Normal saccades but decreased fixation stability in a population of children with dyslexia

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Received April 14, 2021, accepted November 7, 2021.

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Abstract

Developmental dyslexia affects around 5–15% of the population and has a heterogeneous aetiology. Optometric disorders are more prevalent in dyslexic populations but the relationship between eye movement control and dyslexia is not well established. In this study, we investigated whether children with dyslexia show saccadic or fixation deficits and whether these deficits are related to deficits in visual acuity and/or accommodation.

Thirty-four children with and without dyslexia were recruited for the project. All participants had an optometric examination and performed a saccade and fixation experiment. We used two eye movement paradigms: the step and the gap task. Eye movements were recorded by an infrared eye-tracker and saccade and fixation parameters were analysed separately.

Saccadic latencies, premature saccades, and directional errors were similar between children with dyslexia and typically developing children. In contrast, fixations were significantly less stable in the dyslexic group. Neither saccades nor fixations were associated with deficits in accommodation or visual acuity.

Children with dyslexia showed no difficulties in saccadic performance, but their fixation stability was reduced compared to the control group. The reduced fixation stability can be explained by general deficits in the cognitive processes that underpin eye movement control, that have also been found in other neuro-developmental disorders.

Keywords: Eye movements, dyslexia, fixation, saccades

Introduction

Developmental dyslexia is a prevalent condition affecting about 5-15% of the population (Heim et al., 2008; Helland et al., 2011; Schulte-Korne, 2010; Shaywitz et al., 2006). A child with developmental dyslexia (hereafter dyslexia) struggles with word recognition, spelling and word decoding, and therefore finds reading demanding (Bishop & Snowling, 2004). It is important that children with dyslexia are identified quickly and provided with effective interventions to prevent disruption to academic development (Bishop & Snowling, 2004). In Norway, dyslexia is commonly diagnosed around the age of 10–11 years, which is when the importance of learning through text increases (Morken & Helland, 2013). While efforts have been made to identify children at an earlier age, there is currently no program for early detection or intervention for at-risk children (Helland et al., 2011). There is no consensus on how to assess dyslexia and there is a lack of valid screening tools as well as a "gold standard" for diagnosis (Nergård-Nilssen & Eklund, 2018).

The phonological deficit theory is a well-known and often referenced explanation for dyslexia (Melby-Lervåg et al., 2012). In spite of its importance, a phonological deficit does not explain all facets of dyslexia, and a deficit in visual attention has been suggested as an additional risk factor (Leonard et al., 2002; Peterson & Pennington, 2012; Vidyasagar, 2019). Reading requires both spatial and temporal integration of multiple still-pictures from fixations across several saccades. Correct sequencing of letters during reading is an extensive task for the brain, and problems with this sequencing are not solely due to a phonological deficit (Leonard et al., 2002; Vidyasagar & Pammer, 2010; Williams & Lecluyse, 1990). Recent longitudinal studies have added knowledge about other important correlations between cognitive functions and reading performance (Peterson & Pennington, 2012; Vidyasagar, 2019). These may contribute to a broader understanding of the aetiology of dyslexia and help with early diagnosis and recognition of the problem. For instance, it has been suggested that visuo-spatial memory could be an early marker of literacy skills in transparent orthographies like Norwegian (Nergård-Nilssen & Eklund, 2018).

There is evidence for both visual and oculomotor deficits in dyslexia (Bucci et al., 2008b; Stein, 2014). These deficits are most often attributed to a dysfunction of the magnocellular pathway, specifically a visuospatial attention deficit (see for instance (Stein, 2014)). However, there is no consensus with regards to the presence of a visuospatial deficit in dyslexia. For instance, Lukov et al. examined 110 adults and children with dyslexia and/or attention deficit, and found that all types of dyslexia were in fact dissociated with attention problems (Lukov et al., 2014). Vidyasagar have suggested that there are two possible explanations for dyslexia in addition to a phonological deficit. One is a deficit in visual spatial attention and the other is a deficit in synchronised neuronal oscillations which are essential for communication between brain areas (Vidyasagar, 2019). The latter may contribute to the understanding of the prevalent comorbidities between dyslexia and other developmental disorders, due to impaired cerebellar functions (Nicolson et al., 1999; Stoodley & Stein, 2013).

