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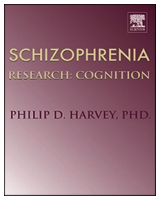

Research Paper

Eye movements and the perceptual span in disordered reading: A comparison of schizophrenia and dyslexia

Veronica Whitford [a,*], Narissa Byers [a], Gillian A. O'Driscoll [b,c,d,e,f], Debra Titone [b,g]

[a] Department of Psychology, University of New Brunswick, 38 Dineen Drive, Fredericton, New Brunswick, E3B 5A3, Canada
[b] Department of Psychology, McGill University, 2001 Avenue McGill College, Montréal, Québec, H3A 1G1, Canada
[c] Department of Psychiatry, McGill University, 1003 Avenue des Pins Ouest, Montréal, Québec, H3A 1A1, Canada
[d] Douglas Mental Health University Institute, 6875 Boulevard LaSalle, Verdun, Québec, H4H 1R3, Canada
[e] Montreal Neurological Institute and Hospital, 3801 Rue University, Montréal, Québec, H3A 2B4, Canada
[f] Department of Neurology & Neurosurgery, McGill University, 1003 Avenue des Pins Ouest, Montréal, Québec, H3A 1A1, Canada
[g] Centre for Research on Brain, Language and Music, 3640 Rue de la Montagne, Montréal, Québec, H3G 2A8, Canada

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ABSTRACT

Increasing evidence of a common neurodevelopmental etiology between schizophrenia and developmental dyslexia suggests that neurocognitive functions, such as reading, may be similarly disrupted. However, direct comparisons of reading performance in these disorders have yet to be conducted. To address this gap in the literature, we employed a gaze-contingent moving window paradigm to examine sentence-level reading fluency and perceptual span (breadth of parafoveal processing) in adults with schizophrenia (dataset from Whitford et al., 2013) and psychiatrically healthy adults with dyslexia (newly collected dataset). We found that the schizophrenia and dyslexia groups exhibited similar reductions in sentence-level reading fluency (e.g., slower reading rates, more regressions) compared to matched controls. Similar reductions were also found for standardized language/reading and executive functioning measures. However, despite these reductions, the dyslexia group exhibited a larger perceptual span (greater parafoveal processing) than the schizophrenia group, potentially reflecting a disruption in normal foveal-parafoveal processing dynamics. Taken together, our findings suggest that reading and reading-related functions are largely similarly disrupted in schizophrenia and dyslexia, providing additional support for a common neurodevelopmental etiology.

1. Introduction

Despite their different clinical presentations and functional outcomes, growing evidence suggests that schizophrenia (a psychiatric disorder characterized by disruptions in perception, cognition, language, and behaviour; American Psychiatric Association, 2013) and developmental dyslexia (a language-based learning disorder characterized by difficulties with accurate and/or fluent word reading and spelling; American Psychiatric Association, 2013) may share a common neurodevelopmental basis (Condray, 2005; Vanova et al., 2021; Whitford et al., 2018). Support for this notion comes from biological-molecular and population-based research reporting genetic, familial, and pathophysiological overlap between the two disorders (Becker et al., 2012; Duboc et al., 2015; Jamadar et al., 2011; Leonard et al., 2008; Paracchini et al., 2016; Stefansson et al., 2014; Trulioff et al., 2017). For instance, studies have found that the unaffected first-degree relatives of people with schizophrenia have higher rates of dyslexia than the general population (Erlenmeyer-Kimling et al., 1984; Fish, 1987; Götz and Edmonstone, 1992; Horrobin et al., 1995; Marcus, 1974; Roberts et al., 2013); that people with dyslexia and their first-degree relatives are at a greater risk of developing schizophrenia and other psychiatric disorders (Cederlöf et al., 2017; Reichenberg et al., 2002; Weiser et al., 2007); that people with dyslexia exhibit greater schizotypal traits (attenuated expressions of schizophrenia-spectrum psychopathology) than controls and the general population (Barkus et al., 2022; Richardson, 1994; Richardson and Stein, 1993); and that some people with schizophrenia meet diagnostic criteria for dyslexia, depending on the model used (Bersani et al., 2006; Revheim et al., 2006).

The commonality between schizophrenia and dyslexia may be driven

* Corresponding author.
E-mail address: veronica.whitford@unb.ca (V. Whitford).

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by developmental brain dysfunction, where aberrant brain development (due to genetic, epigenetic, and/or environmental perturbations) leads to a variety of neurodevelopmental and psychiatric disorders with similar perceptual, cognitive, linguistic, and behavioural impairments (Coe et al., 2012; Cristiano et al., 2014; Evans et al., 2015; Gonzalez-Mantilla et al., 2016; Moreno-De-Luca et al., 2013; Torres et al., 2016). Consistent with this concept, research has reported premorbid dyslexia-like reading impairments in people with schizophrenia, through the retrospective examination of cohort reports, guidance counsellor reports, parental self-reports, and scholastic test records (Ambelas, 1992; Crow et al., 1995; DeLisi et al., 1991; Fuller et al., 2002; Jones et al., 1994; Reichenberg et al., 2002; Weiser et al., 2007), as well as post-morbid dyslexia-like reading impairments through the use of standardized and/or experimental measures of word-level reading (Curzietti et al., 2018; Leonard et al., 2008; Martínez et al., 2008, 2013, Revheim et al., 2014; Vinckier et al., 2014; cf. Mitelman et al., 2021; Revheim et al., 2006) and sentence/text-level reading (Arnott et al., 2011; Bersani et al., 2006; Dias et al., 2021; DiSimoni et al., 1977; Dondé et al., 2019; Fernández et al., 2016a; Fernández et al., 2016b; Hayes and O'Grady, 2003; Leonard et al., 2008; Martínez et al., 2013; Revheim et al., 2006, 2014; Roberts et al., 2013; Whitford et al., 2013).

The latter includes eye-tracking studies that have reported more effortful reading behaviour in people with schizophrenia relative to controls, including more and longer fixations, slower reading rates, more progressive (forward-going) and regressive (backward-going or re-reading) saccades, reduced saccade amplitudes, and smaller perceptual spans (size of the effective visual field or breadth of parafoveal processing; see Roberts et al., 2013; Whitford et al., 2013). Although similar oculomotor markers of reading difficulty have been extensively reported in people with dyslexia, the primary focus has been on children (Adler-Grinberg and Stark, 1978; Elterman et al., 1980; Franzen et al., 2021; Hutzler and Wimmer, 2004; Hyönä and Olson, 1995; Jones et al., 2007; Morris and Rayner, 1991; Nilsson Benfatto et al., 2016; Parshina et al., 2022; Pavlidis, 1978; Prado et al., 2007; Rayner, 1986; Zangwill and Blakemore, 1972; reviewed in Rommelse et al., 2008; Whitford et al., 2018), with only one case study reporting reduced perceptual span and parafoveal processing in adult dyslexia (Rayner et al., 1989).

Similar reading impairments between schizophrenia and dyslexia may be driven by common disturbances in the perceptual and neurocognitive systems that subserve reading. For instance, there is increasing evidence that both disorders involve similar deficits in language, particularly phonological processing; low-level auditory processing, including speech and non-speech sound recognition; low-level visual processing, including magnocellular/dorsal stream functioning; non-linguistic oculomotor control, including smooth pursuit and anti-saccade performance; and executive functioning, including attention, inhibition, and working memory (reviewed in Whitford et al., 2018).

