldi et al., 2000; Gooding & Basso, 2008; Sereno & Holzman, 1995). A common neurodevelopmental etiology is further supported by our findings of skilled reading deficits in schizophrenia, which are comparable to those reported in dyslexia (Adler-Grinberg & Stark, 1978; Hutzler & Wimmer, 2004; Jones et al., 2007; Pavlidis, 1978; Rayner, 1985, 1986, 1998, 2009).

Cognitive, perceptual, and motor abnormalities seen in schizophrenia are thought to reflect neurodevelopmental aspects of schizophrenia vulnerability because they are observed at the beginning of the illness and in populations at elevated risk for schizophrenia, such as clinically well first-degree relatives of individuals with schizophrenia and individuals with schizotypal traits (Gottesman & Gould, 2003). For example, reduced evoked potentials on semantic processing tasks have been found in individuals with schizotypal traits and in clinically well first-degree relatives (e.g., Foxe et al., 2011; Kimble et al., 2000; Niznikiewicz et al., 1999). Moreover, impairments on magnocellular tasks (motion perception), smooth pursuit tasks, and oculomotor tasks of cognitive control have also been documented in both of these groups (e.g., Chen et al., 1999; O’Driscoll, Lenzenweger, & Holzman, 1998; Radant et al., 2010; Richardson & Gruzelier, 1994).

Recently, E. O. Roberts et al. (2012) observed eye movement abnormalities during reading in both individuals with schizophrenia and their clinically well first-degree relatives. Consistent with the notion that schizophrenia and dyslexia share neurodevelopmental precursors (Condray, 2005), there is also evidence of the co-occurrence of schizophrenia and dyslexia, or cosegregation of the disorders, within families. For example, high-risk studies have documented elevated rates of dyslexia in the children of individuals with schizophrenia (Erlenmeyer-Kimling et al., 1984; Fish, 1987; Marcus, 1974) and impairments on tasks assessing the perception of speech sounds, an index on phonological processing (Hallett & Green, 1983). Similarly, there have been reported elevations of schizotypal symptoms in individuals with dyslexia (Claridge & Broks, 1984; Richardson & Gruzelier, 1994). Taken together, accumulating evidence suggests that schizophrenia involves a collection of disrupted mechanisms, some of which are in common with dyslexia, including impaired linguistic, phonological, visual, and oculomotor processes (reviewed in Fuller et al., 2002; Leonard et al., 2008; Revheim et al., 2006). Future investigations that make more explicit comparisons of reading behaviors and the component processes of reading in individuals with dyslexia may provide new insight into the neurodevelopmental paths of both disorders.

While our results clearly demonstrate reading impairments in people with schizophrenia, the interpretation of our findings is constrained by several factors. The first factor involves sample size. Although, the sample size of our study is at the larger end of studies examining naturalistic reading in schizophrenia (Arnott et al., 2011; Revheim et al., 2006) and was large enough to detect the hypothesized effects, it was not sufficient for assessing other potential correlates of reading difficulty in schizophrenia (e.g., those relating to differences among patients with specific symptoms). The second factor is that our patient sample was relatively high functioning (mean Global Assessment of Functioning Scale = 66.31) and thus may not be representative of all individuals with schizophrenia. However, the gap between the patients’ academic achievement and reading performance (~1.8 years) was typical of what has been reported in the literature (Fuller et al., 2002). A third factor is that our sentence stimuli may have underestimated the demands of normal reading, given that they consisted of syntactically simple, single sentences that included mostly high-frequency words. The decision to use such simple sentences was intentional, however, as it was our explicit aim to determine whether individuals with schizophrenia would exhibit reading impairments under the best of circumstances. The fact that our patient sample was relatively high functioning is relevant to this aim as well.

To conclude, individuals with schizophrenia exhibited robust eye movement markers of reading difficulty for sentence-level text and reduced perceptual spans compared with controls. Moreover, individual differences among patients in two fundamental processes that normally drive the eyes during reading, language and oculomotor control, were linked to impaired reading in schizophrenia. The deficits observed were similar to those found in dyslexia and included impairments in both phonological processing (e.g., Hatcher et al., 1994) and oculomotor control (e.g., Biscaldi et al., 1994). Thus, a common neurodevelopmental etiology may underlie the types of reading deficits observed in schizophrenia and dyslexia (Condray, 2005). If true, remediation strategies used to address phonological processing issues in dyslexia could potentially be extended to address phonological processing issues in schizophrenia. Further, given that reading skills are developed and mastered before the typical onset of schizophrenia, we believe that reading difficulty may provide an early window into cognitive aspects of vulnerability to schizophrenia (Ambelas, 1992; Crow et al., 1995; Fuller et al., 2002; Reichenberg et al., 2002; Weiser et al., 2004, 2007). If true, reading measures, in combination with other information such as family history, might be used to better identify people in the early stages of the illness and thus allow for better targeting of early interventions.

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