Atypical eye movement pattern has been frequently observed in dyslexics during reading (Kulp & Schmidt, 1996; Rayner, 1998; Rommelse et al., 2008). It is generally accepted that the eye movements reflect the visual processing and not the actual ability to move the eyes. However, research in this area has not concluded whether the oculomotor disorder is primary or secondary to the decoding problem (Quercia et al., 2013). Eye movement control is frequently examined in studies but there is no clear consensus about the associations with dyslexia.

The "visual attention" construct is extremely broad but measures of saccades can provide a useful operationalisation of this construct (Kowler et al., 1995). It follows that measures related to the integrity of saccadic control provide a test of the hypothesis that visual attention contributes towards dyslexia. A deficit in visuospatial attention can therefore be indexed through an increase in saccadic latency (Bellocchi et al., 2013). There have been a number of studies that have measured saccadic latency in dyslexia to test the hypothesis that saccadic latency is reduced in dyslexics. These studies have not yielded consistent findings: both longer (Biscaldi et al., 1998; Bucci et al., 2008a), similar (Bucci et al., 2014) and shorter (Bednarek et al., 2006) saccadic latencies have been reported in dyslexia.

The allocation of visual attention through eye movements also requires fixation to remain stable once a visual target has

been acquired through a shift in eye position. It has been proposed that the fixation instability observed in children with dyslexia reflects decreased cognitive control rather than oculomotor dysfunction per se (Vagge et al., 2015). This interpretation is supported by the fact that children with dyslexia have normal eye movements when they perform tasks with control demands similar to reading but without the "cognitive" component (Hutzler et al., 2006). However, Kapoula, Bucci and their colleagues have published several studies on the neurophysiology of eye movements in dyslectic children, and they have found that dyslexics have poor binocular coordination of saccades, during a saccade the eyes show more variable conjugacy and after the saccade they have larger drift resulting in fixation instability (Bucci et al., 2008a; 2008b; Jainta & Kapoula, 2011; Kapoula et al., 2007). Other studies have suggested that children with dyslexia have unstable binocular fixation (Castro et al., 2008; Vagge et al., 2015), and monocular instability has also been reported (Biscaldi et al., 1994; Fischer, 2012). These findings suggest that unstable fixation might be prevalent in a population of children with dyslexia.

The aim of this study was to investigate if saccadic latency and fixation stability were different in children with developmental dyslexia (DD) and typically developing children (TD) when performing a non-reading task. We used two different eye-movement paradigms: the saccadic step task and the saccadic gap task. We used these two paradigms as different tasks can result in different saccade latencies. When observers are instructed to look at a central fixation point and then make an eye movement to a visual stimulus presented in the periphery, this is often referred to as a visually guided saccade (Rommelse et al., 2008). When the central fixation point is extinguished at the same time as the stimulus appears, the task is called the step task. In general, the latency of visually guided (step) saccades in healthy adults is around 200 ms with a standard deviation of about 10% (Holmqvist et al., 2011). When a central fixation point is extinguished before the peripheral stimulus is presented, saccadic latency decreases, and this is known as the gap effect. It is assumed that saccades in the gap task are more reflexive and influenced to a lesser degree by higher level cognitive processes compared to saccades in the step task (Kristjànsson2011, 2011). It has also been reported that step latency decreases as the child gets older whereas gap latency does not change significantly with development (Bucci et al., 2012).