Disturbances in the above reading-related systems likely multiplicatively give rise to reading impairments in both disorders. For instance, previous research by Whitford et al. (2013) using a gaze-contingent moving window paradigm (a technique that manipulates the amount of parafoveal information available in the direction of reading; McConkie and Rayner, 1975) found that reduced sentence reading fluency, including reduced forward saccade amplitudes (6.89 vs. 8.71 characters) and perceptual span (~6 vs. ~14 characters rightward of fixation), in people with schizophrenia relative to controls related to deficits in language processing (reduced phonological awareness) and non-linguistic oculomotor control (reduced predictive saccade amplitudes). Moreover, impairments in higher-order oculomotor control/executive functioning (increased antisaccade errors) related to deficits in sentence comprehension and text-level reading comprehension on a standardized test. It is important to note, however, that direct comparisons of reading and reading-related disturbances in schizophrenia and dyslexia have yet to be conducted—an issue that the present work addresses.

1.1. Current study

Our overarching goal was to bridge the two separate lines of research on reading in schizophrenia and reading in dyslexia by determining whether individuals with these disorders exhibit similar deficits in reading (and, as a secondary focus, reading-related processes, such as language/phonological processing and higher-order oculomotor control/executive functioning). To this end, we extended Whitford et al.'s (2013) work by employing the same materials to examine whether the nature and magnitude of reading impairments in their sample of people with schizophrenia were comparable to those in a newly collected sample of psychiatrically healthy people with dyslexia.

2. Methods

2.1. Participants

2.1.1. Dyslexia and control groups

Nineteen individuals with dyslexia and 17 matched controls were recruited from Montréal, Canada (see Table 1). Diagnosis of developmental dyslexia was confirmed through official neuropsychological reports, with no concurrent diagnosis of attention-deficit/hyperactivity disorder (ADD/ADHD). Both groups were screened/excluded for DSM-IV Axis I Disorders using the non-patient version of the Structured Clinical Interview for DSM Disorders (SCID-NP; First et al., 1996).

2.1.2. Schizophrenia and control groups

As reported in Whitford et al. (2013), 20 clinically stable outpatients with schizophrenia and 16 matched controls were also recruited from Montréal, Canada (see Table 2). Diagnosis of schizophrenia was confirmed through chart review and the patient version of the SCID (SCID-P; First et al., 1996). Controls were screened/excluded for DSM-IV Axis I Disorders using the SCID-NP.

All participants provided oral and written informed consent after the study was fully explained to them and were compensated $18/h. The study was approved by McGill University's Research Ethics Board (#58-0711).

2.2. Materials

As described in Whitford et al. (2013), the materials were standardized language/reading assessments (see Tables 3 and 4); an anti-saccade task—an oculomotor measure of executive functioning (see Tables 3 and 4); and a gaze-contingent moving window reading task. For the latter, stimuli were 90 (15 practice, 75 experimental) short, syntactically simple English or French sentences (depending on participants' first acquired/dominant language). The sentences were

Table 1
Demographic characteristics of the dyslexia and control groups.

	Dyslexia group ($n = 19$)	Control group ($n = 17$)
	Mean (SD)	Mean (SD)
Sex (male-to-female ratio)	5:14	4:13
Age (years)	23.00 (5.39)	23.24 (2.44)
Native language (English-to-French ratio)	16:3	14:3
Verbal IQ (scaled scores)	13.79 (1.99)	13.94 (1.92)
Parental socioeconomic status (SES)	2.58 (0.90)	2.53 (1.18)
Education (years)	14.29 (1.85)	15.35 (1.80)

Note 1: Participant groups were matched on all variables (all p values $> .05$).
Note 2: Sex was based on self-reported assigned sex at birth; native language was based on an adaptation of the Language Experience and Proficiency Questionnaire (LEAP-Q; Marian et al., 2007); verbal IQ was based on the Vocabulary subtest of the Wechsler Adult Intelligence Scale–Revised (WAIS–R; Wechsler, 1981); and parental SES was based on the Hollingshead Occupational Scale (Hollingshead, 1975).

Table 2
Demographic and clinical characteristics of the schizophrenia and control groups.

	Schizophrenia group ($n = 20$)	Control group ($n = 16$)
	Mean (SD)	Mean (SD)
Sex (male-to-female ratio)	16:4	13:3
Age (years)	31.05 (9.08)	31.56 (10.08)
Native language (English-to-French ratio)	10:10	10:6
Verbal IQ (scaled scores)	10.83 (3.76)	12.75 (2.86)
Parental SES	3.95 (2.07)	3.87 (1.81)
Education (years)**	11.85 (1.99)	13.66 (1.87)
BPRS		
Total score	53.05 (11.78)	
Positive subscales (1–7)	2.73 (0.88)	
Negative subscales (1–7)	1.69 (0.54)	
Chlorpromazine equivalent dose (mg/day)	443.57 (277.55)	
Illness duration (years)	10.85 (9.43)	

Note 1: Participant groups were matched on all variables (all p values $> .05$), except education (** $p < .01$).
Note 2: Sex, native language, verbal IQ, and parental SES were based on same measures reported in Table 1.
Note 3: Current symptoms in the schizophrenia group were rated with the Brief Psychiatric Rating Scale (BPRS; Overall and Gorham, 1962), with all but three people medicated and following the same prescriptions/dosage for at least one month before the study.
Note 4: Although the schizophrenia and dyslexia samples were not matched (all p values $< .01$) on sex, age, native language, verbal IQ, parental SES, and years of education, the pattern of results remained unchanged even when the analyses included a subset of schizophrenia participants matched as closely as possible to the dyslexia sample (see Appendix—Table A.1).
Note 5: Inclusion criteria for all participants in study: aged 18 to 50 years; first acquired/dominant language either English or French; and verbal IQ >80.
Note 6: Exclusion criteria for all participants in study: history of neurological impairment (other than schizophrenia for the schizophrenia group); current substance abuse/dependence or history of abuse/dependence within one month of testing; current use of drugs that affect saccade velocities (e.g., benzodiazepines, chloral hydrate); and any uncorrected deficits in visual acuity (based on the Snellen eye chart).

Table 3
Standardized language/reading and antisaccade measures for the dyselxia and control groups.

	Dyslexia group ($n = 19$)	Control group ($n = 17$)
	Mean (SD)	Mean (SD)
WIAT-II Word Reading (standard scores)*	109.16 (7.36)	112.88 (2.18)
WIAT-II Pseudoword Decoding (standard scores)***	102.26 (8.73)	117.53 (4.21)
NDRT Comprehension (scaled scores)***	200.84 (24.16)	233.24 (13.75)
NDRT Reading Rate (scaled scores)***	194.26 (19.48)	217.53 (19.10)
CTOPP Phonological Awareness (composite scores)***	100.16 (8.34)	114.12 (3.31)
Elision (standard scores)***	9.16 (1.61)	11.35 (0.70)
Blending Words (standard scores)***	10.89 (1.56)	13.47 (0.62)
CTOPP Phonological Memory (composite scores)***	104.21 (8.26)	120.65 (5.50)
Memory for Digits (standard scores)***	11.05 (2.37)	13.76 (1.52)
Nonword Repetition (standard scores)***	10.84 (1.68)	13.12 (0.86)
CTOPP Rapid Naming (composite scores)***	86.42 (14.98)	106.71 (11.98)
Rapid Digit Naming (standard scores)***	8.53 (2.55)	11.12 (2.15)
Rapid Letter Naming (standard scores)***	6.95 (2.76)	11.12 (2.09)
Antisaccade Task (percent errors)**	15.21 (12.39)	4.27 (3.17)

Note 1: Participant groups significantly differed on all above measures (* $p < .05$; ** $p < .01$; *** $p < .001$).
Note 2: WIAT-II (Wechsler Individual Achievement Test – Second Edition; Wechsler, 2005; English-Canadian and French-Canadian adaptations). Raw subtest scores were converted to standard scores ($M = 100 \pm 15$).
Note 3: NDRT (Nelson-Denny Reading Test; Brown et al., 1993; French-Canadian adaptation available upon request). Raw subtest scores were converted to scaled scores ($M = 200 \pm 25$).
Note 4: CTOPP (Comprehensive Test of Phonological Processing; Wagner et al., 1999; French-Canadian adaptation: Béland and Hébert, 2009). Raw subtest scores were converted to scaled scores ($M = 10 \pm 3$), which were then converted to three composite scores: Phonological Awareness, Phonological Memory, and Rapid Naming ($M = 100 \pm 15$).
Note 5: As reported in Whitford et al. (2013), for the antisaccade task, participants fixated a central target (0.5° by 0.5° of visual angle) on a computer screen. After 800 to 1400 ms, a peripheral target (0.5° by 0.5° of visual angle) appeared 11° leftward or rightward of the central target in a pseudorandomized order. Participants were instructed to look in the opposite direction of the peripheral target as quickly as possible. Fifty-seven trials were administered (nine practice, 48 experimental). Maximum trial duration was 2000 ms. Errors were first saccades exceeding 2° toward the peripheral target. An EyeLink II headband-mounted system (sampling rate = 250 Hz, spatial resolution = 0.01°, mean accuracy = 0.25°; SR-Research, Canada) recorded eye movements in a darkened room. Viewing was binocular; however, recording was dominant eye monocular. Eye movements were calibrated with a 3-point horizontal line (average fixation error < 0.4° of visual angle), with drift-correction checks before each trial. The task was displayed on a 19-in. CRT monitor (screen resolution = 1024 × 768 pixels, refresh rate = 120 Hz), positioned 57 cm from participants.
Note 6: A comparison of the schizophrenia and dyslexia groups revealed similar performance on all standardized language/reading and antisaccade measures (all p values $> .05$), suggesting similar impairments in these areas.

Table 4
Standardized language/reading and antisaccade measures for the schizophrenia and control groups.

	Schizophrenia group ($n = 20$)	Control group ($n = 16$)
	Mean (SD)	Mean (SD)
WIAT-II Word Reading (standard scores)**	104.80 (11.13)	113.50 (1.59)
WIAT-II Pseudoword Decoding (standard scores)**	105.45 (10.25)	118.31 (3.09)
NDRT Comprehension (scaled scores)***	191.00 (25.14)	230.88 (11.19)
NDRT Reading Rate (scaled scores)***	189.20 (17.29)	220.19 (26.64)
CTOPP Phonological Awareness (composite scores)***	97.00 (10.75)	113.88 (3.07)
Elision (standard scores)***	9.30 (1.69)	11.25 (0.58)
Blending Words (standard scores)***	9.85 (2.39)	13.38 (0.72)
CTOPP Phonological Memory (composite scores)***	103.60 (14.49)	120.63 (4.50)
Memory for Digits (standard scores)**	10.45 (3.43)	13.38 (1.15)
Nonword Repetition (standard scores)***	11.10 (1.65)	13.38 (0.72)
CTOPP Rapid Naming (composite scores)**	94.75 (16.77)	113.06 (14.40)
Rapid Digit Naming (standard scores)***	9.25 (2.49)	12.56 (2.28)
Rapid Letter Naming (standard scores)*	8.90 (3.67)	11.88 (3.12)
Antisaccade Task (percent errors)*	17.91 (17.68)	10.06 (15.44)

Note 1: Participant groups significantly differed on all above measures (* $p < .05$; ** $p < .01$; *** $p < .001$).
Note 2: WIAT-II (Wechsler Individual Achievement Test – Second Edition; Wechsler, 2005; English-Canadian and French-Canadian adaptations).
Note 3: NDRT (Nelson-Denny Reading Test; Brown et al., 1993; French-Canadian adaptation available upon request).
Note 4: CTOPP (Comprehensive Test of Phonological Processing; Wagner et al., 1999; French-Canadian adaptation: Béland and Hébert, 2009).
Note 5: Antisaccade task described in Table 3.
Note 6: As reported in Table 3, a comparison of the schizophrenia and dyslexia groups revealed similar performance on all standardized language/reading and antisaccade measures (all p values $> .05$).

Table 5
Sample experimental sentence presented across all window sizes.

Window Size	Sentence
No-window	He visits a new country each year on vacation. *
4L/14R window	----------ew country each yea---------- *
4L/10R window	----------ew country each---------- *
4L/6R window	----------ew country ---------- *
4L/2R window	----------ew coun---------- *

Note: L = characters to the left of fixation; R = characters to the right of fixation; * = fixation point.

Table 6
Reading comprehension accuracy (%) for the experimental sentences.

Dyslexia group ($n = 19$)	Control group ($n = 17$)	Schizophrenia group ($n = 20$)	Control group ($n = 16$)
Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
86.03 (8.06)	87.96 (6.63)	83.79 (7.40)	87.58 (4.98)

Note: Accuracy was comparable between the dyslexia and control groups ($p > .05$); significantly lower in the schizophrenia vs. control group ($p < .05$); and comparable between the schizophrenia and dyslexia groups ($p > .05$).

translation equivalents (e.g., *My French teacher is a very funny man / Mon professeur de français est un homme très drôle*) and matched across both languages for key properties (e.g., number of words, word frequency). The sentences were matched and equally distributed across five conditions: four window conditions that manipulated the amount of parafoveal information available rightward of fixation (2, 6, 10, and 14 characters; each window was fixed at 4 characters leftward of fixation) and one no-window (full text) condition. Text was presented normally within each window during fixation; however, beyond this area, dashes replaced characters and spaces (see Table 5). This paradigm allowed us to examine global (i.e., sentence-level) reading performance for the no-window (full text) condition (e.g., average reading rate, average forward saccade length), as well as the perceptual span (breadth of parafoveal processing) for the window conditions versus the no-window (full text) condition.