The relationship between accommodation, visual acuity and fixation stability have been investigated previously (Evans et al., 1994; Vikesdal et al., 2020; Wahlberg-Ramsay et al., 2012; Ygge et al., 1993). Some individuals with dyslexia exhibit a reduction in acuity compared to controls, and lower levels of accommodation amplitude has been reported (Evans et al., 1994; Vikesdal et al., 2020; Wahlberg-Ramsay et al., 2012; Ygge et al., 1993). In a previous study we found that degraded visual acuity and active accommodation (induced by adding positive and negative refractive lenses) resulted in a decrease in fixation stability, suggesting that uncorrected refractive errors might be a contributing factor to poor fixation stability. In contrast, saccadic latency was independent of changes to visual acuity and accommodation (Vikesdal & Langaas, 2016a).

The lack of consistency in previous studies made it difficult to generate formal hypotheses about the expected findings. Nevertheless, previous research has indicated that some, but not all, children with dyslexia may have poor fixation relative to their peers (Raymond et al., 1988). We also predicted that the children with dyslexia would not show saccadic abnormalities, based on evidence showing that dyslexia is a phonological problem (Norton et al., 2015). Moreover, the fact that children with specific motor difficulties do not show saccadic abnormalities (Gonzalez et al., 2016; Sumner et al., 2016) suggested it would be unlikely that children with dyslexia would have problems with the sub-cortical systems associated with saccade generation. We tested these predictions in an empirical study of saccadic eye movements and fixation stability in children with and without dyslexia.

Materials and methods

Study Sample

In Norway, developmental dyslexia is typically diagnosed when the child is around age 10–11, after children are referred by their teacher to the local Educational and Psychological Counselling Service. This is a governmental body responsible for the investigation and counselling of children with learning difficulties. Children with confirmed developmental dyslexia (DD) who attended the local Educational and Psychological Counselling Service during the study period were invited to participate. An age-matched control group of typically developing children (TD) was recruited from the same school catchment areas through information meetings with parents at school and advertising in the local newspaper. Twenty-three DD and 17 TD children participated with informed consent. Children gave verbal assent, and the primary carer signed the informed consent. The experiment was conducted in accordance with the Declaration of Helsinki ("WMA Declaration of Helsinki - Ethical Principles for Medical Research Involving Human Subjects", 2013), and was approved by the Regional Committees for Medical and Health Research Ethics.

Participants were all healthy with normal vision, no developmental disorders (besides dyslexia), no prematurity, no history of neurological disease or use of medication, and Norwegian was their primary language. Participants were tested with a linguistics test for detection of language problems ("Språk 6–16"). This test is standardised for Norwegian school children and consists of several subtests, including measurements of reading speed, phonological ability and verbal short-time memory (Ottem & Frost, 2011). Children were included in the DD group if the Educational and Psychological Counselling Service had diagnosed them with dyslexia and their phonological ability score from the linguistics test was below 1 *SD* of the mean. Children were included in the TD group if they had no history of dyslexia or reading problems and scored within the normal range (mean ± 1 *SD*) on all subtests of the language tests.

All participants had a thorough optometric examination including cycloplegic refraction, logMAR visual acuity, stereoacuity, accommodation and binocular vision assessment. Accommodation and binocular vision were tested and analysed according to established clinical criterion (Scheiman & Wick, 2002). The test procedures are described in a previous publication (Vikesdal et al., 2020). The preferred sighting eye was determined by a sighting test at distance (6 m) and near (40 cm). Sighting tests have high test-retest reliability, and the vast majority of studies agree that there is a sighting-preferred eye for each person (Mapp et al., 2003; Rice et al., 2008). This sighting test is recommended for determining the eye to track when measuring eye movements (Holmqvist et al., 2011), and has been reported as being clinically repeatable (Rice et al., 2008).

Procedure

Children were brought to a dimly illuminated room for testing. Participants sat in a firmly mounted chair 100 cm from a computer screen adjusted in height, so the eyes were in line with the centre of the screen. A chin- and forehead rest was used to minimise head movements. The stimuli were presented on a fast phosphor monitor with a 100 Hz refresh rate (Phillips 20T), size 56 cm (22") and a resolution of 1024×768 pixels. The background on the monitor was dark grey with an even luminance

of 22 cd/m^2 across the screen. The stimulus was light yellow, with an even luminance of 276 cd/m^2, thus the contrast level was 92%.

Participants performed saccades and fixations in oculomotor paradigms including both the step- and gap tasks. In the step task, each trial started with the appearance of a fixation cross (side length 0.4°) at the centre of a computer screen. After one second, the fixation cross was extinguished and simultaneously, the stimulus (a dot 0.2° in diameter) appeared in one of eight possible stimulus positions chosen at random (see Figure 1). The stimulus was visible for 2 seconds. The gap task was identical to the step task, except there was a 200 milliseconds (ms) gap between the fixation cross extinguishing and the appearance of the stimulus. In both tasks, participants were instructed to look at the stimulus as quickly and accurately as possible. They performed two practice runs prior to the experiment and there were breaks between the tasks. There were 48 trials in the step task and 48 trials in the gap task. Depending on the ease of recording and the need for breaks, the experiment lasted 10 to 20 minutes for each subject.

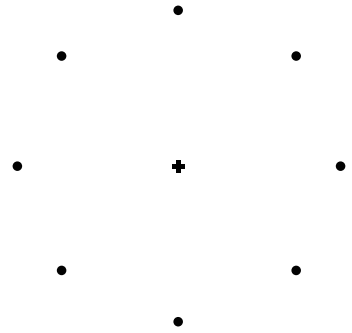

Figure 1: *Possible stimulus positions, placed at the vertices of a regular octagon, 5° from the fixation cross. Stimulus positions were the same in both the step task and the gap task.*

Eye movements were recorded with both eyes open to allow both accommodation and vergence as in natural viewing. Vertical and horizontal positions of the preferred sighting eye were measured with an IScan ETL-300 video-based eye-tracking system, which had a temporal resolution of 8 ms and a spatial resolution of 0.161° (RMS) (Vikesdal & Langaas, 2016b). Prior to each experimental session, the eye-tracker was calibrated by the presentation of five 0.5° boxes, located in the centre and in the four corners of a square subtending 20°×20°. Eye position data, with accompanying time stamps, were exported for post-experimental analysis.

Data Analysis

The first trial of each task and trials where the participant blinked were not included in the analysis.

Saccades

Saccadic latency is the time between stimulus appearance and saccade onset. Saccade onset was defined as the time at which eye velocity exceeded 20°/s and lasted for more than 32 ms (i.e. four consecutive eye tracker sampling points). A trial started with the child fixating the central fixation cross and finished when the child had fixated the indicated position, thus each trial elicited one saccade (the return saccade for next trial was not included in the analysis). Premature saccades (latency ≤ 120 ms) and directional errors were counted, but not included in the saccadic latency and duration calculations.

Fixation

Fixation on the saccadic target was defined as starting 80 ms after saccade offset and ending 80 ms prior to saccade onset as saccadic suppression typically persist for approximately 80 ms (Holmqvist et al., 2011). Each trial elicited one fixation period. In order to ensure homogeneity of fixations across trials and participants, fixation durations lasting less than 50 sampling points, or 400 ms, were excluded from the analysis.

The bivariate contour ellipse area (BCEA) was used to define the stability of fixation. BCEA refers to the area in which the eye is positioned for a given percentage of time, and is a reliable measure of fixation stability with good internal consistency (Vikesdal & Langaas, 2016b). To approximate normal distribution for analysis purposes, logBCEA including 68.2% of highest density points was used (Amore et al., 2013; Cesareo et al., 2014). Horizontal and vertical standard deviations of the eye position (σH and σV), as well as fixation duration, were also reported.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics version 22 (IBM Corp., New York, USA). The α level was set at 0.05. The One-Sample Kolmogorov-Smirnov test was performed to check for normality. Multivariate Analysis of Variance (ANOVA) and Mann-Whitney U tests were used to compare between-group differences in parametric and non-parametric data sets respectively. Pearson's correlations were used to identify associations between variables.