2.3. Apparatus

An EyeLink 1000 desktop-mounted system (sampling rate = 1 kHz, spatial resolution = 0.01°, mean accuracy = 0.25°; SR-Research, Canada) recorded eye movements during the gaze-contingent moving window task. Viewing was binocular, but recording was right-eye monocular.[1] Calibration was performed with a 5-point cross (average fixation error < 0.4° of visual angle), with drift-correction checks before

[1] Deficits in the binocular coordination of eye movements (e.g., vergence errors) have been reported in both schizophrenia (e.g., Bolding et al., 2014; Chrobak et al., 2022; Levin et al., 1982) and dyslexia (e.g., Bucci et al., 2008a, 2008b; Stein et al., 1988). However, following the assumption that both eyes fixate the same location during reading (see Liversedge et al., 2006), the current study only investigated right-eye reading behaviour in these groups (which represented the dominant eye for most participants). As a result, we were unable to evaluate the relationship between right-eye and left-eye fixation durations (i.e., determine whether they were correlated or not), leaving fixation disparity a possibility and potential limitation of our work.

each sentence. A padded head-rest minimized head movements. Sentences were displayed on a 21-in. CRT monitor (screen resolution = 1021 × 768 pixels, refresh rate = 144 Hz, display change delay = 8.7 ms), positioned 57 cm from participants. Sentences were presented in yellow 11-point Courier New font (maximum characters per line = 75; characters per 1° of visual angle = 3.2) against a black background using EyeTrack software (http://www.psych.umass.edu/eyelab/software).

2.4. Procedure

Participants first completed the clinical and demographic measures, followed by the remaining tasks, which were randomized.

For the gaze-contingent moving window reading task, participants silently read sentences for comprehension, which was assessed via yes/no questions following 20% of trials (see Table 6). The 75 experimental sentences (15 per condition) were presented as single blocks across the five conditions. Block order was randomized using three separate lists, which were counterbalanced across participants.

3. Results

We conducted two sets of analyses. The first set examined global (sentence-level) reading performance for the no-window (full text) condition between the different participant groups. We focused on five eye movement measures: (1) reading rate (number of words per minute); (2) forward saccade length (number of characters); (3) forward fixation duration (ms); (4) number of forward fixations; and (5) number of regressions (backward saccades). The second set examined the perceptual span (amount of visual information extracted during fixation) for the window conditions versus the no-window (full text) condition between the different participant groups. We focused on reading rates as estimates of the perceptual span (following Rayner, 1986). For both sets of analyses, blinks and fixations <80 ms were excluded (<5% data loss); no upper cutoff was applied to maximize data inclusion. Saccades had a minimum velocity of 30°/sec, minimum acceleration of 8000°/sec^2, and minimum change in eye position of 0.15°, as per SR-Research's saccade detection algorithm.

The data were analyzed using linear mixed-effects models within the lme4 package (Bates et al., 2015) of R (version 4.2.0; Baayen et al., 2008; R Development Core Team, 2022). The same model was applied to all eye movement measures within each analysis.

Table 7
Model specifications for the dyslexia vs. control group comparison (full text reading).

Fixed effect	Control predictors/ covariates	Random effects
▪ Clinical status	▪ Age (continuous) ▪ Native language ▪ Verbal IQ (continuous) ▪ Parental SES (continuous) ▪ Education (continuous) ▪ List order ▪ Trial number (continuous)	▪ By-participant random intercepts ▪ By-item random intercepts

Note 1: Clinical status (dyslexia vs. control) was treatment coded (baseline = control); native language (English vs. French) was deviation coded (−0.5, +0.5); and list order (1 vs. 2 vs. 3) was deviation coded (+1, 0; 0, +1; −1, −1).
Note 2: All continuous control predictors/covariates were centered and scaled (i.e., standardized, z-scored) to reduce collinearity.

Table 8
Model specifications for the schizophrenia vs. dyslexia group comparison (full text reading).

Fixed effect	Control predictors/covariates	Random Effects
▪ Clinical status	▪ Age (continuous) ▪ Native language ▪ Verbal IQ (continuous) ▪ Parental SES (continuous) ▪ Education (continuous) ▪ List order ▪ Trial number (continuous) ▪ Chlorpromazine equivalent dose (continuous)	▪ By-participant random intercepts ▪ By-item random intercepts

Note 1: Clinical status (schizophrenia vs. dyslexia) was treatment coded (baseline = schizophrenia); native language (English vs. French) was deviation coded (−0.5, +0.5); and list order (1 vs. 2 vs. 3) was deviation coded (+1, 0; 0, +1; −1, −1).
Note 2: All continuous control predictors/covariates were centered and scaled (i.e., standardized, z-scored) to reduce collinearity.

3.1. Global sentence reading performance (no-window)

3.1.1. Dyslexia and control groups

The model specifications are comparable to those reported in Whitford et al. (2013); however, some additional control predictors/covariates were included here: parental SES, verbal IQ, and list order (see Table 7 and Appendix—Table A.2 for complete model output).

The effect of clinical status was significant for all eye movement measures except forward fixation duration. Compared to controls, the dyslexia group had slower reading rates (157 vs. 206 words/min; $\beta = -44.93$, $SE = 12.59$, $t = -3.57$, $p = .001$), shorter forward saccades (7.45 vs. 8.34 characters; $\beta = -0.89$, $SE = 0.42$, $t = -2.12$, $p = .044$), more forward fixations (9 vs. 7; $\beta = 1.52$, $SE = 0.48$, $t = 3.14$, $p = .004$), and more regressions (3 vs. 1; $\beta = 1.60$, $SE = 0.38$, $t = 4.19$, $p < .001$). Thus, people with dyslexia exhibited robust oculomotor markers of reading difficulty during normal reading conditions—a pattern similar to that found for schizophrenia participants vs. controls, even when controlling for medication (Note: the schizophrenia vs. control group results are not repeated for parsimony; see Appendix—Table A.3).

3.1.2. Schizophrenia and dyslexia groups

The model specifications follow those of the previous model, with

Table 9
Model specifications for the dyslexia vs. control group comparison (perceptual span).

Fixed effect	Control predictors/ covariates	Random effects
▪ Clinical status ▪ Window size	▪ Age (continuous) ▪ Native language ▪ Verbal IQ (continuous) ▪ Parental SES (continuous) ▪ Education (continuous) ▪ List order ▪ Trial number (continuous)	▪ By-participant random intercepts ▪ By-item random intercepts

Note 1: Clinical status (dyslexia vs. control) was treatment coded (baseline = control); window size (full text vs. 2-, 6-, 10-, and 14-character windows) was treatment coded (baseline = full text); native language (English vs. French) was deviation coded (−0.5, +0.5); and list order (1 vs. 2 vs. 3) was deviation coded (+1, 0; 0, +1; −1, −1).
Note 2: All continuous control predictors/covariates were centered and scaled (i.e., standardized, z-scored) to reduce collinearity.

the addition of chlorpromazine equivalent dose (see Table 8 and Appendix—Table A.4 for complete model output).

There were no significant between-group differences in reading performance across all eye movement measures (all p values $> .05$): reading rate (139 vs. 157 words/min), forward saccade length (6.89 vs. 7.45 characters), forward fixation duration (241 vs. 216 ms), number of forward fixations (9 vs. 9), and number of regressions (2 vs. 3). Thus, people with schizophrenia and people with dyslexia exhibited similar oculomotor markers of reading difficulty during normal reading conditions.

3.2. Perceptual span (parafoveal processing) performance

3.2.1. Dyslexia and control groups

The model specifications follow those of previous models, with the addition of window size (see Table 9 and Appendix—Table A.5 for complete model output).