Results

Participants

Of the recruited children with dyslexia, three were excluded because they had diagnosed attention disorders, one was excluded because of possible neurological disease and two children were excluded because their sub-scores on language testing were within the normal range. As a result, 17 children (three females) were included in the developmental dyslexia (DD) group, and 17 children (seven females) in the typically developing (TD) group. The DD group were aged between 9 and 13 years, and the TD group were aged between 8 and 12 years. There was an increased prevalence of hyperopia and accommodation insufficiency in the DD group compared to the TD group, however no single optometric measure was significantly different between groups, for details see (Vikesdal et al., 2020). All DD children had significantly lower score on all sub-scores on language testing except from Grammar (see Table 1).

Table 1: *Language testing data.*

Group	Sum-score	Phonologic ability	Grammar	Decoding	Reading speed
TD	105.8	10.8	11.0	11.2	12.4
(*n*=17)	(± 10.5)	(± 1.8)	(± 2.4)	(± 2.1)	(± 3.4)
DD	89.9	5.6	9.8	5.9	6.2
(*n*=17)	(± 13.0)	(± 1.9)	(± 2.8)	(± 2.4)	(± 2.1)
p-value	< 0.01	< 0.01	0.21	< 0.01	< 0.01

Note: Mean values for all participants, ± standard deviation (SD) for the other variables. Scores are scaled such that mean = 10 and *SD* = 3 in a normal population (for sum-score: mean = 100 and *SD* = 15). All subtests except grammar were significantly different between groups.

Saccades

In total, 2677 valid saccade trials were analysed (1405 in the step task and 1272 in the gap task). The mean (± *SD*) number of trials in each task per participant was 38.8 (± 4.6) saccades (range 26–46). The number of included trials was similar across groups. The mean saccadic latency, number of premature saccades, and directional errors were extracted for all participants (see Table 2). Saccadic latency was normally distributed in the gap task (p=0.200), but not in the step task (p=0.027). Mann-Whitney U showed that there were no significant differences

Table 2: *Saccade data.*

Group	Step task				Gap Task			
	Latency (ms)	Premature saccades (number)	Direction errors (number)	Valid trials (number)	Latency (ms)	Premature saccades (number)	Direction errors (number)	Valid trials (number)
TD	227.1	0.1	1.6	42.9	204.3	0.9	7.4	36.6
(n=17)	(± 7.3)	(± 0.3)	(± 1.5)	(± 1.7)	(± 7.8)	(± 1.3)	(± 4.2)	(± 4.8)
DD	230.9	0.6	2.0	39.7	205.2	0.4	4.9	38.2
(n=17)	(± 4.7)	(± 0.9)	(± 1.9)	(± 3.4)	(± 7.3)	(± 0.6)	(± 4.6)	(± 5.0)

Note: Mean values for all participants, ± standard error (*SE*) for saccadic latency, and standard deviation (*SD*) for the other variables. There were no significant differences between groups.

between DD and TD groups for any of the included saccade parameters.

Fixations

In total, 2601 valid fixation trials were analysed (1301 in the step task and 1300 in the gap task). The mean (± *SD*) number of trials in each task per participant was 38.2 (± 5.0) fixations, range 18–46. The number of included trials was similar for each group. All fixation parameters were normally distributed (p=0.200) except for σV in the gap task (p=0.002). Table 3 shows the mean logBCEA, mean fixation duration and standard deviation of eye position (σH and σV) for all participants.

An ANOVA was used to test for differences between DD and TD groups, and showed that fixation stability was significantly poorer in the DD group compared to the TD group in both the step task and the gap task. Horizontal deviation of eye position was also greater in the DD group in both tasks. Figure 2 shows examples of fixations plots from four participants. To look for associations between fixation stability and visual acuity or accommodation, Pearson's correlations were performed. There was no correlation between fixation stability and either visual acuity or the accommodation amplitude. We have previously reported that participants with dyslexia more frequently have hyperopia and/or accommodation insufficiency (Vikesdal et al., 2020). However, these participants performed no differently from participants without these deficits – thus the reduction in fixation stability could not be explained by any optometric factor.

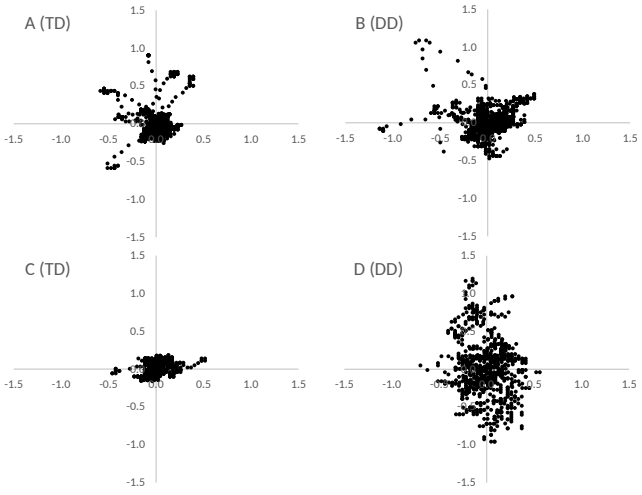

Figure 2: *Examples of fixation plots from TD participants with logBCEA = 2.56 ± 0.10 (A) and logBCEA = 2.17 ± 0.11 (C), and DD participants with logBCEA = 2.68 ± 0.13 (B) and logBCEA = 2.97 ± 0.14 (D). The x- and y-axes denote degrees of visual angle away from stimulus position.*

Pearson's correlations were also performed to look for associations between fixation stability and saccade parameters, which showed no correlation between fixation stability and either of the saccadic parameters.

Discussion

This study found no difference in saccadic parameters between children with and without dyslexia. Both groups had shorter saccadic latency in the gap task than in the step task, with a similar size of the expected gap effect. This suggests that disengaging visual attention is not problematic in this sample of children with dyslexia. In contrast, Bednarek et al. (2006) found that saccadic latency was shorter in children with dyslexia compared to controls, a difference that disappeared with central or peripheral cues. However, Bednarek et al.'s study included saccade latencies below 120 ms which could have biased their data. It has been suggested that saccade latencies have a bimodal distribution and that the so-called "express saccades" (with latencies below 120 ms) are more reflexive compared to "regular" saccades (Kristjànsson2011, 2011). Thus, previous conflicting findings of shorter saccade latencies in children with dyslexia may be due to increased prevalence of reflexive eye movements and not an actual increase in the regular saccadic latency. Reflexive saccades are not considered voluntary, and thus represent a different feature of oculomotor control, which is why we did not include these saccades in the present study. The findings do suggest that saccadic abnormalities are neither a necessary nor sufficient feature of dyslexia. It is unsurprising that children with dyslexia show abnormal saccades when reading (given that, by definition, they have a reading difficulty), but our findings are consistent with other reports of children with dyslexia having normal saccades when the cognitive demands of reading are removed (Hutzler et al., 2006).

Fixation stability was poorer in children with dyslexia compared with the typically developing children in both the step task and the gap task. Contrary to what was expected from our previous study (Vikesdal & Langaas, 2016a), fixation stability was not associated with visual acuity or accommodation measures. This suggests that factors other than refractive errors and accommodation ability are important to the stabilisation of gaze. It is well-known that the rate of microsaccades can be inhibited voluntarily. However, stability of fixation position does not always result after voluntary inhibition of microsaccades, which indicates that slow drifts play a large role in the stability of fixation (Rolfs, 2009). It has been suggested that poor stability may be created by gaze holding systems (e.g. vergence or the vestibular system) and that decreased stability may reflect noise in these systems (Otero-Millan et al., 2014). Even though fixation stability was measured monocularly, the test situation allowed for binocular viewing. Previous research has shown that dyslexics may have problems with binocular coordination after a saccade, which can lead to monocular instability (Bucci et al., 2008b). The findings in the present study support this finding.