The effects of clinical status and window size were both significant. Compared to controls, the dyslexia group had slower reading rates (142 vs. 178 words/min; $\beta = -46.98$, $SE = 8.83$, $t = -5.32$, $p < .001$). Compared to the full text condition, reading rates were slower in the 2-character (114 vs. 180 words/min; $\beta = -84.82$, $SE = 4.02$, $t = -21.09$, $p < .001$), 6-character (157 vs. 180 words/min; $\beta = -29.33$, $SE = 4.01$, $t = -7.32$, $p < .001$), and 10-character (165 vs. 180 words/min; $\beta = -23.40$, $SE = 4.01$, $t = -5.84$, $p < .001$) window conditions. The interaction between clinical status and window size was also significant. Compared to controls, the dyslexia group's reading rates were less negatively impacted (relative to the full text condition) by the 2-character (difference of 51 vs. 84 words/min; $\beta = 32.40$, $SE = 5.52$, $t = 5.87$, $p < .001$), 6-character (difference of 16 vs. 30 words/min; $\beta = 13.37$, $SE = 5.52$, $t = 2.42$, $p = .016$), and 10-character (difference of 8 vs. 23 words/min; $\beta = 14.01$, $SE = 5.51$, $t = 2.54$, $p = .011$) window conditions. Thus, people with dyslexia exhibited reductions in parafoveal processing for all window sizes, except the 14-character window (see Fig. 1)—a pattern similar to that found for schizophrenia participants vs. controls, even when controlling for medication (Note: the schizophrenia vs. control group results are not repeated for parsimony; see Appendix—Table A.6 of the Appendix).

To examine the size of the perceptual span in each participant group, we ran separate sub-models for the dyslexia and control groups. The dyslexia sub-model revealed that relative to the full text condition, reading rates were significantly slower in the 2-character (106 vs. 157 words/min; $\beta = -53.56$, $SE = 3.45$, $t = -15.55$, $p < .001$), 6-character (141 vs. 157 words/min; $\beta = -14.79$, $SE = 3.45$, $t = -4.29$, $p < .001$), and 10-character (149 vs. 157 words/min; $\beta = -9.92$, $SE = 3.42$, $t = -2.90$, $p = .004$) window conditions. The control sub-model revealed the same pattern; reading rates were also significantly slower in the 2-character (122 vs. 206 words/min; $\beta = -83.21$, $SE = 4.41$, $t = -18.86$, $p < .001$), 6-character (176 vs. 206 words/min; $\beta = -30.05$, $SE = 4.38$, $t = -6.87$, $p < .001$), and 10-character (183 vs. 206 words/min; $\beta = -22.57$, $SE = 4.38$, $t = -5.15$, $p < .001$) window conditions. Thus, both groups' perceptual spans were ~14 characters rightward of fixation, as the perceptual span estimates (reading rates) were not reduced at that window size. These results differ from those found for schizophrenia participants vs. controls: ~6 vs. ~14 characters rightward of fixation (see Whitford et al., 2013).

Table 10
Model specifications for the schizophrenia vs. dyslexia group comparison (perceptual span).

Fixed effect	Control predictors/covariates	Random effects
▪ Clinical status ▪ Window size	▪ Age (continuous) ▪ Native language ▪ Verbal IQ (continuous) ▪ Parental SES (continuous) ▪ Education (continuous) ▪ List order ▪ Trial number (continuous) ▪ Chlorpromazine equivalent dose (continuous)	▪ By-participant random intercepts ▪ By-item random intercepts

Note 1: Clinical status (schizophrenia vs. dyslexia) was treatment coded (baseline = schizophrenia); window size (full text vs. 2-, 6-, 10-, and 14-character windows) was treatment coded (baseline = full text); native language (English vs. French) was deviation coded (−0.5, +0.5); and list order (1 vs. 2 vs. 3) was deviation coded (+1, 0; 0, +1; −1, −1).
Note 2: All continuous control predictors/covariates were centered and scaled (i.e., standardized, z-scored) to reduce collinearity.

3.2.2. Schizophrenia and dyslexia groups

The model specifications follow those reported previously (see Table 10 and Appendix—Table A.7 for complete model output).

The effect of clinical status was non-significant, whereas that of window size was. Compared to the full text condition, reading rates were slower in the 2-character window condition (101 vs. 148 words/min; $\beta = -46.85$, $SE = 3.38$, $t = 13.88$, $p < .001$), but faster in the 14-character window condition (153 vs. 148 words/min; $\beta = 13.03$, $SE = 3.34$, $t = 3.90$, $p < .001$). The interaction between clinical status and window condition was significant. Compared to the schizophrenia group, the dyslexia group's reading rates were more negatively impacted (relative to the full text condition) by the 6-character (difference of 16 vs. -1 words/min; $\beta = -15.57$, $SE = 4.78$, $t = -3.26$, $p = .001$), 10-character (difference of 8 vs. -8 words/min; $\beta = -15.14$, $SE = 4.76$, $t = -3.18$, $p = .002$), and 14-character (difference of 2 vs. -12 words/min; $\beta = -13.51$, $SE = 4.77$, $t = -2.83$, $p = .005$) window conditions. Thus, people with dyslexia exhibited greater parafoveal processing for all window sizes,

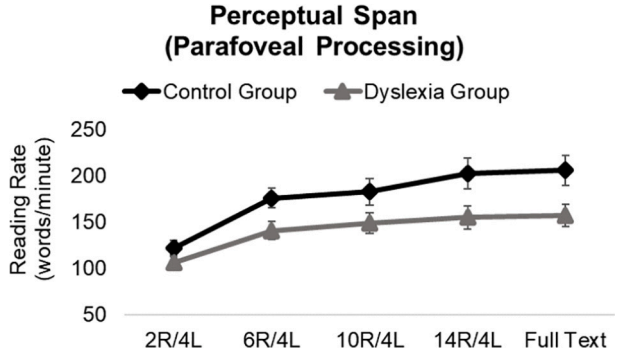

Fig. 1. Reading rates (mean values) across the different window conditions for the dyslexia and control groups. Error bars represent standard errors of the mean.

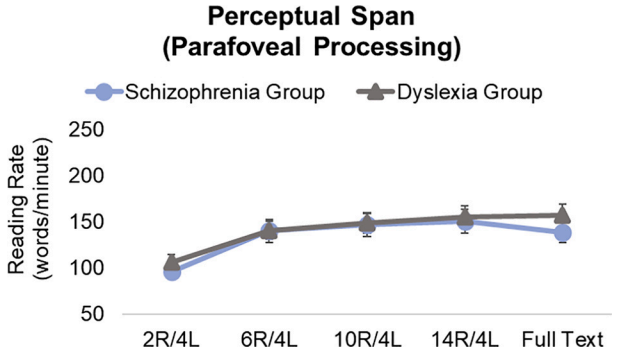

Fig. 2. Reading rates (mean values) across the different window conditions for the schizophrenia and dyslexia groups. Error bars represent standard errors of the mean.

except the 2-character window, than people with schizophrenia (see Fig. 2). As presented earlier in the sub-model analyses, people with dyslexia also exhibited a larger perceptual span than people with schizophrenia: ~14 vs. ~6 characters rightward of fixation.