Table 3: *Fixation data.*

	Step task				Gap Task			
Group	logBCEA (arcmin²)	σH (arcmin)	σV (arcmin)	Valid trials	logBCEA (arcmin²)	σH (arcmin)	σV (arcmin)	Valid trials
TD	2.51	7.42	10.18	39.6	2.48	8.17	11.75	40.5
(*n*=17)	(± 0.04)	(± 1.70)	(± 3.26)	(± 4.0)	(± 0.04)	(± 1.44)	(± 2.75)	(± 3.3)
DD	2.65	9.31	12.39	36.9	2.59	10.23	12.44	36.0
(*n*=17)	(± 0.04)	(± 1.89)	(± 3.41)	(± 6.2)	(± 0.04)	(± 2.03)	(± 3.89)	(± 4.7)
p	.021*	.005*	n.s.		.049*	.002*	n.s.	

Note: Values are mean values for all participants, ± standard error (*SE*) for logBCEA, and standard deviation (*SD*) for the other variables. *indicates significant differences between groups.

Fixation stability was not correlated with any optometric measure, nor with any saccade measure. The findings of reduced fixation stability but normal saccades in the participants with dyslexia suggest that these two systems operate independently, which is consistent with the findings from our previous study (Vikesdal & Langaas, 2016a).

The clinical consequences of the reduction in fixation stability are not obvious. The foveal region used to discriminate letters extends 1° either side of fixation, and the visual span for word recognition is much larger than this (Rayner, 1998). Thus, the reduction in fixation stability measured in this experiment is not large enough to reduce reading speed due to a temporal perception of a blurred image. Moreover, an experimental study performed on normal adult readers found that reading speed decreased with induced fixation instability (by random jittering), even though visual acuity was not affected (Falkenberg et al., 2007). Previous findings of reduced motion perception in dyslexic populations have been attributed to a lack of reading experience, and it has been found that a reading intervention targeting phonological ability improved motion perception (Olulade et al., 2013). It is possible that the poorer fixation stability found in the present experiments reflects poor cortical control because of a lack of reading experience. This possibility seems unlikely, however, as not all the children with dyslexia had poor fixation stability and yet they all had reduced experience of reading.

We suggest that the most likely explanation of the findings is that some of the children with dyslexia have general difficulties with the complex cognitive control processes that underpin saccadic response inhibition and stable fixation. This explanation is consistent with the co-morbidity known to exist between dyslexia and other neuro-developmental disorders (such as attention deficit hyperactivity disorder or developmental coordination disorder), as these other disorders have been shown to be associated with deficits in cognitive eye movement control processes (Gonzalez et al., 2016; Munoz et al., 2003; Sumner et al., 2016). Another explanation of our findings is that some of the children with dyslexia may also have difficulties with binocular coordination which may result in a temporarily unstable fixation after a saccade. This explanation is consistent with previous findings (Bucci et al., 2008b; Bucci et al., 2012), and together these explanations also reflect the multifactorial aetiology of dyslexia.

All dyslexic participants in this study had a phonological ability below the mean, however not all had poor fixation, which supports the claim that dyslexia is not solely explained by a phonological deficit. The fact that dyslexia is associated with alterations in occipito-temporal, temporo-parietal, and inferior frontal cortical areas (Richlan, 2012) is consistent with the idea that some of the higher-order cognitive eye movement control processes are likewise affected.