4. Discussion

The present work represents the first head-on comparison of reading in schizophrenia and reading in dyslexia. Using a gaze-contingent moving window task, we found that psychiatrically healthy adults with dyslexia (a population whose reading behaviour has been little investigated) exhibit robust oculomotor markers of sentence-level reading difficulty compared to matched controls, including slower reading rates, shorter forward saccades, more forward fixations, and more regressions—a pattern similar to that found for adults with schizophrenia compared to matched controls (dataset from Whitford et al., 2013). While these findings are consistent with the limited eye-tracking reading research on schizophrenia (Dias et al., 2021; Fernández et al., 2016a; Fernández et al., 2016b; Roberts et al., 2013; Whitford et al., 2013; reviewed in Whitford et al., 2018) and adult dyslexia (Franzen et al., 2021; Jones et al., 2007; Zangwill and Blakemore, 1972; reviewed in Whitford et al., 2018), our work links these separate bodies of research by demonstrating that eye movement reading behaviour is similarly impaired in both conditions (no significant group differences were observed). Though a secondary focus, we also found similar impairments in neurocognitive processes that support reading: language and higher-order oculomotor control/executive functioning. Thus, our findings demonstrate that reading and reading-related functions are comparably impaired in schizophrenia and dyslexia.

Using a gaze-contingent moving window task also allowed us to examine the perceptual span (breadth of parafoveal processing) between schizophrenia and dyslexia. We found an intact perceptual span in adults with dyslexia (~14 characters rightward of fixation), although their reading fluency was less negatively impacted by restrictive window sizes compared to matched controls (indicative of reduced parafoveal processing). In contrast, both the perceptual span (~6 characters rightward of fixation) and parafoveal processing for restrictive window sizes were reduced in adults with schizophrenia compared to matched controls (see also Whitford et al., 2013), as well as compared to the dyslexia group. While the schizophrenia group's reduced perceptual span during reading is consistent with the limited extant research in this area (Roberts et al., 2013; see also Elahipanah et al., 2011, for similar reductions during non-linguistic visual search) and other related areas, including research involving developing, less skilled, and/or second-language readers (Bélanger et al., 2012; Häikiö et al., 2009; Rayner, 1986; Rayner et al., 2010; Sperlich et al., 2016; Whitford and Titone, 2015; Whitford and Titone, 2016), the dyslexia group's intact allocation of visual attention during reading is not. For instance, Rayner et al. (1989) reported reductions in perceptual span (~7 vs. ~15 characters rightward of fixation) and parafoveal processing in an adult with dyslexia relative to controls (see also Jones et al., 2013; Silva et al., 2016, for similar reductions in parafoveal preview benefits during rapid automatized naming).

Given the interdependence between foveal and parafoveal processing (i.e., fewer visuo-attentional resources are allocated to parafoveal processing when foveal processing load or word encoding difficulty increases; reviewed in Henderson and Ferreira, 1990; Schotter et al., 2012), the schizophrenia group's reading fluency deficits (e.g., slower reading rates), potentially driven by greater foveal processing loads, may have contributed to their reductions in parafoveal processing (see Whitford et al., 2013, for a more detailed discussion). Relatedly, considering that the lexical control of eye movements is mediated by the extraction of visual information to the right of fixation (reviewed in Andrews and Veldre, 2019; Reichle and Reingold, 2013; Schotter et al., 2012) and that our measures of reading fluency (e.g., reading rates during full text reading) may have probed both foveal and parafoveal processing, the schizophrenia group's reductions in parafoveal processing may have contributed to their reading fluency deficits (see also Dias et al., 2021; Roberts et al., 2013, for additional visuo-oculomotor contributions to reading deficits in schizophrenia).

However, despite experiencing similar deficits in reading fluency (e.g., comparably reduced reading rates), which may have been driven by similarly heightened foveal processing loads, the dyslexia group exhibited a larger perceptual span and greater parafoveal processing than the schizophrenia group—potentially reflecting a dissociation between foveal and parafoveal processing. A similar dissociation has been found in previous research by our group involving healthy bilingual older adults, where they too maintained an intact perceptual span, despite experiencing age-related word encoding difficulties (Whitford and Titone, 2016). This reading strategy was, however, adaptive in nature, as those with better executive functioning (e.g., higher backward digit span scores) differentially employed it. As such, their reading strategy reflected a prioritization of parafoveal processing (additional time was allocated during fixation to maintain an age-invariant attentional span) rather than a disruption in foveal-parafoveal processing dynamics. To determine whether our dyslexia group employed a similar strategy, we examined the relationship between perceptual span and executive functioning (antisaccade task performance). However, our post hoc analyses failed to reveal a significant association ($p > .05$). Thus, the dyslexia group may have employed a sub-optimal reading strategy, driven by a disruption in normal foveal-parafoveal processing dynamics.

Another potential explanation for the differences in perceptual span and parafoveal processing between the dyslexia and schizophrenia groups comes from research reporting superior peripheral detail vision in both children and adults with dyslexia. For instance, studies have found that relative to controls, people with dyslexia are better at correctly identifying letters and letter strings presented in the peripheral visual field, with detrimental effects to letter and letter string identification in the foveal field (Geiger and Lettvin, 1987; Lorusso et al., 2004; Perry et al., 1989; but see Klein et al., 1990; Slaghuis et al., 1992, for conflicting findings). This may reflect a different perceptual strategy, where some people with dyslexia are more apt at extracting visual information to the right of fixation, yet fail to benefit from the expected parafoveal priming advantage when words are identified foveally during fixation.

Taken together, the similar disturbances in reading and reading-related functions (except perceptual span and parafoveal processing) between our schizophrenia and dyslexia samples lend further support for a common neurodevelopmental basis, potentially driven by developmental brain dysfunction (Moreno-De-Luca et al., 2013). Although the generalizability of our findings may be limited by our small sample sizes, our findings provide some elucidation on the shared nature and magnitude of reading impairments between the disorders. Continued research in this area is crucial to early intervention and remediation strategies centred on addressing reading and other neurocognitive impairments in these disorders, as well as improving the functional independence and quality of life of those experiencing them.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A

Table A.1
Demographic characteristics of the more closely matched schizophrenia and dyslexia samples.

	Schizophrenia group ($n = 12$)	Dyslexia group ($n = 19$)
	Mean (SD)	Mean (SD)
Sex (male-to-female ratio)***	10:2	5:14
Age (years)	26.08 (3.90)	23.00 (5.39)
Native language (English-to-French ratio)*	5:7	16:3
Verbal IQ (scaled scores)	12.50 (3.09)	13.79 (1.99)
Parental SES	3.92 (2.27)	2.58 (0.90)
Education (years)**	12.04 (2.14)	14.29 (1.85)

Note 1: Sex was based on self-reported assigned sex at birth; native language was based on an adaptation of the Language Experience and Proficiency Questionnaire (LEAP-Q; Marian et al., 2007); verbal IQ was based on the Vocabulary subtest of the Wechsler Adult Intelligence Scale–Revised (WAIS–R; Wechsler, 1981); and parental SES was based on the Hollingshead Occupational Scale (Hollingshead, 1975).
Note 2: *$p < .05$; **$p < .01$; ***$p < .001$.

Table A.2
Effect sizes (β), standard errors (SE), *t* values, and *p* values for no-window (full text) reading in the dyslexia vs. control groups.

	Reading rate				Forward saccade length				Forward fixation duration				Number of fixations				Number of regressions			
	β	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*
Fixed effect																				
Clinical status	−44.93	12.59	−3.57	0.001**	−0.89	0.42	−2.12	0.044*	7.40	7.55	0.98	0.336	1.52	0.48	3.14	0.004**	1.60	0.38	4.19	0.000***
Control predictors																				
Age	−1.89	6.64	−0.28	0.779	−0.09	0.22	−0.42	0.680	−0.28	3.98	−0.07	0.945	0.02	0.26	0.09	0.928	0.00	0.20	0.01	0.989
Years of education	2.79	6.92	0.40	0.690	−0.06	0.23	−0.24	0.811	−0.44	4.15	−0.11	0.917	−0.10	0.27	−0.38	0.709	−0.04	0.21	−0.20	0.842
Parental SES	−8.22	7.44	−1.11	0.278	−0.40	0.25	−1.61	0.119	8.32	4.46	1.87	0.073	0.22	0.29	0.78	0.441	0.00	0.23	0.00	0.998
Verbal IQ	6.96	7.50	0.93	0.361	−0.11	0.25	−0.44	0.665	−3.73	4.50	−0.83	0.414	−0.10	0.29	−0.36	0.726	−0.10	0.23	−0.44	0.666
Native language	−0.15	17.17	−0.01	0.993	−0.31	0.57	−0.55	0.589	1.66	10.29	0.16	0.873	0.21	0.66	0.32	0.751	0.34	0.52	0.65	0.523
List order1	−31.41	33.66	−0.93	0.365	−0.15	0.89	−0.17	0.869	−5.37	12.84	−0.42	0.680	−1.59	1.63	−0.97	0.345	0.01	0.77	0.01	0.990
List order2	36.21	33.46	1.08	0.296	0.50	0.88	0.56	0.580	−3.00	12.63	−0.24	0.815	1.18	1.63	0.73	0.479	0.01	0.76	0.01	0.993
Trial number	0.98	1.09	0.90	0.385	0.00	0.03	−0.05	0.964	0.33	0.39	0.84	0.415	0.05	0.05	0.98	0.344	−0.01	0.02	−0.31	0.765
Intercept	167.05	42.78	3.91	0.001**	8.29	1.12	7.40	0.000***	196.59	16.03	12.26	0.000***	5.18	2.08	2.49	0.026*	1.51	0.96	1.56	0.137

Random effects	Variance	Variance	Variance	Variance	Variance
	Intercept	Intercept	Intercept	Intercept	Intercept
Participants	1125.56	1.22	399.77	1.72	1.04
Items	269.48	0.13	16.35	0.75	0.10
Residual	2326.77	3.13	915.73	2.58	2.01

Table A.3
Effect sizes (β), standard errors (SE), t values, and p values for no-window (full text) reading in the schizophrenia vs. control groups.
Note: *$p < .05$; **$p < .01$; ***$p < .001$.

	Reading rate				Forward saccade length				Forward fixation duration				Number of forward fixations				Number of regressions			
	β	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*
Fixed effect																				
Clinical status	−50.22	17.30	−2.90	0.008**	−1.35	0.71	−1.90	0.068	39.39	13.76	2.86	0.009**	1.78	0.66	2.71	0.012*	1.12	0.43	2.62	0.014*
Control predictors																				
Age	4.38	6.97	0.63	0.536	0.18	0.29	0.62	0.543	2.63	5.57	0.47	0.641	−0.15	0.26	−0.55	0.585	−0.07	0.25	−0.29	0.776
Years of education	6.19	8.75	0.71	0.486	0.21	0.35	0.60	0.553	4.33	6.88	0.63	0.534	−0.47	0.33	−1.41	0.172	−0.50	0.31	−1.59	0.123
Parental SES	−16.70	6.83	−2.45	0.022*	−0.53	0.28	−1.91	0.067	9.32	5.45	1.71	0.100	0.61	0.26	2.35	0.027*	−0.10	0.24	−0.40	0.691
Verbal IQ	−0.02	7.94	0.00	0.998	0.26	0.32	0.79	0.438	0.34	6.31	0.05	0.958	−0.21	0.30	−0.70	0.489	−0.15	0.28	−0.54	0.592
Native language	21.45	14.94	1.44	0.164	1.75	0.61	2.87	0.008**	−2.14	11.90	−0.18	0.859	−1.94	0.57	−3.42	0.002**	−0.68	0.53	−1.27	0.214
List order1	5.73	17.25	0.33	0.742	1.06	0.59	1.79	0.082	−7.98	10.87	−0.73	0.467	−0.39	0.67	−0.58	0.565	−0.01	0.60	−0.02	0.983
List order2	2.87	15.62	0.18	0.856	−0.24	0.55	−0.43	0.671	6.55	10.32	0.64	0.529	−0.29	0.60	−0.47	0.640	0.06	0.54	0.11	0.912
Trial number	−0.14	0.54	−0.25	0.801	−0.02	0.02	−1.43	0.163	0.42	0.30	1.42	0.167	0.01	0.02	0.56	0.582	0.01	0.02	0.29	0.770
Chlorpromazine equivalent dose	−14.97	8.50	−1.76	0.091	−0.54	0.35	−1.54	0.137	6.25	6.82	0.92	0.369	0.62	0.32	1.93	0.065	0.26	0.30	0.86	0.397
Intercept	204.42	25.69	7.96	0.000***	9.53	0.85	11.20	0.000***	184.83	15.43	11.98	0.000***	6.43	1.01	6.38	0.000***	1.50	0.88	1.70	0.098

Random effects	Variance	Variance	Variance	Variance	Variance
	Intercept	Intercept	Intercept	Intercept	Intercept
Participants	1064.53	1.90	719.40	1.54	1.41
Items	272.81	0.08	12.18	0.61	0.26
Residual	2172.44	2.20	894.45	2.93	1.87

Note: *$p < .05$; **$p < .01$; ***$p < .001$.

Table A.4
Effect sizes (β), standard errors (SE), *t* values, and p values for no-window (full text) reading in the schizophrenia vs. dyslexia groups.