The finding that some children with dyslexia had fixation stability comparable with the control children (and conversely, some of the TD children had poorer stability) shows that fixation instability is neither a necessary nor sufficient feature of dyslexia. It does appear, however, that poor fixation stability is highly prevalent within the dyslexic population – an observation that is entirely consistent with the known co-morbidity between dyslexia and other neuro-developmental problems. This finding suggests that measures of unstable fixation can contribute in detection of developmental disorders including dyslexia.

Conflicts of Interest

The authors declare no conflict of interest.

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Normale sakkader men ustabil fiksering hos barn med dysleksi

Sammendrag

Dysleksi har en heterogen etiologi og rundt 5–15% av befolkningen har dysleksi. Optometriske avvik er mer vanlig blant personer med dysleksi, men sammenhengen mellom øyebevegelseskontroll og dysleksi er ikke etablert. I denne studien undersøkte vi om barn med dysleksi har redusert kontroll av sakkader eller fikseringsstabilitet, og om en redusert øyebevegelseskontroll er relatert til nedsatt synsskarphet og/eller akkommodasjon.

Trettifire barn med og uten dysleksi ble rekruttert til prosjektet. Alle deltakerne fikk en optometrisk undersøkelse og deltok i et sakkade- og fikseringseksperiment. Eksperimentene hadde to oppgaver: 'step' og 'gap'-oppgaven. Øyebevegelser ble registrert med et øyesporingskamera, og sakkade- og fikseringsparametere ble analysert separat.

Sakkade reaksjonstid, premature sakkader og retningsfeil var likt mellom barn med dysleksi og barn uten dysleksi. Derimot var fikseringer signifikant mindre stabile i dyslektikergruppen. Det var ingen sammenheng mellom sakkader eller fikseringsstabilitet og nedsatt synsskarphet og/eller akkommodasjon.

Barn med dysleksi hadde like god kontroll av sakkader som kontrollgruppen, men fikseringsstabiliteten deres var redusert sammenlignet med kontrollgruppen. Den reduserte fikseringsstabiliteten kan forklares med generelle mangler i de kognitive prosessene som ligger bak øyebevegelseskontroll, som også er funnet ved andre utviklingsforstyrrelser.

Nøkkelord: Øyebevegelser, dysleksi, fiksasjon, sakkader

Saccadi normali ma stabilità di fissazione ridotta in una popolazione di bambini con dislessia

Riassunto

La dislessia inerente allo sviluppo colpisce all'incirca il 5–15% della popolazione ed ha una eziologia eterogenea. I disturbi optometrici sono più prevalenti in una popolazione dislessica però la relazione tra controllo dei movimenti oculari e dislessia non è ben stabilita. In questo studio, abbiamo investigato dove bambini con dislessia mostrano deficit di saccadici o di fissazione e dove questi deficit sono relazionati con l'acuità visiva e/o con l'accomodazione. 34 bambini con e senza dislessia sono stati reclutati per questo progetto. Tutti i partecipanti hanno avuta un esame optometrico e saccadi e fissazione sono stato considerate. Noi abbiamo utilizzato il paradigma dei due occhi: l'esercizio del passo e del salto. I movimenti oculari sono stati registrati con un eye-tracker ad infrarossi e i parametri delle saccadi e fissazione sono stati analizzati separatamente. Latenza delle saccadi, saccadi premature, errori direzionali sono stati simili tra bambini con dislessia e bambini in fase di sviluppo. Al contrario, le fissazioni sono state significativamente meno stabili nel gruppo della dislessia. Nessuna tra saccadi e fissazione sono state associate a deficit di accomodazione o di acuità visiva. I bambini con dislessia non hanno mostrato difficoltà nelle saccadi, ma loro stabilità di fissazione è stata ridotta rispetto al gruppo controllo. Tale riduzione può essere spiegata da deficit generali nel processo cognitivo che regola il controllo dei movimenti oculari, i quali sono stati trovati anche responsabili in altro disordini del neuro-sviluppo.

Parole chiave: Movimenti oculari, dislessia, fissazione, saccadi