	Reading rate				Forward saccade length				Forward fixation duration				Number of fixations				Number of regressions			
	β	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*	*β*	*SE*	*t*	*p*
Fixed effect																				
Clinical status	−2.92	15.31	−0.19	0.850	0.14	0.61	0.23	0.818	−14.23	14.81	−0.96	0.344	0.29	0.88	0.33	0.742	0.77	0.69	1.12	0.274
Control predictors																				
Age	−0.47	6.56	−0.07	0.943	0.50	0.26	1.92	0.064	6.33	6.24	1.02	0.318	−0.41	0.38	−1.08	0.291	0.06	0.29	0.20	0.842
Years of education	−3.90	6.46	−0.60	0.551	0.03	0.26	0.11	0.911	0.52	6.24	0.08	0.934	−0.23	0.37	−0.63	0.537	0.01	0.29	0.02	0.983
Parental SES	−10.75	5.61	−1.92	0.065	−0.42	0.22	−1.86	0.073	9.66	5.39	1.79	0.083	0.54	0.32	1.66	0.108	−0.02	0.25	−0.09	0.932
Verbal IQ	3.70	6.88	0.54	0.595	0.54	0.27	1.97	0.058	7.27	6.56	1.11	0.277	−0.47	0.40	−1.19	0.245	−0.25	0.31	−0.83	0.414
Native language	15.88	12.50	1.27	0.214	1.12	0.50	2.25	0.032*	3.83	12.00	0.32	0.752	−1.12	0.72	−1.55	0.132	−0.57	0.56	−1.01	0.320
List order1	−9.38	23.30	−0.40	0.690	0.81	0.69	1.17	0.251	9.85	12.80	0.77	0.448	−1.06	1.38	−0.77	0.447	0.04	0.97	0.04	0.967
List order2	14.88	22.04	0.68	0.504	0.27	0.65	0.41	0.683	−11.47	12.12	−0.95	0.352	0.24	1.31	0.18	0.857	0.24	0.92	0.26	0.795
Trial number	0.43	0.74	0.58	0.568	−0.02	0.02	−0.88	0.388	0.01	0.36	0.03	0.980	0.03	0.04	0.70	0.486	−0.01	0.03	−0.23	0.824
Chlorpromazine equivalent dose	−13.71	7.10	−1.93	0.064	−0.35	0.29	−1.23	0.228	2.75	6.86	0.40	0.692	0.40	0.41	0.97	0.341	0.28	0.32	0.89	0.384
Intercept	136.24	28.63	4.76	0.000***	7.92	0.84	9.38	0.000***	236.38	15.71	15.05	0.000***	7.47	1.70	4.39	0.000***	2.35	1.19	1.98	0.057

Random effects	Variance	Variance	Variance	Variance	Variance
	Intercept	Intercept	Intercept	Intercept	Intercept
Participants	648.39	1.11	656.05	2.32	1.38
Items	221.92	0.10	14.88	0.91	0.34
Residual	1829.77	1.96	968.37	3.74	2.49

Note: *$p < .05$; **$p < .01$; ***$p < .001$.

Table A.5
Effect sizes (β), standard errors (*SE*), t values, and p values for reading rate estimates of perceptual span in the dyslexia vs. control groups.

	Reading rate			
	β	*SE*	*t*	*p*
Fixed effects				
Clinical status	−46.98	8.83	−5.32	0.000***
4 L/14R condition	−1.00	4.02	−0.25	0.804
4L/10R condition	−23.40	4.01	−5.84	0.000***
4L/6R condition	−29.33	4.01	−7.32	0.000***
4L/2R condition	−84.82	4.02	−21.09	0.000***
Clinical status × 4L/14R condition	−0.52	5.52	−0.10	0.924
Clinical status × 4L/10R condition	14.01	5.51	2.54	0.011*
Clinical status × 4L/6R condition	13.37	5.52	2.42	0.016*
Clinical status × 4L/2R condition	32.40	5.52	5.87	0.000***
Control predictors				
Age	−0.79	4.28	−0.18	0.855
Years of education	0.47	4.45	0.11	0.916
Parental SES	−7.27	4.79	−1.52	0.141
Verbal IQ	2.75	4.84	0.57	0.574
Native language	−8.23	11.06	−0.74	0.463
List order1	−8.88	6.18	−1.44	0.162
List order2	12.64	5.62	2.25	0.033*
Trial number	0.16	0.04	3.59	0.000***
Intercept	196.58	7.92	24.83	0.000***

Random effects	Variance
	Intercept
Participants	504.82
Items	101.01
Residual	2036.04

Note: *$p < .05$; **$p < .01$; ***$p < .001$; L = characters to the left of fixation; R = characters to the right of fixation.

Table A.6
Effect sizes (β), standard errors (SE), *t* values, and p values for reading rate estimates of perceptual span in the schizophrenia vs. control groups.

	Reading rate			
	β	*SE*	*t*	*p*
Fixed effects				
Clinical status	−56.84	15.06	−3.78	0.000***
4L/14R condition	10.92	4.20	2.60	0.009**
4L/10R condition	−13.32	4.12	−2.93	0.000***
4L/6R condition	−15.01	4.21	−3.56	0.000***
4L/2R condition	−85.09	4.13	−20.63	0.000***
Clinical status × 4L/14R condition	2.12	5.55	0.38	0.702
Clinical status × 4L/10R condition	8.60	5.54	2.01	0.019*
Clinical status × 4L/6R condition	15.25	5.60	2.72	0.007**
Clinical status × 4L/2R condition	37.96	5.59	6.79	0.000***
Control predictors				
Age	0.00	5.90	0.00	1.00
Years of education	−2.25	7.17	−0.31	0.756
Parental SES	−14.66	5.80	−2.53	0.018*
Verbal IQ	2.67	6.71	0.40	0.694
Native language	19.19	12.66	1.52	0.142
List order1	−3.12	7.78	−0.40	0.691
List order2	8.47	8.37	1.01	0.321
Trial number	0.27	0.04	6.09	0.000***
Chlorpromazine equivalent dose	−10.78	7.28	−1.48	0.150
Intercept	192.40	10.56	18.22	0.000***

Random effects	Variance
	Intercept
Participants	867.82
Items	139.11
Residual	1997.54

Note: *$p < .05$; **$p < .01$; ***$p < .001$; L = characters to the left of fixation; R = characters to the right of fixation.

Table A.7
Effect sizes (β), standard errors (SE), *t* values, and p values for reading rate estimates of perceptual span in the schizophrenia vs. dyslexia groups.

	Reading rate			
	β	*SE*	*t*	*p*
Fixed effects				
Clinical status	1.23	14.82	0.08	0.935
4L/14R condition	13.03	3.34	3.90	0.000***
4L/10R condition	5.45	3.34	1.63	0.103
4L/6R condition	0.30	3.32	0.09	0.927
4L/2R condition	−46.85	3.38	−13.88	0.000***
Clinical status × 4L/14R condition	−13.51	4.77	−2.83	0.005**
Clinical status × 4L/10R condition	−15.14	4.76	−3.18	0.002**
Clinical status × 4L/6R condition	−15.57	4.78	−3.26	0.001**
Clinical status × 4L/2R condition	−6.26	4.77	−1.31	0.190
Control predictors				
Age	−3.97	6.08	−0.65	0.519
Years of education	−2.72	6.10	−0.45	0.659
Parental SES	−11.74	5.27	−2.23	0.034*
Verbal IQ	0.73	6.40	0.12	0.910
Native language	6.88	11.72	0.59	0.562
List order1	−8.69	6.81	−1.28	0.212
List order2	11.59	6.44	1.80	0.082
Trial number	0.26	0.04	6.80	0.000***
Chlorpromazine equivalent dose	−6.05	6.71	−0.90	0.375
Intercept	139.07	9.16	15.18	0.000***

Random effects	Variance
	Intercept
Participants	669.82
Items	86.73
Residual	1643.38

Note: *$p < .05$; **$p < .01$; ***$p < .001$; L = characters to the left of fixation; R = characters to the right of fixation.

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